THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

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This Issue in the Journal

Peritonsillar infection in Christchurch 2006–2008: epidemiology and microbiology

Rachelle L Love, Rob Allison, Stephen T Chambers

Peritonsillar infection is a common and potentially life-threatening complication of acute tonsillitis. There are changes reported in the causative bacteria seen in overseas populations, along with resistance to some antibiotics. However, infections treated in our unit over the past 30 years still respond favourably to penicillin, along with evacuation of pus by aspiration and incision and drainage.

Acute medical admissions for older people from Residential Care Facilities: are they appropriate?

H Carl Hanger, Valerie Fletcher , Andrew Sidwell

Some people believe that many admissions from residential care facilities are potentially inappropriate. This study explores whether the acute hospital doctors treating these patients thought they were appropriate or not. Most admissions in this study were appropriate and many returned to their usual residence. The admission of a smaller number of very frail, or dying, older people could have been avoided.

Campylobacteriosis rates show age-related static bimodal and seasonality trends

Warrick Nelson, Ben Harris

Campylobacter gastroenteritis is far more common in New Zealand than other developed countries (over twice the Australian rate). People of different ages are affected at different rates. Babies/toddlers under 5 and young adults (20–29 years) are most likely to be affected compared to other age groups, why? *Campylobacter* infections are also highly seasonal, most common in summer and least in winter, whereas chicken consumption remains relatively even throughout the year. However, young children have an enhanced degree of seasonal summer/winter infection variation but at lower rates than other ages, while babies/toddlers under 5 and young adults (20–29) show reduced summer winter seasonal variation. *Campylobacter* infection is commonly associated with chicken meat consumption or contamination of other foods from chicken meat.

These age-related differences suggest a far more complex relationship than simply exposure to chicken meat, the more recent conventional explanation. While it is commonly assumed previous infection will result in some acquired immunity, these observations suggest more is at play as young adults (20–29) suddenly appear to lose immunity, but teenagers and older age groups remain relatively protected—why?

Auckland City Hospital's Ortho-Geriatric Service: an audit of patients aged over 65 with fractured neck of femur
Lucy Fergus, Greer Cutfield, Roger Harris

This is an audit of the process of care that 115 consecutive elderly patients with a hip fracture received at Auckland city Hospital in 2007. It demonstrates that hip fracture has a significant negative effect on older people in terms of medical problems, ability to mobilise and to live independently. A 'fast-track' system to transfer these patients from Orthopaedics to Older Peoples Health departments as soon as possible after operation reduced time in hospital without any obvious negative consequence. Comparison with previous audits at Auckland City Hospital showed that fewer patients received their surgery within 24 hours. This may be an unintended consequence of both the reconfiguration of Orthopaedic services within the Auckland region and also the move into the new hospital that took place in 2003. Comparison with other New Zealand centres shows significant variability in the process of care.

Prevalent dietary supplement use in older New Zealand men
Catherine J Bacon, Mark J Bolland, Ruth W Ames, Amanda T Y Siu, Barbara H Mason, Anne M Horne, Andrew Grey, Ian R Reid

Dietary supplement use and reasons for it were surveyed in middle-aged and older men volunteering for a large regional trial of calcium supplements. Almost half (47%) took other supplements during the study, reporting use of a very wide range of different products. Half of the men spent $20 or more per month on these products, with a fifth spending more than $50 per month. Health professionals should remain alert to supplement use by their patients, including males.

Patterns of chronic pain in the New Zealand population
Clare Dominick, Fiona Blyth, Michael Nicholas

This paper describes the prevalence of chronic and recent pain in the New Zealand population by a range of sociodemographic factors, the use of treatment services for chronic pain and the impact of chronic pain on health related quality of life. Data from the 2006/07 New Zealand Health Survey, commissioned by the Ministry of Health, were analysed and provide the first opportunity to gauge the prevalence and impact of chronic pain on the New Zealand population as a whole. Patterns of chronic pain in New Zealand are similar to those found internationally showing increased prevalence with age and a higher prevalence with lower socioeconomic living standards. The impact of chronic pain on health related quality of life is dramatic with much poorer quality of life associated with greater numbers of pain sites and greater intensity of recent pain. Older age groups, women and those with greater intensity of recent pain are more likely to seek medical treatment for their chronic pain, but a substantial minority do not seek any treatment (whether medical or other treatment) for their chronic pain. The paper indicates that chronic pain represents a major health issue in New Zealand

Do senior medical students know enough clinical anatomy? ((letter))
Sultan Al-Shaqsi, Mark D Stringer

A good working knowledge of clinical anatomy is essential for safe and effective medical practice. There is widespread concern that students are failing to learn sufficient clinical anatomy. Anatomical ignorance has been linked to an increase in medical errors. A straightforward test of clinical anatomy highlighted patchy deficiencies among senior medical students at the Dunedin School of Medicine. These deficiencies might be rectified by incorporating a modest amount of clinical anatomy teaching into the later years of medical undergraduate training.

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The importance of promoting physical activity for cancer survivorship

Justin W L Keogh, Lynnette Jones

Cancer is one of the leading causes of death in many countries, including New Zealand. Projections suggest that New Zealand will have over 22,000 new cancer cases in 2011, a substantial increase from the 15,000 cases reported in 2005.[1] This increased number of new cancer cases may reflect the ageing of the population, insufficient levels of physical activity, poor dietary choices, other unhealthy lifestyle choices such as smoking as well as improvements in cancer detection.[2] Improvements in treatment modalities for many common cancers also appear to be contributing to many more individuals with cancer living longer post-diagnosis.[3]

Regardless of the treatment option, many cancer patients (survivors) experience significant fatigue, physical disability and reductions in overall quality of life.[4] Moreover, these decrements often become more pronounced over time or with additional treatment(s).[4] Fatigue and physical impairment imposes many challenges on the lives of cancer survivors by significantly interfering with their ability to perform self-care, work and leisure activities,[5] thus further contributing to reductions in their quality of life.

These debilitating side-effects of treatment may also prevent many cancer survivors from engaging in sufficient physical activity for health benefits[6] and increase their risk of developing metabolic syndrome.[2] As a result, we would argue that more services and resources need to be allocated to target these key psychosocial and physical issues facing the burgeoning population of cancer survivors worldwide. One way to address this shortfall could be the development of physical activity programmes for cancer survivors that focus on improving their quality of life and overall health.

The rationale for this is recommendation is that many cross-sectional studies[7] including the paper by Keogh and colleagues[8] reprinted in this issue, have demonstrated a link between levels of physical activity and quality of life in a range of cancer groups. Further, recent systematic reviews of experimental research in this area have demonstrated that general physical activity and structured exercise programmes have many physiological and psychosocial benefits for cancer survivors.[2,9,10]

These and other reviews indicate that resistance training and aerobic training (although possibly to a lesser extent) can significantly improve body composition, muscular strength and endurance, aerobic fitness, functional performance in activities of daily living and various aspects of quality of life as well as reduce fatigue in a variety of cancer groups. Some evidence also suggests that exercise may improve immune function and reduce markers of cancer progression, postponing the need to initiate treatments with known side-effects, thereby possibly leading to increased longevity and healthier survivorship.

Unfortunately, many cancer survivors do not perform sufficient physical activity for health benefit. For example, Keogh and colleagues[8] reported that only 45% of prostate cancer survivors were physically active—i.e. performing 150 minutes of moderate intensity or 60 minutes of vigorous physical activity per week. It would therefore appear that more research needs to be conducted into determining ways to increase physical activity levels (perhaps via structured exercise programmes) in cancer survivors.

The article in this issue by Szymlek-Gay and colleagues[11] addresses this concern in a number of ways. It not only presents some of the evidence for the benefits of exercise in a variety of cancer groups, but also discusses some of the common barriers and motives to exercise for those with cancer and the issues surrounding the relative lack of specific cancer exercise programmes available in New Zealand and many other countries.

Of particular interest to readers of the *New Zealand Medical Journal*, Szymlek-Gay et al[11] discuss the important role that cancer clinicians such as surgeons, oncologists, urologists, physicians and practice nurses have in discussing quality of life concerns and encouraging regular physical activity in their patients. Such a recommendation is consistent with the theory of planned behaviour (the most widely used psychological theory to explain exercise behaviour in cancer groups)[7,8] and is supported by Jones et al[12] who found that cancer survivors significantly increased their level of physical activity when recommended to by their cancer clinician.

Based on these findings[12] as well as the evidence for the benefits of physical activity,[2,9,10] we would like to challenge all those who work with cancer patients and survivors (especially cancer clinicians) to focus more on improving the quality of life and not just the quantity of life these individuals may have left. Based on the evidence provided in this editorial and the paper of Szymlek-Gay and colleagues,[11] one way to achieve this could be to develop better interdisciplinary links with local clinical exercise specialists and providers. Such collaborations could arguably lead to the development of a number of evidence-based, accessible physical activity programmes for cancer survivors throughout the country, with the likely result being improvements in many cancer survivors' quality of life and overall health.

We would therefore argue that the development and availability of such physical activity programmes is an important national health care issue, one with particular relevance to the New Zealand Cancer Control Strategy. Some progress has been made in this area with three breast cancer exercise programmes (Beyond Pink, YWCA Encore and Pink Pilates) available in some parts of New Zealand. Unfortunately, there appears to be very few options available for survivors of other cancers (with the possible exception of Club Physical's Cancer Wellfit programme in Auckland), particularly for cancers affecting men. This is hopefully set to change with the introduction of a programme for men called Steel Pilates in the near future.

In conclusion, while regular physical activity and structured exercise have many benefits for cancer survivors,[2,9,10] most cancer survivors do not engage in sufficient physical activity to improve their quality of life and overall health.[6] Cancer clinicians are in an ideal place to improve this, as their recommendations can significantly increase the physical activity levels of their patients.[12] However, a barrier to long-term

physical activity in cancer survivors may be a lack of suitable programmes and on-going support.[6]

Thus, we would strongly encourage cancer clinicians to develop better links with local cancer support workers and clinical exercise specialists, so that they can confidently refer their patients to evidence-based physical activity programmes in their region. If no such programmes are currently available in their region, we would encourage cancer clinicians to lobby the relevant authorities for their introduction. By these actions, cancer clinicians will further contribute to maximising the health and quality of life of cancer survivors nation-wide.

For those interested in these cancer exercise programmes, contact details can be found at:

- Beyond Pink: Dr Lynnette Jones, lynnette.jones@otago.ac.nz
- Encore: http://www.breastcancersupport.co.nz/Support/Coping+with+breast+cancer
- Pink Pilates: http://www.pinkpilates.co.nz/
- Cancer Wellfit: http://www.clubphysical.co.nz/
- Steel Pilates: http://www.pinkpilates.co.nz/mens-cancer-programme

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Author information: Justin W L Keogh[1,2,3] and Lynnette Jones[4]

1. Person Centred Research Centre, AUT University, Auckland, New Zealand
2. Centre for Physical Activity and Nutrition Research, AUT University, Auckland, New Zealand
3. Faculty of Health Sciences and Medicine, Bond University, Gold Coast, Australia
4. School of Physical Education, University of Otago, Dunedin, New Zealand

Correspondence: Associate Professor Justin Keogh, School of Sport and Recreation, AUT University, Private Bag 92006, Auckland, New Zealand. Fax: +64 (0)9 9219960; email: justin.keogh@aut.ac.nz

References:

1. Cancer Control Task Force. The New Zealand Cancer Control Strategy: Action Plan 2005-2010. Wellington: Ministry of Health and the New Zealand Cancer Control Trust; 2005.
2. Newton R, Galvão D. Exercise in prevention and management of cancer. Curr Treat Options Oncol. 2008;9:135-46.
3. Etzioni R, Tsodikov A, Mariotto A, et al. Quantifying the role of PSA screening in the US prostate cancer mortality decline. Cancer Causes Control. 2008;19:175-81.
4. Baker F, Denniston M, Smith T, et al. Adult cancer survivors: how are they faring? Cancer. 2005;104:2565-76.
5. Truong PT, Berthelet E, Lee JC, et al. Prospective evaluation of the prevalence and severity of fatigue in patients with prostate cancer undergoing radical external beam radiotherapy and neoadjuvant hormone therapy. Can J Urol. 2006;13:3139-46.
6. Thorsen L, Courneya K, Stevinson C, et al. A systematic review of physical activity in prostate cancer survivors: outcomes, prevalence, and determinants. Support Care Cancer. 2008;16:987-97.

7. Blanchard CM, Stein KD, Baker F, et al. Association between current lifestyle behaviors and health-related quality of life in breast, colorectal, and prostate cancer survivors. Psychol Health. 2004;19:1-13.
8. Keogh JWL, Shepherd D, Krägeloh CU, et al. Predictors of physical activity and quality of life in New Zealand prostate cancer survivors undergoing androgen-deprivation therapy. N Z Med J. 2010;123:20-9.
9. Spence RR, Heesch KC, Brown WJ. Exercise and cancer rehabilitation: a systematic review. Cancer Treat Rev. 2010;36:185-94.
10. Keogh JWL, MacLeod RD. Body composition, physical fitness, functional performance, quality of life and fatigue benefits of exercise for prostate cancer patients: a systematic review. J Pain Symptom Manage. in press. doi:10.1016/j.jpainsymman.2011.03.006
11. Szymlek-Gay EA, Richards R, Egan R. Physical activity among cancer survivors: a literature review N Z Med J. 2011;124(1337). http://www.nzma.org.nz/journal/124-1337/4746
12. Jones LW, Courneya KS, Fairey AS, et al. Effects of an oncologist's recommendation to exercise on self-reported exercise behavior in newly diagnosed breast cancer survivors: a single-blind, randomized controlled trial. Ann Behav Med. 2004;28:105-13.

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Peritonsillar abscess

Emily Macassey, Patrick J D Dawes

In this issue of the *NZMJ*, Love et al[1] report interesting observations about patterns of epidemiology and microbiology of peritonsillar abscess (PTA) in Canterbury and make comparisons with previous studies performed at Christchurch Hospital. The report contains some valuable observations when it comes to the treatment of the condition.

Peritonsillar abscess (also known as quinsy) is a potentially life-threatening infection of the potential space adjacent to the tonsillar capsule in the oropharynx. It can be difficult for doctors unfamiliar with PTA to differentiate it from severe tonsillitis but unilaterality of symptoms and inability to swallow saliva are good indicators. The main differentiating signs seen are trismus, swelling or bulging of the soft palate, medialisation of the tonsil and deviation of the uvula.[2] PTA is life-threatening because of both its potential for airway obstruction and spread to the parapharyngeal and retropharyngeal spaces. It is reported that George Washington probably died from quinsy in 1799.[3]

It is reassuring that 97.3% of isolates are reported as penicillin sensitive. Penicillin remains the first-line antibiotic for all tonsillar infections and this is the sole agent used in many New Zealand hospitals. In other countries resistance rates vary from 10–50%.[4] In a survey of UK consultants, 28% had a preference for penicillin monotherapy, whilst penicillin combined with metronidazole was the choice of 44%.[3] Research has shown that even when patients have penicillin resistant organisms, treatment with aspiration and parenteral penicillin still achieves clinical resolution.[5] This is in accordance with principles of abscess management where drainage is paramount.

Penicillin remains an effective drug especially when given at adequate dosage. The usual recommended dosage for tonsillitis and PTA is 500 mg phenoxymethylpenicillin four times a day for duration of 5 to 10 days. It is effective for all peritonsillar bacterial infections ranging from tonsillitis to quinsy. A Cochrane review concludes that use of oral antibiotics in sore throat reduces the risk of suppurative complications such as PTA.[6] Some patients with severe tonsillitis and those developing early unilateral tonsillar symptoms may require intravenous antibiotics.

With the increasing use of community nurse antibiotic administration there is scope to effectively manage such patients with the aim of preventing progression of the illness and PTA formation. Using penicillin has two main advantages; avoidance of both side-effects such as diarrhoea and candidiasis associated with broader spectrum antibiotics, and the commonly observed maculopapular rash when infectious mononucleosis is treated with amoxycillin and its derivatives. If a patient is penicillin allergic, erythromycin or a first-generation cephalosporin is an effective alternative.

The report by Love et al also touches on some changing aspects in the management of PTA. The ENT literature debates aspiration versus incision and drainage of the abscess,[7,8] and many centres may perform both depending on the clinical situation. Both techniques are generally performed under local anaesthesia owing to the advantage conferred by a patient protecting their own airway. If drainage is done under general anaesthesia (such as might be done in children or poorly-compliant patients) abscess drainage is usually achieved by tonsillectomy.

Some centres perform acute or "hot" tonsillectomy for PTA. In 2001 only 1% of UK surgeons performed hot tonsillectomies, whilst 12% would perform tonsillectomy for those with abscesses that were slow to settle.[9] The advocates of acute tonsillectomy promote one recovery time from both infection and surgery. Argument against this approach is that there is increased intra-operative blood loss and a presumed increase in the rate of postoperative haemorrhage; and critics maintain that following a single episode of PTA patients are statistically unlikely to develop any further tonsillar problems, thus negating the need for tonsillectomy.[10]

Controversy exists over the rate of reactionary and secondary haemorrhage following hot tonsillectomy. Retrospective studies comparing rates of bleeding show an increased rate of haemorrhage following both acute and interval tonsillectomy compared to elective tonsillectomy. In one study the interval tonsillectomy group had a haemorrhage rate of 11.6% compared to 8% in the acute tonsillectomy group, this difference was not statistically significant.[11,12]

In summary, peritonsillar abscess is a moderately common complication of tonsillitis which requires urgent treatment because of discomfort, airway obstruction and risk of deep neck space abscess formation. Reassuringly the local pattern of antibiotic sensitivities allows the use of penicillin as a first-line monotherapy. Most patients have a single episode of PTA and will require no further intervention. Future research clarifying the role of "hot tonsillectomy" in the management of peritonsillar abscess is awaited with interest.

Competing interests: None.

Author information: Emily Macassey, Otolaryngology Senior Registrar; Patrick J D Dawes, Otolaryngology Consultant, Department of Otolaryngology, Head and Neck Surgery; Dunedin Public Hospital, Great King Street, Dunedin

Correspondence: Associate Professor Patrick Dawes, Otolaryngology Head and Neck Surgery Department, Dunedin Public Hospital Private Bag 1921, Dunedin 9001, New Zealand. Fax: +64 (0)3 4747956; email: patrick.dawes@southerndhb.govt.nz

References:

1. Love RL, Allison R, Chambers ST. Peritonsillar infection in Christchurch 2006–2008: epidemiology and microbiology. N Z Med J. 2011;124(1337). http://www.nzma.org.nz:8080/journal/124-1337/4732/content.pdf
2. Rahn R, Hutten-Czapski P. Quinsy (peritonsillar abscess). Can J Rural Med 2009;14(1):25-6.
3. Scheidemandel HH. Did George Washington die of quinsy? Arch Otolaryngol 1976 Sep;102(9):519-21
4. Visvanathan V, Nix P. National UK survey of antibiotics prescribed for acute tonsillitis and peritonsillar abscess. J Laryngol Otol 2010;124:420–423.
5. Hanna B, McMullan R, Gallagher G, Hedderwick S. The epidemiology of peritonsillar abscess disease in Northern Ireland. J Infect 2006;52(4):247-253.

6. Del Mar CB, Glasziou PP, Spinks AB. Antibiotics for sore throat (systematic review) Cochrane acute respiratory infections group. Cochrane Database Syst Rev 2006;(4):CD000023
7. Galioto NJ. Peritonsillar abscess. Am Fam Physician. 2008;77(2):199-202.
8. Johnson RF, Stewart MG, Wright CC. An Evidence-Based Review of the Treatment of Peritonsillar Abscess. Otolaryngol Head Neck Surg 2003;128:332-343.
9. Mehanna HM, Al-Bahnasawi L, White A. National audit of the management of peritonsillar abscess. Postgrad Med J 2002;78:545-8.
10. Raut VV, Yung MW. Peritonsillar abscess: The rationale for interval tonsillectomy. Ear Nose Throat J. 2000;79(3):206-9.
11. Giger R, Landis BN, Dulguerov P. Hemorrhage Risk After Quinsy Tonsillectomy Otolaryngol Head Neck Surg 2005;133:729-34.
12. Lehnerdt G, Senska K, Jahnke M, Fischer M. Post-tonsillectomy haemorrhage: A retrospective comparison of abscess and elective tonsillectomy. Acta Otolaryngol 2005;125:1312-1317.

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Can New Zealand do better in colorectal cancer?

Ian P Bissett

How does New Zealand (NZ) fare in managing bowel cancer? The incidence of the disease is one of the highest in the World but the outcomes of treatment in terms of overall survival fall short of some of our OECD neighbours.[1] Even within NZ the results for patients with colorectal cancer are worse for Māori both in terms of cancer related survival and post-operative mortality. While some of this is explained by higher comorbidity in Māori there is also evidence that access to and quality of care is poorer for this group.[2] Identifying the problem is one thing but implementing changes that will result in improving outcomes is quite another.

This issue of the *Journal* includes a summary of the guidance for the management of Early Bowel Cancer produced and published by the NZ Guidelines Group.[3] This guideline provides recommendations for those responsible for the management of bowel cancer once the disease has been diagnosed. Rather than starting from scratch this is based on a document produced by the National Health and Medical Research Council in Australia in 2005. In a pragmatic approach, using advice from a group of experts who were nominated for their knowledge of colorectal cancer in NZ, sections of the guideline that were considered out of date, inadequately supported by evidence, or inappropriate to the NZ context were identified.

Further literature searches were performed to answer clinical questions developed to address these areas using evidence dating from 2004 onwards. The draft guideline was then widely circulated within NZ to groups with a strong involvement in colorectal cancer management. The feedback received was also incorporated into the final document as appropriate.

This summary contains a list of 85 recommendations for the management of the patient journey from preoperative assessment to long term follow-up and includes cultural, communication, and reporting issues. Twenty-seven of these are ‘good practice points’ based not on published evidence but on the experience or opinion of leaders in the field. Many of these are ‘common sense’ such as “supportive and rehabilitative care should be available to all people with colorectal cancer.” The inclusion of these ‘good practice points’ serves to highlight the importance of many of the less easily measured components of the care of bowel cancer patients. These may nevertheless have a powerful impact on the patient and their experience of treatment. The other 58 recommendations have at least some published evidence to support them even if this merely shows that there is no clear advantage to a particular intervention.

Can this guideline make a difference for individuals with bowel cancer and for the provision of bowel cancer services nationally? I would suggest that there are at least four ways in which this may occur. The guideline provides a tool to help demonstrate evidence, calibrate performance, advocate for patients, and legislate for change.

The greatest limitation of a guideline is that it may be wrong and this may come about for several reasons, such as lack of supporting data, strongly held opinions of

influential players or conflicting priorities among the stakeholders.[4] This guideline, however, gathers together the evidence relating to essential steps in the patient's management and presents it in a way that clearly outlines the strength of that evidence. For busy clinicians it provides a resource of well researched answers to common questions. Where widely held beliefs are refuted by good evidence (such as the need for oral bowel preparation in colonic surgery) this is clearly demonstrated. The guideline has also incorporated contributions from the professional groups who manage colorectal cancer in NZ and therefore offers the clinician some further confidence that there is strong peer support for the recommendations stated.

The second possible advantage of the guideline is illustrated by a slight variation of an accepted management adage, 'You cannot improve what you do not measure'. The guideline offers an accepted standard against which bowel cancer management can be assessed. Where there is good evidence for a particular treatment regimen there is also the ability to calibrate individual and group performance against that standard. This measurement itself tends to be a strong driver for improving compliance. Consider, for instance, the recommendation that postoperative chemotherapy should be offered to all Stage III colorectal cancer patients unless there is a particular contraindication. A requirement to document whether this has occurred is likely to increase the rate of delivery of adjuvant chemotherapy to this group, of whom only 69% were offered chemotherapy in a recent study.[5]

Detractors of guidelines consider them to be too rigid and likely to encourage doctors to practise 'cookbook' medicine with little thought of how they apply to individuals. This guideline includes five recommendations in relation to multidisciplinary teams, particularly that all patients with colon or rectal cancer should be discussed at a Tumour Board or Multidisciplinary meeting. This is the venue for clinicians to ensure that all patients have their management individually tailored to their specific situation. It is at the Tumour Board meeting that those who know the patient best can advocate for them. The treatment of many patients may appear straightforward but consideration, for instance, of the likelihood of a familial syndrome or the need for more extensive surgery may easily be overlooked by a busy clinician practising in isolation. In my experience the inconvenience of fitting a meeting into my schedule is greatly outweighed by the benefits of intellectual input from colleagues and improved decision making and outcomes for the patients.[6]

Finally, implementation of these recommendations may be seen as impractical on a national scale. How can multidisciplinary teams, for instance, cater for patients from all over NZ? The guideline itself can be a powerful force to improve services. If it is accepted as the appropriate standard of care for patients, it gives surgeons, oncologists, and hospital boards the mandate to require change. At present there are multidisciplinary meetings advising the optimal management of patients with bowel cancer at most of the large centres, where oncology, pathology, and radiology specialists are available. It would not require a great capital investment to make these available by video-conference to all those who at present are functioning without them. The guideline itself has the potential to be used as a tool to put pressure on those who control budgets and ensure that its recommendations can be met at each institution. Rather than a threat to clinicians functioning in a smaller centre, it should be seen as a way to assist them to get what they and their patients need.

The success of this guideline in improving outcomes nationally is in our hands. It will depend on clinicians taking it seriously and bringing its recommendations to the attention of colleagues, managers, planners, and patients.

Competing interests: None.

Author information: Ian P Bissett, Associate Professor and Head of Colorectal Surgical Unit[1,2]

1. Department of General Surgery, Auckland City Hospital, Auckland
2. Department of Surgery, School of Medicine, Faculty of Medical and Health Sciences, University of Auckland

Correspondence: Ian P Bissett, Department of Surgery, School of Medicine, Faculty of Medical and Health Sciences, University of Auckland, Park Road, Grafton, Auckland 1001, New Zealand. Fax: +64 (0)9 3779656; email: i.bissett@auckland.ac.nz

References:

1. OECD: Health at a Glance. 2009; Available from: http://www.oecd.org/document/11/0,3746,en_2649_34631_16502667_1_1_1_1,00.html
2. Hill S, Sarfati D, Blakely T, et al. Survival disparities in Indigenous and non-Indigenous New Zealanders with colon cancer: the role of patient comorbidity, treatment and health service factors. J Epidemiol Community Health. 2010;64(2):117-23.
3. Fitzgerald A, Frizelle F, Jeffery M, et al. Summary of guidance for the management of early bowel cancer. N Z Med J. 2011;124(1337). http://www.nzma.org.nz/journal/124-1337/4740
4. Woolf SH, Grol R, Hutchinson A, et al. Clinical guidelines: potential benefits, limitations, and harms of clinical guidelines. BMJ 1999;318(7182):527-30.
5. Cunningham R, Sarfati D, Hill S, et al. Colon cancer management in New Zealand: 1996-2003. N Z Med J. 2009;122(1294):51-60. http://www.nzmj.com/journal/122-1294/3581/content.pdf
6. Burton S, Brown G, Daniels IR, et al. MRI directed multidisciplinary team preoperative treatment strategy: the way to eliminate positive circumferential margins? Br J Cancer. 2006;94(3):351–7.

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Peritonsillar infection in Christchurch 2006–2008: epidemiology and microbiology

Rachelle L Love, Rob Allison, Stephen T Chambers

Abstract

Aim Peritonsillar infection is a complication of acute tonsillitis. It is common and complications can be life-threatening. This study audits all cases of peritonsillar infection presenting to our unit between 2006 and 2008 in order to determine if the epidemiology, bacteriology and antibiotic sensitivity has changed since previous audits in our unit in 1981–1984 and 1990–1992.

Methods Retrospective chart review.

Results 213 patients were admitted acutely with peritonsillar infection between January 2006 and December 2008. The average age was 29 years with 30.5% patients in the modal age group of 15–19 years. Male to female ratio was 1.5:1. 54% presented with their first episode. 39% received antibiotics prior to presentation. In two-thirds of cases, the duration of admission was less than 24 hours. Culture results were obtained from 69% of specimens. Cultures mostly contained mixed anaerobic and aerobic bacteria. The most common aerobes were streptococcal species. Organisms were almost uniformly sensitive to penicillin. 21% of patients subsequently underwent tonsillectomy, usually as a delayed procedure.

Conclusion Peritonsillar infection is a common complication of tonsillitis and can be life-threatening. The number of cases presenting at Christchurch Hospital has increased disproportionate to the population increase since the previous audits. Culture results demonstrate a preponderance of mixed organisms, which may be pathological. Organisms and their sensitivities have not changed since the previous audits. Infection usually responds favourably to drainage in combination with penicillin as the first-line antimicrobial agent.

Peritonsillar infection is a common complication of acute tonsillitis. It mostly affects young healthy adults and is frequently seen in patients without a prior history of tonsillitis. Common presenting complaints are odynophagia, dysphagia and trismus. Clinically the palate may be cellulitic, with a unilateral, fluctuant swelling pushing the uvula across the midline.

The development of a peritonsillar abscess (PTA) can be life-threatening because of potential complications. These include airway swelling, abscess formation in adjacent deep neck tissues, and systemic sepsis. The mainstays of treatment are antimicrobial therapy plus evacuation of pus by aspiration, incision and drainage or acute tonsillectomy.

The identity of organisms isolated from PTA specimens and their significance in its pathogenesis has been widely studied. The results of these reports vary significantly.

This may reflect differing culture methods and incubation times[1] or possibly a change in the biologic behaviour of the bacteria.[2]

The value of routine microbiological examination of PTA specimens has been questioned[3] because antibiotic regimes are usually prescribed empirically, based on the clinical condition of the patient rather than the culture results, which are often not available until after a patient has been discharged.

Multiple reports have shown penicillin to be an effective first line antibiotic for peritonsillar infection[3–5] although emerging penicillin resistance in aerobic species[1] has been documented.

This study audits all cases of peritonsillar infection managed within our unit over a recent 3-year period to determine if the epidemiology, bacteriology and antibiotic sensitivity has changed since similar audits undertaken in our unit in 1981–1984 and 1990–1992.

Methods

The charts of all patients admitted with a diagnosis of peritonsillar infection at Christchurch Public Hospital, a tertiary referral centre, between January 2006 and December 2008 were retrospectively reviewed.

Demographic data, pre-hospital antibiotic management, presence of peritonsillar cellulitis or abscess (PTA), surgical and medical management of the infection, rates of acute and interval tonsillectomy, and bacteriology results were collected by comprehensive chart and electronic record review.

All patients admitted with peritonsillar infection were commenced on empiric intravenous (IV) antibiotics. The presence of cellulitis or abscess was determined by clinical examination. As per the protocol in our unit, both types of infection were aspirated by wide-bore needle and evacuated pus sent to the laboratory for gram-stain, aerobic and anaerobic culture and antimicrobial susceptibility testing.

Any abscesses were then formally incised and drained under local anaesthetic. Patients in whom aspiration was negative were diagnosed as having peritonsillar cellulitis. The majority of patients were discharged before culture and susceptibility testing results were available. Once microbiology results were available, the patients' general practitioner was informed, to enable adjustment of antibiotics as necessary.

The antibiotic given at discharge was in all cases the oral equivalent of the intravenous antibiotic the patient received as an inpatient.

Bacteriological technique—Pus was collected via a 20-gauge needle in a plastic syringe, the air expelled and the needle replaced with a rubber stopper. Specimens were sent directly to the laboratory. Chocolate and 5% sheep blood and agar plates were inoculated for aerobes, incubated at 37 degrees Celsius in a CO_2 enriched environment and examined at 24 hours. Anaerobes were plated on 5% sheep blood agar and examined at 48–72 hours.

Results were compared with two previous audits from our unit in 1981–4 and 1990–2. Bacteriology and penicillin sensitivities were compared, along with epidemiology.

Results

There were 228 episodes of peritonsillar infection involving 215 patients. Thirteen patients were readmitted with unresolved or recurrent peritonsillar infection. Two further patients were excluded due to incomplete records, leaving 213 in the study group.

Age and sex—The average age was 29 (range 6–82) with 30.5% patients in the modal age group of 15–19 years (see Table 1). The ratio of male to female patients was approximately 1.5:1 (120/93).

Table 1. Epidemiology of patients with peritonsillar infection presenting to Christchurch Hospital 2006–2008

Variables		Number	Percentage
Sex (n=213)	Male	120	56
	Female	93	44
Pre-hospital antibiotics (n=213)	No	129	61
	Yes	84	39
	Penicillin family	64	76
	Other	20	24
Previous episodes of tonsillitis or PTA (n=213)	No	115	54
	Yes	98	46
	Single	38	18
	Recurrent	60	28
Tonsillectomy (n=213)	No	166	79
	Yes	47	21
	Acute	14	30
	Interval	33	70

Pre-hospital antibiotic use—129 patients (61%) did not receive antimicrobial therapy prior to acute admission (129/213). Of those who did, 71/84 (85%) received five or less days of antimicrobial therapy (range 1–10 days). These included penicillin V (48/84), amoxicillin-clavulanic acid (10/84) and amoxicillin (6/84).

History of throat infections—The majority of patients presented with their first episode of peritonsillar infection (115/213). In total, 18% had a single prior episode of either tonsillitis (25/213) or of quinsy (13/213); while 28% presented with recurrent episodes(60/213). Timing between previous episodes and presentation was not reliably obtainable from chart review.

Bacteriology—Aspirates were obtained and cultured in 147 (69%) of cases. Important organisms isolated on culture are shown in Table 2. Beta-haemolytic streptococcus was the most commonly isolated single organism (70/147). A mix of anaerobic and aerobic bacteria was identified in 84 cultures (57%). Six cultures contained organisms resistant to penicillin, usually *Staphylococcus aureus* (5/6).

Patients who received antibiotics prior to admission had aspirates containing the same pattern of bacteria as those who did not.

Hospital antibiotic use—175 patients (82%) received IV benzylpenicillin for 24 hours. The most common dosing schedule was 6-hourly (119/1175). Amoxicillin-clavulanic acid was prescribed for 18 patients. Fourteen patients with penicillin allergy received first- (3/14), second- (8/14) or third-generation (3/14) cephalosporins. One patient had metronidazole with a cephalosporin.

Table 2. Bacterial growth by species in 147 peritonsillar abscess specimens taken in individuals who had received antibiotics (Prior antibiotic) prior to culture, and those who had received none (No antibiotic)

Aerobic/facultative anaerobes	Prior antibiotic	No antibiotic
Beta-haemolytic streptococci		
Group A	12	26
Group C	11	7
Non A or C	7	7
Alpha-haemolytic streptococci	1	2
Staphylococcus aureus	5	1
Haemophilus influenza	3	2
Obligate anaerobes		
Bacteroides fragilis	0	1
Fusobacterium nucleatum	4	2
Eikenella corrodens	2	0
Prevotella species	0	1
Arcanobacterium	0	1
Not further classified	1	3
Mixed aerobic and anaerobic		
Oropharyngeal flora		
Heavy	11	
Moderate	16	
Scanty	11	
Not further classified	28	
No growth (n=1)		
No specimen (n=65)		

Tonsillectomy—Twenty-one percent of patients (47/213) received tonsillectomy, either acutely (14/47) or after an interval (33/47). Patients receiving acute tonsillectomy had refractory infection on clinical assessment. Seven patients had previously had a tonsillectomy. Patients receiving interval tonsillectomy gave a history of multiple episodes of tonsillitis or PTA. The recommendation for acute or interval tonsillectomy was made by the Consultant surgeon.

Length of stay—The average length of stay was 1.6 days. 143 patients (67%) had an inpatient stay of one night.

Complications—Post-tonsillectomy bleeds occurred in four patients (9%) of patients, all of whom underwent interval rather than acute tonsillectomy. These patients were re-admitted for observation and none returned to theatre. There was one death in a patient transferred from a provincial hospital with airway compromise. His transfer was delayed and by the time of presentation he had a near total occlusion of the oropharynx due to unilateral swelling caused by PTA. Immediately upon arrival at our hospital he was taken to theatre for an emergency tonsillectomy and airway management, but died while this was being performed due to airway complications.

Table 3. Comparison of bacterial growth by species in three audits over 3 decades in our unit

Year of audit	2006–8		1990–2		1981–4	
	No. of specimens (n=147)	% of specimens	No. of specimens (n=46)	% of specimens	No. of specimens (n=42)	% of specimens
Aerobic and facultative anaerobes						
B-haem strep						
A	38	25.9	12	26.1	5	11.9
C	18	12.2	6	13	10	23.8
Non A-C	14	9.5	10	21.7		
Alpha-haemolytic strep	3	2	4	8.7	18	42.9
S. aureus	6	4.1	2	4.3	1	2.4
H. influenza	5	3.4	1	2.2	1	2.4
Obligate anaerobes						
Bacteroides sp.	2	1.4	7	15	13	31
Fusobacterium	6	4.1	1	2.2	3	7.1

Discussion

Infectious peritonsillar abscess is common and complications can be life-threatening. It has previously been described as a condition of young, adult males. Whilst male preponderance was demonstrated in this study (120:93), the prevalence of peritonsillar infection in females has increased compared with a similar review in our unit a decade ago.[5]

International literature suggests that incidence is increasing in females also, with Risburg (2008)[6] and Hanna (2006)$_3$ reporting an almost 1:1 ratio. The age distribution in our study is similar to previous studies in our unit[4,5] with 30.5% patients in the modal age group of 15–19 years. Affected females were younger than males overall (27 vs 30 years) and they had an earlier peak incidence (15 vs 19 years). This finding has also been observed in other studies. Risberg et al (2008)[6] attributed the sex-based difference to hormonal maturation-based differences in immunological response.

The number of cases of peritonsillar infection in our population per year has approximately doubled since a previous audit was undertaken in our unit in 1990-1992 (28.7/100,000 compared with12.2/100,000), and approximately tripled since an audit 1981–1984 (9.8/100,000).[7] The increased number of presentations is only in part attributable to a growing population. It may also reflect increased rates of referral from primary care providers, together with lower thresholds for hospital admission. At present, we have no clear evidence to suggest that the true incidence is actually increasing, but this seems very likely

Variations in the microbiology of PTA over time have been described, and recently increased rates of anaerobes$_2$ and gram-negative aerobes$_8$ have been noted. The influence of pre-admission antibiotic therapy on subsequent culture results is unclear$_9$ Jokipii (1988)$_{10}$ found an increased incidence of Group A streptococci from specimens in patients treated with pre-admission antibiotics.

However, many studies have not found such differences[1,3,5] suggesting that pre-hospital antibiotics did not affect subsequent microbiology. In our study, 61% of patients did not receive antibiotics prior to acute admission (129/213). The distribution of culture results in this group was no different from those patients who had pre-hospital antibiotic therapy.

The establishment of a particular organism's role in the pathogenesis of PTA is difficult to determine, due to the fact that a wide variety of organisms are found in both healthy tonsils and in PTA specimens. A large number of specimens in our study were reported as containing normal oropharyngeal organisms (84/112). It is possible that these organisms exist in such a way as to be pathogenic in the individual patient, for example, in large numbers or as biofilm. Although biofilms have been identified in inflamed tonsils, the clinical significance is yet to be determined and further research is needed.[11]

The emergence of penicillin-resistant species has lead to the use of broad-spectrum antibiotics in other centres.[1] In our study, beta-haemolytic streptococci and obligate anaerobes predominated, with uniform sensitivity to penicillin.

Only 2.7% of specimens (6/147) contained organisms resistant to penicillin, mostly *Staphylococcus aureus.* This is unchanged compared with the previous audits in our

unit. Of note, penicillin has been shown to be effective clinically, even when culture results demonstrate resistant organisms,[3,5] and in our population, penicillin remains the empiric antibiotic treatment of choice.

Just over half of patients in our study presented with their first episode of peritonsillar infection, which supports the observation that PTA occurs most commonly in patients without a prior history of tonsillitis. In their evidence-based review of the treatment of PTA, Johnson et al (2003)[12] identified two level II studies reviewing rates of PTA recurrence and indications for tonsillectomy.

Those studies reported recurrence rates of 5% and 17%, which may be lower than our recurrence rate, (28.2%) because we included all cases of peritonsillar cellulitis as well as PTA in our study. In general, these patients had longer hospital stays than the overall PTA group (4.4 versus 1.6 days) and were more likely to undergo tonsillectomy (36% versus 21%).

Tonsillectomy is relatively uncommon (47/213) in patients with peritonsillar infection presenting to our unit and like elsewhere[13] is normally offered to patients with multiple episodes of tonsillitis or PTA. Some authors advocate quinsy tonsillectomy because of rapid resolution of symptoms, low complication rate andcost-effectiveness.[14]

While evacuation of pus and antimicrobial treatment remain the mainstay of treatment there is still debate in the literature over drainage techniques, with options including aspiration, incision and drainage or quinsy tonsillectomy.

We feel that our primary management protocol of incision and drainage under local anaesthetic combined with empiric antibiotic therapy, usually with penicillin, remains efficacious. Using this protocol, the duration of hospitalisation was low with over two-thirds staying only 1 night (143/213).

The re-admission rate was 5.8% (13/228), although no distinction was made between early recurrence and delayed resolution of a primary episode. Our length of stay compares favourably with those reported in other studies. Children with PTA in the southern district of Israel had an average stay of three days[15] as did patients in a study from Northern Ireland.[3] The cost implications of being able to safely reduce duration of hospitalisation is an important consideration in management of PTA.

Conclusion

Peritonsillar infection is a common and potentially life-threatening complication of tonsillitis. It seems likely that the incidence is increasing in our population, and it is still a disease of young adults, with similar male and female prevalence. PTA responds favourably to penicillin, combined with evacuation of pus by aspiration and incision and drainage.

Over the series of audits carried out in our unit, the mix of organisms causing PTA has not changed significantly; nor has their almost uniform sensitivity to penicillin. Our current recommendation is that patients presenting to their GPs with signs and symptoms of PTA should be commenced on oral penicillin and referred to hospital for aspiration and further management.

Competing interests: None.

Author information: Rachelle L Love, Otorhinolaryngology Registrar, Rob Allison, Consultant Otolaryngologist, Head and Neck Surgeon, ORL Department, Christchurch Public Hospital, Christchurch, Stephen T Chambers, Infectious Diseases Consultant, Christchurch Public Hospital, Christchurch

Correspondence: Dr Rachelle Love, ORL Department, Christchurch Public Hospital, Private Bag 4710, Christchurch, New Zealand. Fax: +64 (0)3 3640273; email rsalter@clear.net.nz

References:

1. Mitchelmore I, Prior A, Montgomery P, Tabaqchai S. Microbiological features and pathogenesis of peritonsillar abscesses. Eur J Clin Microbiol Infect Dis. 1995; 14: 870-7.
2. Gavriel H, Lazarovitch T, Pomortsev A, Eviatar E. Variations in the microbiology of peritonsillar abscess. Eur J Clin Microbiol Infect Dis. 2009; 28: 27-31.
3. Hanna BC, McMullan R, Gallagher G, Hedderwick S. The epidemiology of peritonsillar abscess disease in Northern Ireland. J of Infect. 2006; 52: 247-53.
4. Stegehuis HR, Schousboe M. Peritonsillar infection in Christchurch 1981-84. NZ Med J. 1986; 99:536-8.
5. Muir D, Papesh M, Allison R. Peritonsillar infection in Christchurch 1990-2: microbiology and management. NZ Med J. 1995; 108: 53-4.
6. Risberg S, Engfeldt P, Hugosson S. Incidence of peritonsillar abscess and relationship to age and gender: retrospective study. Scand J Infect Dis. 2008; 40(10): 792-6.
7. Statistics NZ: Sub national Population Projections
8. Megalamani S, Suria G, Manickam U, et al. Changing trends in bacteriology or peritonsillar abscess. J Laryngol Oto. 2008; 122: 928-30.
9. Rusan M, Klug T, Ovesen T. An overview of the microbiology of acute ear, nose and throat infections requiring hospitalization. Eur J Clin Microbio Infect Dis. 2009; 28: 243-51.
10. Jokippi A, Jokippi L, Sipila P, Jokinen K. Semiquantatative culture results and pathogenic significance of obligate anaerobes in peritonsillar abscesses. J Clin Microbiol. 1988; 26: 957-61.
11. Vlastarokos P, Nikolopoulos T, Maragoudakis P, et al. Biofilms in ear, nose and throat infections: how important are they? Laryngoscope 2007;117:668-673.
12. Johnson R, Stewart M, Wright C. An evidence-based review of the treatment of peritonsillar abscess. Otolaryngol Head Neck Surg. 2003;128:332-43.
13. Raut V, Yung M. Peritonsillar abscess: The rationale for interval tonsillectomy. ENT J. 2000;79(3):206-9.
14. Fagan J, Wormald P. Quinsy tonsillectomy or interval tonsillectomy: a prospective randomized trial. S Afr Med J. 1994;84:689-90.
15. Segal N, El-Saied S, Puterman M. Peritonsillar abscess in children in the southern district of Israel. Int J Pediatr Otorhinolaryn. 2009;73:1148-50.

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Acute medical admissions of older people from residential care facilities: are they appropriate?

H Carl Hanger, Valerie Fletcher, Andrew Sidwell

Abstract

Aim Acute medical admissions are increasing and potentially avoidable admissions (PAA) from residential care facilities (RCF) have been blamed. Estimates for the proportion of PAA from RCFs vary enormously in the literature. This study aimed to prospectively determine the level of PAA to a New Zealand hospital.

Methods Two cohorts of consecutive acute medical admissions of older (65 years and older) people from RCFs were reviewed (one retrospective and one prospective). Discharge domicile and survival at 6 months were determined for all patients. PAAs were determined by the treating general physician/geriatrician in the prospective cohort.

Results Admissions from RCF are a very heterogeneous group with a wide range of diagnoses, levels of dependency and outcomes. Most admissions (88%) from lower level care (LLC) were appropriate and most returned to their usual RCF on discharge. Patients from higher level care (HLC) patients had poorer outcomes (5/8 died in the acute hospital and only 1/8 alive at 6 months). Twenty percent of all RCF admissions were potentially avoidable and could have been managed in a different setting

Conclusions Most admissions from RCF were appropriate. However for a minority of admissions, other models of care within RCFs and community care are needed to provide alternative options of care. These may reduce some acute hospital admissions.

Acute medical admissions to hospital are increasing, both in New Zealand and globally.[1,2] Some postulated reasons for this increasing demand include an ageing population, more treatment options available, less continuity of care in primary sector, fragmented out of hours cover, higher public expectations, and aversion to risk management.[3]

Inappropriate admissions of older people to the acute hospital from residential care facilities (RCF) have also been blamed.[4] Estimates for these potentially avoidable admissions from institutional care vary enormously around the globe from 1–68%.[2,4–9]

These variations may reflect differences in health care settings and RCFs, access to primary care, and availability of community based options in different countries. The variation may also depend on how the terms “avoidable” or “inappropriate” are determined. Some studies have determined this retrospectively with case note review.[4,6] Others use diagnostic groupings to determine ambulatory sensitive admissions (ASA), which are diagnoses which could potentially be managed in a community setting.

Reliance on a single or primary diagnosis for ASA, may poorly reflect the care needs of frail older people. This is because the presenting problems are often multiple and

interact with comorbidities to cause delirium and functional decline.[10] Furthermore data on ASA is not routinely collected in New Zealand for those over 75 years old.

Acute hospital care can greatly benefit many people irrespective of age. This is equally important in older, frail or disabled people where prompt and accurate diagnosis and treatment is vital.[11] Most of the "old-old" population have a good quality of life, in spite of illness and disability,[12] and need access to acute medical care, just as for any other person residing in the community. Failure to admit this group to hospital when required would clearly be wrong.

However older people are also at risk from nosocomial complications[13] and patients from RCF are at high risk of functional decline in the acute hospital.[14] Some of these patients are very frail and may be better cared for in their familiar institution. Others may be on a dying trajectory and need good palliative care in their usual setting, rather than being uplifted to the acute hospital.[9,15,16] Many residents have clear preferences for their future treatment options,[17] and it is important these are discussed and documented.

Thus it is important to recognize the wide range of health care needs within RCF and tailor our acute treatments accordingly. The main aim of this study was to prospectively determine the level of "potentially avoidable" acute medical admissions to a New Zealand hospital. A second aim was to determine mortality in the 6 months after discharge, as a surrogate for those who may have already been on a dying trajectory at the time of admission.

Methods

Christchurch Hospital is the sole acute general hospital serving Christchurch and surrounding area, with a total population of 466,000 people of whom 62,500 (13.4%) are aged 65 years old or over).[18] Acute general medicine admits 11,500–12,000 people per year, with an average length of stay (LOS) of 4.4 days.

Two cohorts of institutional care admissions were reviewed:

- All older people (65 years and over) admitted to any of the 12 General Medical (GM) teams over a 2-week period (26/7/2008–8/8/2008) were collected prospectively as part of another audit.[19] Patients from RCF were identified from this cohort. Their case notes were reviewed retrospectively by one geriatrician (HCH) who was not involved in their clinical care. This review was to determine reasons for admission, discharge diagnoses, appropriateness of admission and to collect outcomes for domicile at discharge and mortality. Appropriateness of the admission was retrospectively assessed (by the sole reviewer) against the question "Could this person have been managed in the community with existing supports, based on admission and discharge diagnoses, as well as the interventions delivered in hospital".
- All admissions from RCF (aged 65 and over) who were admitted under two of the GM teams were prospectively collected (June–August 2009 inclusive). These GM teams were chosen because each of the 3 consultant physicians involved were also geriatricians, two as community geriatricians and so were aware of community options available. During the acute admission, the treating physician judged whether each admission was avoidable or not. This clinical judgement was made within the first 1–2 days of admission (to avoid bias from additional results and complications) and was based on the severity of presenting illness, need for hospital based investigations and the answer to the question "Could this person have been managed in the community with currently available supports, based on admission diagnoses and information available at that time?". If the admission was deemed avoidable, the reasons why were collated. Discharge diagnoses and discharge outcomes were collected prospectively during the admission.

Community supports within our DHB during both these periods were well developed with a wide variety of community investigation and treatment options available to general practitioners.[25–27,33]

For both cohorts, mortality during the subsequent 6 months was ascertained using CDHB computerised patient databases which are linked to the national databases.

Results

Retrospective cohort—During the 2-week period, 259 older patients were admitted, of whom 47 (18.1%) were from RCF. Forty were from rest home or lower level care (LLC) and seven from hospital or higher level care (HLC). The mean age of this cohort was 84 years (62% female) and mean (median) LOS was 6.3 (4) days. Four (9%) patients died during the admission- three from pneumonia and one from a stroke.

The reasons for admission (as given by referring practitioner) are shown in Table 1. At least 20 residents (50% of LLC) had a significant change in physical or cognitive functioning and just over one-quarter were for shortness of breath. During the index hospital stay, six patients (five LLC and one HLC) were changed to palliative management of whom two died in hospital and all had died by 90 days.

Retrospectively, four (8.5%) were thought to be potentially avoidable admissions (PAA) as they could have been managed in a different setting (with a further nine (19%) possibly avoidable).

Discharge and survival outcomes after the index admission are shown in Table 2.

Table 1. Reasons for admission (retrospective cohort) as documented on admission notes (some patients had more than one reason)

Reason for Admission	Lower Level Care (N=40)	Higher Level Care (N=7)	Total (N=47)
Shortness of breath	12		12
Delirium	9		9
Fall or altered mobility	6		6
Febrile	5		5
“Unwell”	3		3
Stroke	2		2
Reduced level of consciousness	5	3	8
Palliation		1	1
Hypotension		1	1
Severe anaemia		1	1
Vomiting		1	1
Congestive cardiac failure	1		1
Cellulitis	1		1
Epigastric or chest pain	2		2

Table 2. Discharge domicile and mortality outcomes in retrospective cohort

Variables		Lower Level Care (N=40)	Higher Level Care (N=7)
At discharge	Return to same domicile	29	7
	New Higher Level Care	7	
Mortality	At discharge	4 (10%)	0 (0%)
	At 1 month	8 (20%)	2 (29%)
	At 6 months	13 (32%)	4 (67%)

Prospective cohort—There were 50 older patients admitted from RCF of whom 34 (68%) were from LLC, 8 (16%) from HLC and 8 (16%) from specialist dementia rest homes (D). The mean age was 84.7 years and 66% were female. The mean (median, interquartile range) LOS was 9.5 (5, 2–12) days and there was one outlier staying 71 days in hospital.

Eleven (22%) patients died during the admission—four from devastating strokes, four from pneumonia or sepsis, one from acute myocardial infarction, one from progressive renal failure and one sudden death from unknown causes in a very frail older person. At least three of the deaths were in patients who were already deteriorating and expected to die.

The reasons for admission (as given by referring practitioner) varied and are shown in Table 3. Discharge outcomes (domicile and survival) are shown in Table 4. Eighteen of the 22 who returned to LLC after the hospital admission were still residing in LLC at 6 months.

Prospectively, the treating physician thought that 10 (20%) were potentially avoidable admissions (PAA) as they could have been managed in a different setting (see Table 5).

Table 3. Reasons for admission (prospective cohort) as documented at time of admission (some patients had more than one reason)

Reason for Admission	Lower Level Care (N=34)	Higher Level Care (N=8)	Dementia Care (N=8)	Total (N=50)
Fall, or altered mobility	11	1	1	13
Shortness of breath or pneumonia	9	3	1	9
Delirium	5		1	6
Stroke	4			4
Reduced level of consciousness	2	2	3	7
Febrile or Sepsis	2		2	4
“Unwell”	2	1		3
Abdominal pain	2	1		3
Chest pain	1			1
Other pain	2			2
Diarrhea	1			1
Epistaxis	1			1
Cellulitis	1			1
Suicidal/ Severe depression	1			1

Table 4. Discharge domicile and mortality outcomes in prospective cohort

Variables		Lower Level Care (N=34)	Higher Level Care (N=8)	Dementia care (N=8)
Domicile at Discharge	Return to same domicile	22	3	2
	New HLC	7	–	5
Mortality	At discharge	5 (15%)	5 (63%)	1 (13%)
	1 month	8 (24%)	5 (63%)	1 (13%)
	6 months	15 (44%)	7 (88%)	4 (50%)

Table 5. Potentially avoidable admissions (with possible reasons) in prospective cohort 2009

Admitted from	Discharge destination	Reason for admission	Reasons why it was thought admission avoidable
HLC**	Died	unwell ?cause	Advanced vascular dementia, poor quality of life prior. Death could have been managed in usual residence
HLC	HLC	Funny turn	From HLC, not acutely unwell- could have been reviewed by after hours GP service rather than admission
HLC	HLC	abdominal pain and possible sepsis	Urinary retention could have been managed in HLC
HLC	Died	pneumonia	Terminal illness could have been managed in HLC
HLC	HLC	pneumonia	Change of antibiotics for pneumonia could have been managed in HLC
LLC*	LLC	pneumonia	Only mildly unwell, mobility not compromised- could have been managed in RH
LLC	LLC	delirium, depression	Urgent domiciliary assessment by Older Persons Health medical or psychiatry team might have been better
LLC	LLC	SOB	Moderate pleural effusion with minimal symptoms -could have been investigated as outpatient
LLC	Died	unwell ?cause	Assessment for higher level care could have been managed in community (death in hospital was unexpected)
Dementia	Dementia	seizures	Myoclonic jerks in person with severe dementia- an ongoing problem which could have been managed in community

* LLC= Lower Level Care

**HLC=Higher Level Care

Discussion

Acute medical admissions from institutional care are a heterogeneous group. Most admission reasons are for sudden changes in function (physical and/or cognitive) or for infections. A small number of patients were dying, but most patients, particularly from LLC, were able to return to their usual residence and were alive at six months. Only a minority (20%) of these acute admissions from RCF were PAA.

Whilst recognising some PAA, the majority of admissions (80%) were appropriate, as judged by the treating hospital physician. Most older people in RCF are disabled and/or have multiple comorbidities, yet many still rate their own quality of life as good to excellent. For these people, they need prompt accurate diagnosis and intervention when their health deteriorates acutely to prevent further disability.[20]

Admission to the acute hospital is often appropriate in these circumstances, particularly if the change in health is sudden or severe, or if there is diagnostic uncertainty, or need for frequent monitoring. Many of the LLC residents in our study appeared to be in this category. Failure to act on this changed health status would be inappropriate and may reflect poor care.[21]

These patients need the services of the acute hospital, but at the same time are at risk of complications and further functional decline.[13–15,22] Thus we need to ensure the hospital system manages these patients in a manner which preserves their function[23] and facilitates return to their usual residence as soon as practical. The environment and practices of many acute hospital settings have not adapted to the needs of frailer older people and this may be one reason why some older people deteriorate in hospital.[13–15,22]

Furthermore, just over half of our sample of LLC presented with a sudden decline in functioning (physical and/or cognitive). Wherever these patients are managed, they need rehabilitation from allied health staff (AHS) and others, to both prevent further functional decline as well as restore as much function as possible. Active and timely rehabilitation is difficult to access in RCF at present.

One strategy may be to build in better and more rapid intervention from existing community teams such as Older Persons Health medical and psychiatric services. An alternative is creating the ability to temporarily boost access to existing nursing and AHS in RCFs, with clear rehabilitation plans. Both strategies would require careful monitoring to ensure the rehabilitation is focused on those with potential to benefit.

In this study, PAA could be divided into (1) patients on a dying trajectory who may have benefited from palliation of symptoms in their usual residence and (2) patients who were not dying, and whose symptoms could have been managed in primary care or an outpatient setting.

PAA are an issue across the full age spectrum of acute admissions, not just older patients.[1,5,24] In order to manage PAAs differently, admitting clinicians must have viable alternatives in the community.[7,8] to deal with the sudden changes in health status.

Options may include primary care empowerment to investigate and manage conditions previously considered the domain of hospital medicine[25,26] and hospital at home models.[22] These should be available to patients in RCF as well as those living at home. In Christchurch, we already have a strong primary care service and outreach programmes with protocols for managing many conditions in the community.[25–27,33] This might account for the lower proportion of PAAs from institutions in this study compared to others,[4,6] Continuity of care and access to general practitioners (GP) after hours is also important in reducing PAAs from RCF settings.[1,24,28]

Whilst we did not collect data on the referring clinician, it is not uncommon for an older person to be sent from LLC to acute hospital without GP involvement. Active and timely GP involvement is critical to managing these unwell frail patients in the community.[1,3,24,29] Access to after hours GP care is particularly important, but is not uniformly available.

Patients with PAA who are already on a dying trajectory are an important group to identify and manage differently.[15] Their palliation needs are quite different to those patients needing acute treatments for reversible illness.[9]

Acute medical wards are unlikely to be the best place to provide palliation, yet many still die in acute hospitals.[9,30] In one USA study, the majority of residents were able to state a clear preference for place of death, with most preferring to die in their continuing care facility.[17]

Greater emphasis on symptom control in their usual environment seems preferable for both patient[31] and the health system, yet not so easy to enact. Anticipatory thinking with advanced care planning (ACP) has been shown to be helpful,[15,31] but a recent NZ study showed a reluctance of staff to discuss them with patients or family.[29]

Advanced care planning in RCF is not a single discussion on admission but has many different elements including (but not limited to) initiation, scope, follow up and documentation.[32] It requires time and continuity for GPs and others to have such important and personal discussions,[28] and therefore funding changes may be necessary to facilitate this change in practice. HLC patients had poorer outcomes in our cohort and they may be the better group to initially target for these management plans.

A wider systems based approach to reduce PAA may be required. This may include some or all of the following options: alternative models of providing medical care in institutions, funding incentives for institutions to manage sicker patients, and greater access to specialist input (palliative care or geriatric medicine) in the institution.[28,29]

This study has several weaknesses. It is from one South Island urban center, and the assessment of PAA was done by single reviewers based on clinical judgement, rather than formal explicit criteria. In mitigation, there is a lack of literature consensus of what defines a PAA.[2,4–6,8] Furthermore, in the prospective cohort, the treating physician could assess not just the admitting syndromes/diagnoses, but also the severity of the presentation, which is hidden from a retrospective case note review.[2]

We may have underestimated the PAAs, as once the person is in hospital with the benefit of some results, it may be harder to say an admission was inappropriate. Ideally the best person to identify PAA is the patient's usual GP, who knows the patient and their preferences, and the services available. However at the time of the decision to admit, such as after hours, the usual GP may not be available.

Involvement of GPs, with their knowledge of available community services could have been beneficial in assessing appropriateness or not. In mitigation, all three of the assessing physicians are also geriatricians and two of them work extensively in the community.

Our estimates of PAA were dependent on what is available currently. Inevitably PAA would be higher if more or alternative community supports were available. In our community, there are already some well developed community based options available[25–27,33] which may result in lower estimates of PAAs, than what might occur in other New Zealand centres.

Strengths of this study include prospective patient selection, both cohorts show similar results (both for PAA and mortality), the admissions were from multiple institutions,[29] and there was complete follow up.

In summary, admissions from institutional care are heterogeneous and most are appropriate and should continue. A minority of admissions were PAA and a smaller number were dying. Alternative clinical and funding models of care within RCFs are required to improve the care of the PAA group.

Competing interests: None.

Author information: H Carl Hanger [1]; Valerie Fletcher [1,2]; Andrew Sidwell [1,2]; [1]Geriatrician, Older Persons Health Specialist Service

[2] General Physician, Department of General Medicine

Canterbury District Health Board, Christchurch

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Correspondence: Dr HC Hanger, Older Persons Health Specialist Service, The Princess Margaret Hospital, PO Box 800, Christchurch, New Zealand. Fax: +4 (0)3 3377823; email: carl.hanger@cdhb.govt.nz

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References:

1. Gillam S. Rising hospital admissions. BMJ. 2010 Feb 2;340:c636. doi: 10.1136/bmj.c636.
2. Hider P, O'Hagan J, Bidwell S, Kirk R. The rise in acute medical admissions. Aust N Z J Med 2000;30:252-260.
3. Dunstan EJ. Rising hospital admissions. Fuelled by risk avoidance. BMJ. 2010 Feb 23;340:c1048. doi: 10.1136/bmj.c1048.
4. Saliba D, Kington R, Buchanan J et al. Appropriateness of the decision to transfer nursing facility residents to the hospital. J Am Ger Soc 2000;48:154-163.
5. Banham D, Woollacott T, Gray J, et al. Recognising potential for preventing hospitalization. Aust Health Rev 2010;34:116-122.
6. Ouslander JG, Lamb G, Perloe M et al. Potentially avoidable hospitalizations of nursing home residents: frequency, causes and costs J Am Ger Soc 2010;58:627-635.
7. Wyman J, Hazzard WR. Preventing Avoidable hospitalizations of nursing home residents: a multipronged approach to a perennial problem. J Am Ger Soc 2010;58:760-761.
8. Finn JC, Flicker L, MacKenzie E, et al. Interface between residential aged care facilities and a teaching hospital emergency department in Western Australia. Med J Aust 2006;184:432-435.
9. Partridge J. Why are nursing home residents sent in to hospital to die? Postgrad Med J 2008;84:281.
10. Wilkinson TJ, Sainsbury R. Diagnosis related groups based funding and medical care of the elderly: a form of elder abuse? NZ Med J 1995 Feb 22;108(994):63-5.
11. Fairweather DS, Campbell AJ. Diagnostic accuracy. The effects of multiple aetiology and the degradation of information in old age. J Roy Coll Phys Lond 1991 Apr;25(2):105-10.
12. Collerton J, Davies K, Jagger C, et al. Health and disease in 85 year olds: baseline findings from Newcastle 85+ cohort study. Br Med J 2010;340:86.
13. Creditor MC. Hazards of hospitalization of the elderly. Ann Intern Med 1993 Feb 1;118(3):219-23.

14. Sager MA, Rudberg MA, Jalaluddin M, et al. Hospital admission risk profile (HARP): identifying older patients at risk for functional decline following acute medical illness and hospitalization. J Am Ger Soc 1996;44(3):251-7.
15. Caplan GA, Meller A, Squires B, et al. Advanced care planning and hospital in the nursing home. Age Ageing 2006;35:581-585.
16. Ahearn DJ, Jackson TB, McIlmoyle J, Weatherburn AJ. Improving end of life care for nursing home residents: an analysis of hospital mortality and readmission rates. Postgrad Med J 2010;86:131-135.
17. Hays JC, Galanos AN, Palmer TA, et al. Preference for Place of Death in a continuing care retirement community. The Gerontologist 2001;41:123-128.
18. Statistics New Zealand Census 2006 Data. http://www.stats.govt.nz/reports/analytical-reports/new-zealands-65-plus-population.aspx# (accessed 1/5/2010)
19. Rodwell J, Fletcher V, Hughes R. How well is cognitive function documented by medical staff in the over 65 age group at the time of acute medical admission? N Z Med J 2010:123:1317. http://www.nzma.org.nz/journal/123-1317/4182
20. Geriatric Services in general hospitals. Australian and New Zealand Society for Geriatric Medicine. Aust J Ageing 2010;29:43-46.
21. Medical Care for people in residential aged care facilities. Australian and New Zealand Society for Geriatric Medicine. http://www.anzsgm.org/pdfdocs/position_statements/PositionStatementNo09.pdf
22. Caplan GA, Coconis J, Board N, et al. Does home treatment affect delirium? A randomised controlled trial of rehabilitation of elderly and care at home or usual treatment (the REACH-OUT trial). Age Ageing 2006;35:53-60.
23. Inouye SK, Bogardus ST, Charpentier PA, et al. A multicomponent intervention to prevent delirium in hospitalised older patients. NEJM 1999;340:669-676.
24. Spence D. Admission of failure. Br Med J 2010;340:c1076.
25. Corwin P, Toop L, McGeoch G, et al. Randomised controlled trial of intravenous antibiotic treatment for cellulitis at home compared with hospital. Br Med J 2005 Jan 15;330(7483):129.
26. Richards DA, Toop LJ, Epton MJ, et al. Home management of mild to moderately severe community-acquired pneumonia: a randomised controlled trial. Med J Aust 2005 Sep 5;183(5):235-8.
27. Canterbury Clinical Network. http://www.ccnweb.org.nz/ (accessed 26/7/10)
28. Donald IP, Gladman J, Conroy S, et al. Care home medicine in the UK-in from the cold. Age Ageing 2008;37:618-620.
29. Sankaran S, Kenealy T, Adair A, et al. A complex intervention to support ‘rest home” care: a pilot study. N Z Med J 2010:123:1308 http://www.nzma.org.nz/journal/123-1308/3948
30. Houttekier D, Cohen J, Bilsen J, et al. Place of death of older people with dementia. A study in five European countries. J Am Ger Soc 2010;58:751-756.
31. Detering KM, Hancock AD, Reade MC, Silvester W. The impact of advanced care planning on end of life care in elderly patients: randomised controlled trial. Br Med J 2010;340:c1345.
32. Shanley C, Whitmore E, Khoo A, et al. Understanding how advanced care planning is approached in the residential care setting: A continuum model of practice as an explanatory device. Aust J Ageing 2009;28:211-215.
33. Canterbury Initiative. http://www.canterburyinitiative.org.nz/ (accessed 1/3/11).

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Campylobacteriosis rates show age-related static bimodal and seasonality trends

Warrick Nelson, Ben Harris

Abstract

Aim Campylobacteriosis is highly characterised by a strongly seasonal rate of incidence. Age is also known to be a risk factor for sporadic campylobacteriosis, but little has been done to quantify age-related rates of campylobacteriosis. This study investigates age-related incidence across countries and up to 12 years of data, as well as differences in seasonality within age groups.

Methods Graphical and statistical analysis of officially collected campylobacteriosis reports from three countries available from official websites.

Results For Australia, New Zealand and Canada, rates of campylobacteriosis show marked peaks at <4 years and 20–29 year age bands. These peaks indicate that stable age-related factors impact on campylobacteriosis epidemiology in all three countries. Seasonality is expressed differently across these age bands, and in years of extremes of incidence.

Conclusion Campylobacteriosis is highly seasonal, but overlying this is a stable age-related pattern of incidence, with two peaks approximately 20 years apart. Highest seasonal differences occur with ages between the two peaks.

Campylobacteriosis is highly prevalent in New Zealand, with rates significantly above rates in other temperate countries.[1] Notwithstanding the recent marked drop in campylobacteriosis reported in New Zealand coinciding with implementation of a chicken health scheme,[2] other trends in campylobacteriosis rates indicate factors beyond chicken meat at play.

A strong seasonal pattern, typically an early winter low and an early summer high rate of incidence, is common to New Zealand and other temperate countries. At an annual level, there is a clear correlation between increased consumption of chicken meat and the rates of reported campylobacteriosis,[3] seemingly confirming that the route to humans is via consumption of contaminated chicken, particularly as the incidence of *Campylobacter* in chickens has a similar seasonal pattern. However, this correlation disappears on considering shorter time intervals.

Specifically, chicken consumption is not seasonal and the seasonal peak in chicken contamination typically occurs after the human peak, leading us to suggest a domestic fly-related epidemiology via fomites and fingers to food.[4] Difficulty with the chicken/human link was also found in a five year German study where the human seasonal incidence preceded that of chickens.[5]

Regional and seasonal variations in campylobacteriosis have been reported through New Zealand, particularly differing seasonal incidence patterns with changing latitude.[6] Similar differences have been reported across Northern Europe.[7]

Age-related trends have also been noted, and an acquired immunity has been suggested as a reason for the rapid drop-off in rates of infection with age,[8] supported by observation that children older than six months tend not to develop diarrhoea except on first exposure.[9] Seasonal peaks and higher incidence in children under five years has also been noted in the United Kingdom,[10–12]

In a rural/urban comparison in Canada, differences in rates of infection were noted not only for babies, but again at adult-onset,[13] although no exploration of this observation was made. A similar twin age-related peak in rates in New Zealand has been noted[14] and this investigation aims to determine the reality and stability of this observation, and to attempt to elucidate an epidemiological profile to account for it.

Methods

Epidemiological data were obtained primarily from on-line resources, www.nzpho.org.nz for New Zealand, http://www9.health.gov.au for Australia and http://dsol-smed.hc-sc.gc.ca for Canada, accessed November 2009. In all three countries, campylobacteriosis rates (case numbers per 100 000 population) are provided. Data is offered in different formats and broken into various categories.

Data was analysed to determine trends in rates over years using rates of reported illness from the on-line resources that are expected to have been normalised for population changes during the reporting period. The official data reports use different demographic fractions, especially the age bands. These have been normalised where comparisons required it, but otherwise are reported in their native format. Years of data availability also vary across countries. We used the full 12-year period of 1997–2008 for New Zealand, 6 years, 2003–2008 for Australia, and 7 years 1998–2004 for Canada. Data is graphed to show mean, high and low values for the age bands and year periods covered.

Further data was requested from the New Zealand Public Health Observatory's EpiSurv database in more detail than that available through their on-line query tool. Cases by age class by month data were obtained for the years 1997-2008. The Gini coefficient, a measure of inequality of distribution, by age band average over the 12 years of data was used as a measure of seasonality.[15]

Privacy and ethics. No data source offers information identifiable to an individual.

Results

Reported campylobacteriosis rates are age-dependent and can vary quite markedly across the age bands. As noted in previous studies, very young children typically exhibit high rates of campylobacteriosis, and this is clearly evident in Figure 1 across all three countries.

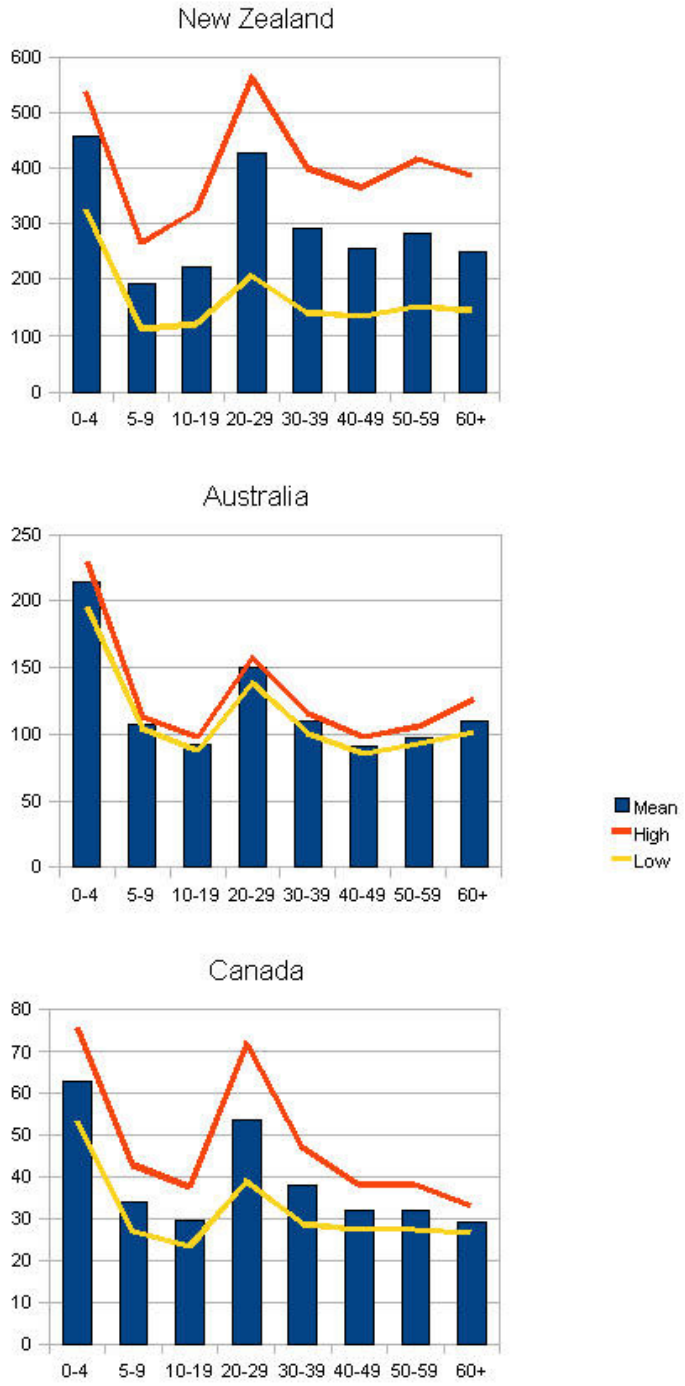
A Canadian study indicated, but did not explore further, a second peak at adult-onset.[13] This is also clearly evident in Figure 1 across all three countries. Furthermore, these peaks in reported rates are approximately 200% and 150% for 0–4 years and 20–29 years bands respectively above the background rates for other age bands in each country, even though those background rates are markedly dissimilar.

Australian rates are less than half those of New Zealand, and Canadian rates a third lower again. Clearly the presence of two peaks in campylobacteriosis rates of infection approximately 20 years apart is not an artifact of the very high New Zealand incidence. Even picking the highest and lowest rate for each age group across the years represented does not change the marked presence of these two age-related peaks.

The stability of these age-related peaks in incidence across many years of data and three countries strongly indicates a difference in epidemiology is occurring. Figure 2 shows detail of the stability of these peaks for New Zealand. Note that this graph

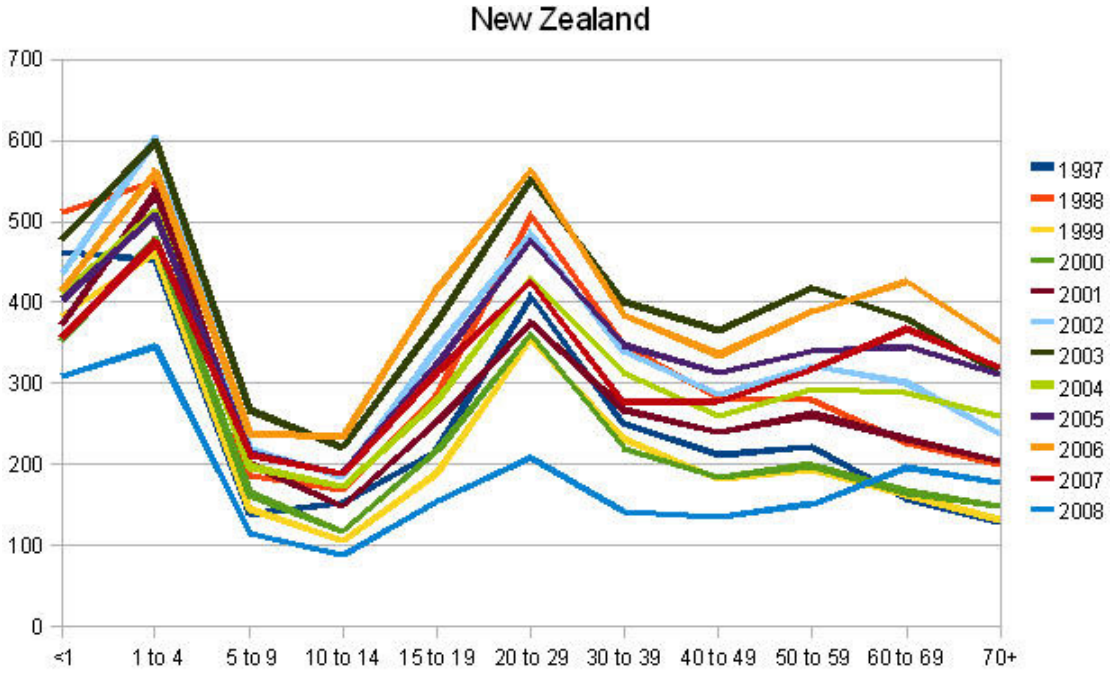
covers a 12-year period, clearly indicating an age-related impact rather than a specific group of people more or less susceptible to campylobacteriosis.

Figure 1. Campylobacteriosis rates (cases per 100,000 population) at different age bands for countries as marked. New Zealand n=12 years 1997–2008; Australia n=6 years 2003–2008; Canada n=7 years 1998–2004



Note: High is the highest rate over the period and low the lowest, regardless of year.

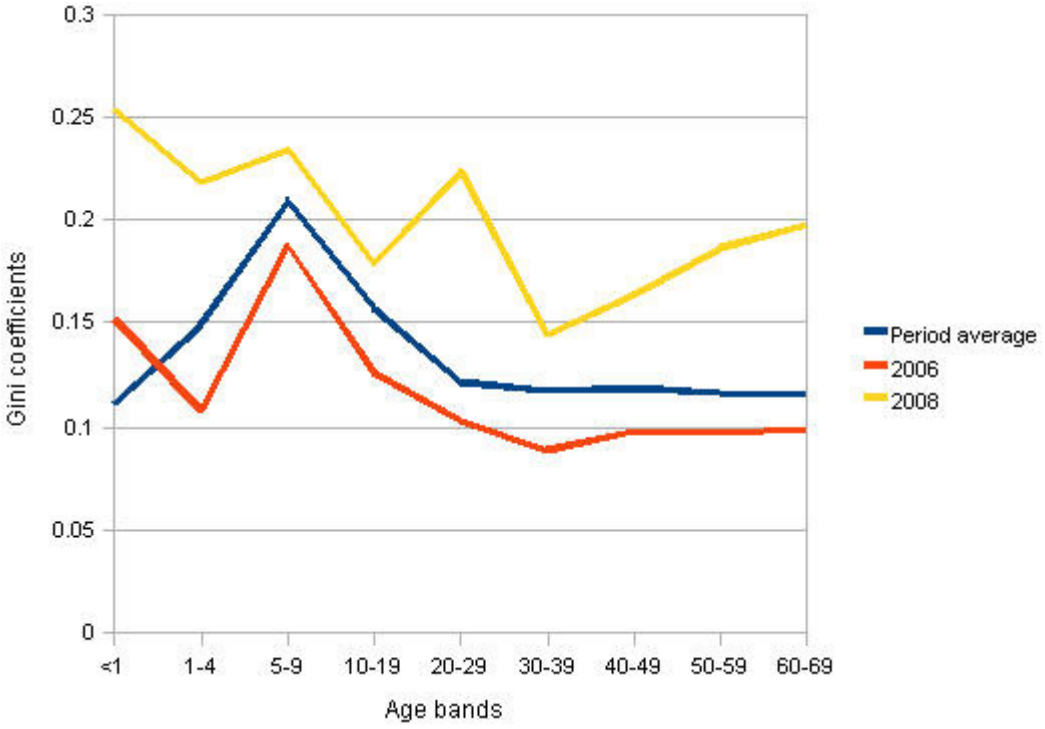
Figure 2. Campylobacteriosis rates (cases per 100,000) in New Zealand at age bands per year reported for the years 1997 to 2008



On a population basis, rates increase in the 1–4 year age band over the <1 year band (data not shown in Figure 1 as these have been amalgamated, but are available separately for Canada and New Zealand). Interestingly, the seasonality impact between these two age groups is markedly different, with the <1 age group showing the least seasonal impact as indicated by the low Gini coefficient in Figure 3. This group is presumably the most susceptible to any *Campylobacter* exposure and also most likely to be reported.

Although the 5–9 year age group has a low incidence, they typically exhibit the highest seasonality. Of interest too is that the pattern of seasonal impact in New Zealand changes dramatically for the 2008 year, and that the highest and lowest rates of incidence years in this series (2006 and 2008) exhibit the lowest and highest seasonal differences respectively across all age groups.

Figure 3. Gini coefficient for monthly New Zealand data averaged over the years 1997–2008, and for the years 2006 (highest incidence year) and 2008 (lowest incidence year) separately



Note: This coefficient approaches zero as seasonality decreases. It is a measure of the inequality of a distribution, a value of 0 expressing total equality and a value of 1 maximal inequality. It can be multiplied by 100 to range between 0 and 100.

Discussion

These data do not appear to indicate the presence of any ongoing acquired immunity with age as may be expected after an early childhood primary infection because of this clearly age-specific static 2nd peak, although short-term immunity can play a role in campylobacteriosis disease expression.[8,11,16]

As *Campylobacter* is relatively ubiquitous in the environment (in animals, wild birds, water and food including poultry), humans can expect relatively frequent ongoing exposures to it regardless of age. This would then be expected to provide an ongoing immunity booster effect to any earlier childhood exposure infections and provide an ongoing intermittently boosted acquired immunity – but it does not for the 20–29 year age group only. It seems likely there must be another explanation rather than loss of acquired immunity. Similarly the absence of a third peak another 20 years later, or shorter for compromised immunity with age, further detracts from the likelihood of a loss of acquired immunity explanation.

Acquired immunity is interesting, although the antibody literature is hard to interpret because of differing methods.[8] Antibodies appear to drop off over a 12-month period leading to the observation that the presence of antibodies indicates a *Campylobacter*

challenge in the preceding year. If this is a general population exposure, it is hard to suggest a reason for the marked increase in reporting rates in early adulthood, and how an immunity reaction could impact on changing seasonality expression for different age bands. Why would frequent exposure, suggested by the antibody studies, result in different disease expression rates for different age bands?

An epidemiological explanation for the lower rates of <1 year and higher rates in the 1-4 age group is easily made. Babies are likely to have a low exposure level, but this rises as they reach teething and become mobile. The subsequent reduction in rates for higher age groups could be explained by an acquired immunity reaction if it was not for this second peak in rates only in the early adult years.

If chicken-consumption is truly the source for most cases, why did rates generally decrease in 2007, before the chicken health scheme began, and what could be the link between chicken-consumption and age, regardless of sex? Further, it is difficult to reconcile chicken consumption with the marked and long-standing reduced seasonality of the 5-9 year band (high Gini coefficient) compared to the other age groups.

The bimodal peaks in rates demonstrated here combined with the age-related differences in seasonality of cases is not readily explained by exposure to chicken products nor to current immunological observations. It is possible that the second 20-29 year old peak reflects high rates of primary infection across this age band, but again it is difficult to provide an explanation that would increase exposure to just them and not any other age band, for instance teenagers.

The 2006 year coincides with an unusually high New Zealand rate of cases in autumn/winter, subsequently found to be associated with a rare sequence type.[17]

Conclusion

The epidemiology of campylobacteriosis is complex. The presence of distinct differences in rates of infection for age groups that remain stable over long periods of time indicate a substantial underlying factor. Further, seasonal factors clearly play different roles at different ages, and the markedly different seasonality expressed in the New Zealand peak year (2006) and lowest year (2008) suggest scope for further investigation. Until specific exposure and susceptibility explanations can be made to account for these age-related differences, the popular assumption that poultry is the primary source for human campylobacteriosis is perhaps rather simplistic.

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Author information: Warrick Nelson, Research and Management Consultant, 888 Management Ltd, Christchurch; Ben Harris, Medical Laboratory Scientist, Southern Community Laboratories, Christchurch

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Correspondence: Warrick Nelson, 888 Management Ltd, 6 Blowers Place, Christchurch, New Zealand. Fax: +64 (0)3 332 9350; email: warrick.nelson@gmail.com

References:

1. Nylen G, Dunstan F, Palmer SR, et al. The seasonal distribution of Campylobacter infection in nine European countries and New Zealand. Epidemiol Infect. 2002;128:383-90.
2. Müllner P, Spencer SEF, Wilson DJ, et al. Assigning the source of human campylobacteriosis in New Zealand: a comparative genetic and epidemiological approach. Infect Genet Evol. 2009;9:1311-9.
3. Baker MG, Sneyd E, Wilson NA. Is the major increase in notified campylobacteriosis in New Zealand real? Epidemiol. Infect. 2006;135:163-70.
4. Nelson W, Harris B. Flies, fingers, fomites, and food. campylobacteriosis in New Zealand - food-associated rather than food-borne. N Z Med J. 2006;119:U2128. http://www.nzmj.com/journal/119-1240/2128/content.pdf
5. Hartnack S, Doherr MG, Alter T, et al. Campylobacter monitoring in German broiler flocks: an explorative time series analysis. Zoonoses Public Health. 2009;56:117-28.
6. Hearnden M, Skelly C, Eyles R, Weinstein P. The regionality of campylobacteriosis seasonality in New Zealand. Int J Environ Health Res. 2003;13:337-48.
7. Jore S, Viljugrein H, Brun E, et al. Trends in Campylobacter incidence in broilers and humans in six European countries, 1997-2007. Prev. Vet. Med. 2010; 93:33-41.
8. Havelaar AH, van Pelt W, Ang CW, et al. Immunity to Campylobacter: its role in risk assessment and epidemiology. Crit Rev Microbiol. 2009;35:1-22.
9. Rao MR, Naficy AB, Savarino SJ, et al. Pathogenicity and convalescent excretion of Campylobacter in rural Egyptian children. Am. J. Epidemiol. 2001;154:166-73.
10. Louis VR, Gillespie IA, O'Brien SJ, et al. Temperature-driven Campylobacter seasonality in England and Wales. Appl. Environ. Microbiol. 2005;71:85-92.
11. Strachan NJC, Gormley FJ, Rotariu O, et al. Attribution of Campylobacter infections in northeast Scotland to specific sources by use of multilocus sequence typing. J. Infect. Dis. 2009;199:1205-8.
12. French NP, Midwinter A, Holland B, et al. Molecular epidemiology of Campylobacter jejuni isolates from wild-bird fecal material in children's playgrounds. Appl. Environ. Microbiol. 2009;75:779-83.
13. Green CG, Krause DO, Wylie JL. Spatial analysis of Campylobacter infection in the Canadian province of Manitoba. Int J Health Geogr. 2006;5:2.
14. Nelson W, Harris B. Is chicken meat the most important source of human Campylobacter infection in New Zealand? N Z J Medical Laboratory Science. 2007;611:36-41.
15. Wessa P. Free statistics software, office for research development and education, version 1.1.23-r4, www.wessa.net
16. Miller G, Dunn GM, Reid TMS, Ogden ID, Strachan NJC. Does age acquired immunity confer selective protection to common serotypes of Campylobacter jejuni? BMC Infect. Dis. 2005;5:66.
17. McTavish SM, Pope CE, Nicol C, et al. Wide geographical distribution of internationally rare Campylobacter clones within New Zealand. Epidemiol. Infect. 2008;136:1244-52.

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Auckland City Hospital's Ortho-Geriatric Service: an audit of patients aged over 65 with fractured neck of femur

Lucy Fergus, Greer Cutfield, Roger Harris

Abstract

Background The process of care of older patients with fractured neck of femur at Auckland City Hospital has recently changed with selected patients "fast-tracked" as soon as possible postoperatively to a specialised Older People's Health (OPH) ward.

Aims The aims of this study were: to evaluate patient characteristics; to analyse process of care; to compare outcomes in those "fast-tracked" patients with those receiving usual care; and to compare this information with previous data from Auckland City Hospital and other centres in New Zealand.

Method Prospective case record audit of patients with fractured neck of femur aged 65 years and over admitted under Orthopaedics over a 4-month period.

Results 115 patients were audited; mean age was 84 years, 77% were female. Inpatient mortality was 5%.

Twenty-four percent of patients had surgery within 24 hours of admission. Of those who did not have surgery within 24 hours, 39% were awaiting operating theatre availability. Median overall length of stay (LOS) was 27 days. Eighty-four percent of patients were transferred to Older Peoples Health. Considering all patients, 70% of those living at home pre-fracture returned home on discharge. However, only 26% of those in Rest Home returned to Rest Home. Overall, 35% of patients were discharged to a higher level of care. Forty-four percent of the group were able to walk unaided prior to hip fracture, but only 1% on discharge. Forty-three patients were "fast-tracked" to Older Peoples Health. Their median overall LOS was 23 days compared to 28 days for those receiving usual care. This was due to the shorter time in Orthopaedics. Thirty-three percent of this group went to a higher level of care on discharge compared to 35% in the group that received usual care.

Conclusions Many patients experience a delay to surgery for non-medical reasons. The percentage transferred to Older Peoples Health is high. Fast-tracking to Older Peoples Health shortens overall length of stay due to fewer days in Orthopaedics. Many patients require a higher level of care after hip fracture, particularly if already resident in Rest Home. Demographics and inpatient mortality are comparable, but total length of stay is longer than similar New Zealand studies due to a longer length of stay in Older Peoples Health. Review of previous data from Auckland City Hospital and from other New Zealand centres shows significant variability in process of care for older patients with hip fracture.

Hip fracture is an important cause of mortality and morbidity in older people. The New Zealand Health Information Service (NZHIS) report on hip fracture services in NZ hospitals 1999–2000 showed that 27% of patients died within 12 months of their

injury.[1] Fransen followed 565 community-dwelling New Zealanders aged 60 and over with a recent hip fracture.[2] By the end of 2 years, 39% of females and 52% of males had died or been institutionalised.

The NZHIS study found that a delay of more than 2 days from hip fracture to surgery is associated with increased mortality.[1] This finding has been corroborated by a large study of NHS hospitals in the United Kingdom, which demonstrated an odds ratio of 1.27 for in hospital mortality in those whose operation was 2 or more days after admission, compared to those who had surgery within the first 2 days.[3]

At Auckland City Hospital, Orthopaedic patients aged 65 and over receive medical input from a Geriatrician or Older Peoples Health Registrar by way of twice-weekly ward rounds. Patients with hip fracture are assessed both pre and postoperatively. Following surgery, those assessed as having potential for rehabilitation are placed on the waiting list for Older Peoples Health. A weekly Ortho-Geriatric Interdisciplinary Team Meeting is held, identifying additional patients who require further rehabilitation via Older Peoples Health.

In 2006 a new initiative was introduced. Selected hip fracture patients are 'fast-tracked' to one particular Older Peoples Health (OPH) ward as soon as possible postoperatively. The aim of this is to provide a specialist care environment for these patients, thereby improving early postoperative management and potentially improving outcomes. The particular OPH ward is one of two that have an enhanced therapy establishment appropriate to rehabilitation of patients with stroke and similar high rehabilitation needs. The decision to 'fast-track' is initiated by the charge nurse on the OPH ward receiving the patient, when a bed is available. The earliest post operative patients are given priority.

Due to limitation of resources, not all hip fracture patients are able to be 'fast-tracked', with the majority receiving 'usual care' i.e. rehabilitation on the Orthopaedic ward and referral to Older Peoples Health for ongoing rehabilitation if required. Patients from Private Hospitals (facilities providing high-level long-term residential care) are not necessarily excluded from transfer to Older Peoples Health, and may be transferred if they have ongoing medical issues which require stabilisation before transfer back to Private Hospital.

The aims of this study were to: evaluate hip fracture patient characteristics; to analyse process of care; and to analyse outcome measures. We also set out to compare process and outcome measures in those "fast-tracked" patients with those receiving usual care. Finally, we set out to compare this information with previous data from Auckland City Hospital and other centres in New Zealand.

Methods

A prospective case notes audit was undertaken of all patients aged 65 and over with hip fracture admitted under the Orthopaedic service at Auckland City Hospital over a 4-month period from 1 April 2007 to 31 August 2007. Patients were identified at the weekly Ortho-Geriatric Interdisciplinary Meeting and by the Orthopaedic Fractured Neck of Femur Nurse Specialist.

A data collection form was designed to collect the required patient information. Clinical notes were reviewed manually by the principal investigator.

Information recorded included patient demographics, premorbid level of function, the American Society of Anaesthesiology physical status classification (ASA) score, comorbidities, type of fracture, time to operation, operative procedure and anaesthetic type, length of stay in Orthopaedic and Older

Peoples Health wards, time waiting for transfer to Older Peoples Health, total length of stay, complications, whether Deep Vein Thrombosis (DVT) prophylaxis was used, mortality, discharge destination, level of function on discharge and treatment of osteoporosis.

Anonymised data was entered into a secure Microsoft Excel spreadsheet. Approval was gained from the Northern Y Regional Ethics Committee.

Patients under the age of 65 years or with fractures at sites other than the neck of femur were excluded.

Walking aids: frame was defined as low walking frame or super-stroller used without hands-on assistance. Patients requiring a Gutter frame or Arjo frame were grouped with those requiring a wheelchair. Bedbound patients included those only able to transfer from bed to chair with maximal assistance.

The patient's ability to wash (shower/bath), dress and toilet themselves was recorded as personal activities of daily living (ADLs). Information relating to instrumental activities of daily living was recorded, focussing on meal preparation, cooking, housework and clothes laundering. Ability to manage finances, medication administration and transportation was not recorded in this audit. Patients residing in Rest Homes or Private Hospitals were presumed to require assistance with both personal and instrumental ADLs.

Comorbidities were grouped into system categories: cardiac disease (including congestive cardiac failure, arrhythmia, ischaemic heart disease and valvular pathologies), respiratory disease (asthma or Chronic Obstructive Pulmonary Disease (COPD), interstitial lung disease) and documented cognitive impairment.

The ASA score allocated by the anaesthetist was recorded for each patient.

The non weight bearing scheme is an initiative that allows patients whose orthopaedic injury requires them to be non weight bearing to be cared for at a Private Hospital until their Orthopaedic surgeon allows them to weight bear. They usually return to Auckland City Hospital for rehabilitation under Older Peoples Health, although some receive rehabilitation in the community. The time spent on the non weight bearing scheme was not included in length of stay calculations.

Medications considered as DVT prophylaxis included aspirin, low molecular weight heparin, unfractionated heparin and warfarin. DVT prophylaxis was recorded without differentiating whether this was a new prescription or a usual medication for the patient.

Prescribing of osteoporosis prophylaxis including calcium, vitamin D and bisphosphonate (oral or intravenous) was recorded.

Results

Group demographics

115 patients aged 65 and over were admitted with a hip fracture during the 4-month audit period. The median age was 85 years (range 67–100). There were 89 females (78%) and 26 males (23%). The mean age for male patients was 82 years and for females was 85 years.

Measures of functional ability and social situation are reported in outcome measures to demonstrate changes that occurred over the time of the admission with a hip fracture.

Clinical and fracture characteristics

Medical comorbidities—Table 1 shows the number of patients with medical comorbidities in each category. Forty-five patients (39%) had comorbidities from a single category. Forty-four (38%) suffered from diseases in two categories and eleven (10%) in three or more categories. Fifteen patients (13%) had no comorbidities listed. Two patients had also sustained an upper limb fracture at the time of hip fracture.

Table 1. Comorbidities (categorised) of patients with hip fracture

Comorbidity	Number
Cardiac	50 (43%)
Respiratory	23 (20%)
Cognitive impairment	49 (43%)

ASA scores—The American Society of Anaesthesiology physical status classification (ASA) score prior to injury was recorded. Seventy-eight patients (68%) were classified as ASA 3, indicating severe systemic disturbance which is not incapacitating or acutely life-threatening.

Table 2. American Society of Anaesthesiology (ASA) scores of hip fracture patients

ASA score	Number
ASA 1	0
ASA 2	19 (17%)
ASA 3	78 (68%)
ASA 4	18 (16%)
Total	**115**

Type of fracture—Table 3 shows the site of hip fractures.

Table 3. Anatomical distribution of hip fractures

Intracapasular fractures	Number	Extracapsular fractures	Number
Subcapital	59	Basal Cervical	4
Midcervical	1	Intertrochanteric	42
		Subtrochanteric	9
Total	**60**		**55**

Type of surgical fixation—Table 7 shows the type of surgical procedure performed.

Table 4. Type of surgical fixation

Procedure	Number
Dynamic hip screw	59
Hemi-arthroplasty	41
Total hip joint replacement	9
Proximal femoral nail	5
Cannulated screw	1
Total	**115**

Of the 60 patients with intracapsular hip fractures, 11 underwent a dynamic hip screw (18%) and 39 had a hemiarthroplasty.

Pathways of care—One patient was transferred to Christchurch postoperatively for rehabilitation, so their final outcome data was unavailable. Overall 108 patients were discharged at the end of their clinical episode and included in this audit.

Ninety-one patients were transferred from Orthopaedic wards directly to Older Peoples Health for medical stabilisation, rehabilitation and discharge planning. Of the others: 17 were discharged directly from Orthopaedics (2 to home, 15 to Private Hospital); one died whilst under the care of Orthopaedics; and one was transferred to Christchurch. Five patients went from Orthopaedics to the non-weight bearing scheme, and all later returned to have rehabilitation under Older Peoples Health. A total of 96 (84%) of the audited group had rehabilitation and treatment by Older Peoples Health.

Two patients went from Older Peoples Health to the non-weight bearing scheme and later returned for further rehabilitation.

Process of care measures

Time from fracture to admission—The time from fracture to admission could not be calculated in 10 patients, for whom either a time of injury or time of admission had not been recorded. For those who had a time of injury recorded, all were admitted within 24 hours of injury.

Time to surgery—Table 5 shows time from admission to surgery.

Table 5. Time from admission to surgery

Time from admission to surgery	Number
<24 hours	27 (24%)
24–48 hours	41
49–72 hours	22
>72 hours	25 (22%)

Two patients were already in hospital for another reason at the time of injury. Sixty-eight patients (59%) had undergone surgery within 48 hours of admission.

Of those 88 patients who did not have surgery within 24 hours of admission, 24 (27%) were delayed because of medically instability or treatment. Thirty (34%) were awaiting further investigation, such as echocardiogram or radiology. The other 34 (39%) were waiting for operating theatre availability.

Length of stay (LOS)—The median and average lengths of stay are shown in Table 6. The average waiting time for Older Persons Health was 1 day (range 0–7).

Table 6. Median and average lengths of stay (LOS)

Length of stay (days)	LOS median (range)	LOS average
Orthopaedics	7 (1-37)	8.8
Older Peoples Health (OPH)	20 (3-60)	22.8
Total (Orthopaedics + OPH)	27 (5-71)	28.1

Outcome measures

Mortality—There were 6 inpatient deaths (5%)—one in the Orthopaedic ward and five in Older Peoples Health. Of those who died, 2 were male and 4 were female.

Complications—The most common postoperative complication was anaemia requiring blood transfusion, which occurred in 24% of patients. Delirium was documented in 23%. Pneumonia occurred in 17% of patients and urinary tract infection in 16%. One patient had failure of dynamic hip screw and was re-operated, receiving a hemiarthroplasty.

Living Situation—Table 7 shows the place of domicile of hip fracture patients on admission and at discharge.

Prior to admission, 70 patients (61%) were living at home. On discharge this had dropped to 49 (45%). The number requiring Private Hospital care rose from 10 (9%) on admission, to 46 (43%) on discharge.

Of those living at home on admission, 70% returned home. Of the 35 people originally living in Rest Homes, 25 (71%) were discharged to Private Hospital after their hip fracture. All of the 10 patients living in Private Hospitals were discharged back to Private Hospitals.

Overall 35% of patients went to a higher level of care on discharge.

Table 7. Place of domicile on admission and on discharge following hip fracture

Living situation Pre-fracture	Number	Living situation On discharge	Number (%)
Home	70	Home	49 (70%)
		Rest Home	4
		Private Hospital	11
		Died	5
		Other[1]	1
Rest Home	35	Rest Home	9 (26%)
		Private Hospital	25
		Died	1
Private Hospital	10	Private Hospital	10
Total	**115**		**115**

[1] One patient transferred to Christchurch Hospital.

Mobility—Table 8 shows patient's requirements for walking aids before hip fracture and on discharge. Fifty patients (44%) did not use any gait aid prior to their hip

fracture. Only one patient was able to walk unaided at discharge; 60 (56%) required a walking frame.

Table 8. Walking aids on admission and at discharge

Walking aid	On admission	On discharge
No walking aid required	50 (44%)	1 (1%)
Walking stick or crutch	20 (17%)	13 (12%)
Walking frame	42 (37%)	60 (56%)
Gutterframe/wheelchair	0	16 (15%)
Bedbound	3 (3%)	18 (17%)
Total	**115**	**108**

Activities of daily living (ADLs)—Table 9 shows that prior to their hip fracture 93 patients (80%) received assistance with one or more instrumental activities of daily living. Sixty-two (54%) required assistance with one or more personal activities of daily living. On discharge, 86% of patients received help with personal ADLs, and 93% received help with instrumental ADLs.

Table 9. Need for assistance with activities of daily living—on admission and at discharge

Activities of daily living (ADLs)	Need for assistance – admission	Need for assistance – discharge
Personal ADLs	62 (54%)	93 (86%)
Instrumental ADLs	93 (80%)	100 (93%)

Prescriptions for prevention

DVT prophylaxis—DVT prophylaxis was prescribed in 89 (77%).

Osteoporosis treatment—Table 10 shows that on discharge 68 patients (63%) were treated with a bisphosphonate (weekly oral Alendronate or annual zolendronate infusion).

Of those who did not receive a bisphosphonate, inappropriate clinical context was cited in 28 patients. The reason given in four of these patients was age less than 75 years, therefore requiring a DEXA scan to qualify for bisphosphonate. Of the others considered to be in the inappropriate clinical context group by their treating geriatrician, three were of advanced age (93, 94 and 95 years respectively) and [21] were in private hospital care.

Allowing for this, overall compliance with osteoporosis prescribing guidelines for bisphosphonates was 93%, for calcium was 89% and for vitamin D was 88%.

Table 10. Prescribing of osteoporosis treatment on discharge

Bisphosphonate	No bisphosphonate	Had a reason for not prescribing	
68 (63%)	40	Yes No	32 8
Calcium 81 (75%)	No calcium 27	Yes No	15 12
Vitamin D 83 (76%)	No Vitamin D 25	Yes No	12 13

Of the 15 patients who were not prescribed calcium, inappropriate clinical context was cited in 14: one patient had documented hypercalcaemia, 13 were discharged to a Private Hospital. For those not prescribed Vitamin D, inappropriate clinical context was cited in 12 (48%); no reason was documented in the other 13 patients.

Comparison of "fast-tracked" with "usual care" patients

Thirty-nine patients were "fast-tracked" on the day of surgery or Day 1 postoperatively, constituting 43% of all Orthopaedic patients transferred to Older Peoples Health. See Table 11 for comparative demographics and Table 12 for fracture type and surgical fixation. These appear broadly similar.

Table 11. Demographics and fracture sites of patients fast-tracked compared with usual care

Demographic	Fast tracked	Usual care
Number	39	57
Age – median (years)	86.0	84.5
Female	34 (87%)	41 (72%)
Male	4	16
ASA 3	27 (69%)	40 (70%)
ASA 4	4	9

Table 12. Type of fracture and surgical fixation of patients fast-tracked

Fracture or procedure type	Fast tracked	Usual care
Intracapsular fracture	25(64%)	23(40%)
Extracapsular fracture	14	34
Dynamic hip screw	19(49%)	33(58%)
Hemiarthroplasty	16	16
Total hip joint replacement	4	3
Proximal femoral nail	0	5

Table 13 shows comparative length of stay data for fast-tracked patients versus those receiving usual care.

Table 13. Length of stay (LOS) in days – fast-track and usual care

Median number of days	Fast-Track	Usual Care
Orthopaedics	2	11
Older Peoples Health	20	21
Total LOS	23	28

Outcomes for fast-tracked patients are shown in Table 14 and for those receiving usual care in Table 15. For fast-tracked patients: 74% of patients originally living at home returned home; 15% of Rest Home patients returned to Rest Home; 33% of patients went to a higher level of care on discharge. For usual care patients: 65% of patients originally living at home returned home; 31% of Rest Home patients returned to Rest Home; 35% of patients went to a higher level of care on discharge.

Table 14. Discharge destination for patients fast-tracked

Living situation Pre-fracture	Number	Living situation on discharge	Number (%)
Home	23	Home Private Hospital Died	17 (74%) 3 3
Rest Home	13	Rest Home Private Hospital Died	2 10 (77%) 1
Private Hospital	3	Private Hospital	3
Total	**39**		**39**

Table 15. Discharge destination for patients receiving usual care

Living situation Pre-fracture	Number	Living situation on discharge	Number (%)
Home	34	Home Rest Home Private Hospital Died Other	22 (65%) 3 6 2 1[1]
Rest Home	16	Rest Home Private Hospital	5 11 (69%)
Private Hospital	7	Private Hospital	7
Total	**57**		**57**

[1] One patient transferred to Christchurch.

Comparative data from previous audits at Auckland City Hospital

Table 16 shows data from previous audits of hip fracture patients aged 65 years and over at Auckland City Hospital.

Table 16. Comparative data – Auckland City Hospital

Patients 65 Years and Over with Hip Fracture	1993	1996	2002	2007
% Living at home pre-fracture	66	60		61
% Transfer to OPH	45	67	60	84
Mean Wait Time for OPH (days)	8	4	2	1
Mean LOS Orthopaedics (days)	13	9	12	9
Mean LOS total (days)	45	38		28
% Surgery in <24hrs	50	49	59	24
% From home returning home post fracture	63	82		70

Note: Orthopaedic services in greater Auckland region were reconfigured in 2003/04 and the new Auckland City Hospital opened in late 2003.

Discussion

This prospective audit of a busy Ortho-Geriatric unit showed that Auckland City Hospital provides its population with a service broadly comparable to other major centres. However, there has been a significant decline in the number of patients undergoing surgery for hip fracture within 24 hours of admission. Lack of operating resources explained why 39% did not receive surgery in this time frame.

The transfer rate to Older Peoples Health from Orthopaedics is higher than other centres, and the overall length of the episode of care is longer. The innovation of "fast-tracked" patients achieved similar outcomes to those receiving usual care, but shorter lengths of stay in the Orthopaedic ward and overall.

Patient age and gender distribution and pre-admission place of residence were comparable to those in a similar audit by Thwaites et al from Christchurch in 2005 [5].

Of the 115 patients with hip fracture in this audit, inpatient mortality was 5%. This is comparable with an in-patient mortality of 8% in a Christchurch study by Elliot et al in 1996,[4] and 5% mortality during the initial hospital episode in the NZHIS study.[1] Several recent studies have shown an increased mortality in male patients following hip fracture.[2,6] The higher inpatient mortality for females in our study (66% of deaths) may be due to smaller sample size and shorter follow-up time.

There is evidence suggesting that delay to operation in hip fracture increases mortality, even when adjustments are made for comorbidities.[1,3] The New Zealand Guidelines Group guideline for acute management and rehabilitation after hip fracture recommends that surgical fixation should take place within 24 hours of admission.[7] There is, however, evidence that mortality is increased when hip fracture surgery is undertaken as a night-time emergency.[8]

In this audit, time to surgery was comparable to the NZHIS study, which reported 73% of patients undergoing surgery on the day of admission or the day after, (27% and 46% respectively).[1] Individual centres, however, have reported better performance in this area.

In Weatherall's study, 94% of patients had been operated on within 48 hours of admission [9]. North Shore Hospital in Auckland reported 58% of hip fracture patients undergoing surgery within 24 hours of admission in a recent audit, although

Middlemore Hospital experienced similar delays to our study due to demand for operating theatre time.[10]

Surgery may need to be delayed in order to treat medical conditions and reduce operative risk. However in this audit 38% of those delayed were not unstable or awaiting investigation, suggesting that access to surgical resources is a key factor. The percentage of patients having surgery within 24 hours of admission at Auckland City Hospital was stable between 1993 and 2002, but approximately halved between 2002 and 2007. We postulate that changes in operating theatre access as a result of completion of the new Auckland City Hospital in late 2003 have lead to increased delays for older patients with hip fractures. Further audit of the orthopaedic service is needed to clarify the reasons for this deterioration in service.

Determining an optimal length of stay for patients after hip fracture is difficult. It is not always clear how best to balance the cost of inpatient hospital stay against maximising functional outcomes through rehabilitation.

In this study, median overall length of stay was 15% longer than that found by Thwaites et al in Christchurch.[5] Weatherall et al in Waikato reported 19.9 days,[9] and an audit of Middlemore and North Shore Hospitals reported mean lengths of stay of 22 and 17 days respectively.[10] The median length of stay in orthopaedics was comparable between these studies, showing that time in rehabilitation is the main variation across different centres. See Table 17.

Table 17. Comparative data – other New Zealand centres

Patients 65 years and over with fractured neck of femur (NOF)	Auckland City Hospital 2007	North Shore Hospital 2004 [10]	Middlemore Hospital 2004 [10]	Christchurch 2005 [5]
Orthopaedics LOS (mean)	9	9	13	8*
Total LOS (mean)	28	17	22	23*
% Transfer to Older Peoples Health	84	75	52	
% Home-home	70	81	81	87

* = median.

A longer length of stay may be justifiable if improved patient outcomes are observed. In the audited population 35% of patients went to a higher level of care on discharge. Of patients living at home prior to their hip fracture, only 70% returned home. This is a lower percentage than that reported in similar studies (see Table 16), where between 81 and 87% of patients from home returned home following hip fracture.[5,9,10] Differences in criteria for inpatient rehabilitation between different centres may account for some of this variation.

Rest Home patients made a disproportionately poor recovery, with the majority (71%) moving to Private Hospital care. This percentage was higher than those of Middlemore and North Shore Hospitals, which reported 49% of patients from Rest Home returning to Rest Home.[10] Rest Home patients in Christchurch and Waikato fared much better,[5,9] which may indicate differences in populations. Ethnicity or socioeconomic factors may be important in these regional differences. However, a

more cogent explanation is that the dependency levels of people in residential care in the Auckland region have significantly increased over the last 20 years [20].

Hip fracture patients with more comorbidities have a higher risk of postoperative complications and mortality.[11] Geriatrician input has been shown to improve these outcomes,[12] hence the development of the fast-track system we have described. These patients had a 15% shorter total length of stay due to a shorter stay in orthopaedics, but time in Older Peoples Health was unchanged. Our small sample size makes it difficult to comment on the effect on complications and mortality for the patients who are fast-tracked. However these patients seem to do no worse in terms of requiring a higher level of care on discharge.

The percentage of hip fracture patients transferred to Older Peoples Health was high, at 84%. Weatherall et al reported 57% of patients were transferred to rehabilitation wards,[9] Middlemore was lower at 51%, and North Shore Hospital reported 75% transferred [10]. This demonstrates that a more open approach to selecting patients for rehabilitation is being used at Auckland City Hospital. By not excluding Private Hospital patients, the frailty of this group is increased, and a larger proportion of patients are likely to make a less complete recovery.

The ASA score, which has been shown to reliably predict perioperative morbidity and mortality, has not been recorded in all studies, making comparison difficult.[13] Eighty-four percent of patients in this audit had ASA scores of 3 or 4, compared to 69% and 75% in a similar audit of hip fractures at North Shore Hospital and Middlemore Hospitals, respectively.[10] The low inpatient mortality rate (0.7%) and high proportion of patients returning home after hip fracture in the Christchurch study[5] may suggest a more robust population.

Dementia was a common comorbidity and was more frequent in this study than in a similar population at Middlemore and North Shore Hospitals.[10] This may impact on a patient's ability to rehabilitate from hip fracture, and could be a contributing factor to the longer length of stay and higher level of dependency seen on discharge in this study.

Patients depend upon therapist input, and changes in staffing levels may affect length of stay and outcomes. Local differences in Rest Home care and availability of Private Hospital beds may also lead to more patients moving to a higher level of care.

Comparative data from earlier audits of hip fracture patients in Auckland City Hospital shows that a similar percentage of patients were living at home prior to their fracture in 1993 and 1996 as in 2007. The length of stay in orthopaedics is similar between these audits, but the mean overall length of stay has reduced from 45 days in 1993 to 28 days in 2007. This is most likely to be a reflection of trends across all hospital inpatients towards earlier discharge. The proportion of patients transferred to Older Peoples' Health post hip fracture has almost doubled between 1993 and 2007, but the average waiting time has dropped from over a week to one day. A similar percentage of patients are able to return home following their hip fracture in 2007 as in 1993.

Further study into the factors contributing to length of stay in Older Peoples Health is required, as improvements in this area would have significant cost implications. It is also important that readmission rates are recorded in future studies of this service, as

this would determine whether longer length of stay increases the frequency of successful discharges.

Although there is good evidence to support the use of osteoporosis treatment in prevention of further fractures, rates of prescribing are often low.[14–18] In this study, no explanation was given in 20% of those not prescribed a bisphosphonate and this was even higher for calcium and vitamin D, suggesting that this is omission rather than conscious decision. Protocols have been shown to be effective in improving osteoporosis prescribing,[19] and other strategies could include the use of a drug-chart "sticker" or discharge check-list.

This audit provides short-term data on outcomes of older hip fracture patients managed by Orthopaedics and Geriatric Medicine at Auckland City Hospital. It is limited by size and duration and has limitations when comparing with other studies and trials due to widely varying aims, methods and outcome measures between the studies. Developing a standardised approach in New Zealand to future audits would help with comparisons. For example adopting the audit tool used for the National Hip Fracture Database in the UK (http://www.nhfd.co.uk) would enhance both local and international comparison.

Conclusion

This audit demonstrates a significant delay to surgery for patients with hip fractures. The percentage of these patients being transferred to Older Peoples Health wards from Orthopaedics is high at 84%. "Fast-tracking" to Older Peoples Health wards shortens overall length of stay due to fewer days in Orthopaedics. Many patients require a higher level of care after hip fracture, particularly if already resident in Rest Home.

Changing structures within hospital systems have the potential to adversely affect older patients with complex care needs. The 'fast-track' process described in this audit was designed to both maximise use of beds on the orthopaedic wards and also to improve care by providing earlier care by a specialist service for older people.

It is reassuring to find that moving older patients with hip fractures to Older Peoples Health on day 1 postoperatively does not seem to disadvantage them in terms of discharge outcomes.

The reduction in the number of patients receiving surgery within 24 hours of admission is a comparatively recent phenomenon coinciding with the reconfiguration of the Auckland regional Orthopaedic services and the opening of the new Auckland City Hospital. It appears that these structural changes have had an unintended adverse effect.

Demographics and inpatient mortality are comparable, but total length of stay is longer than similar New Zealand studies due to a longer length of stay in Older Peoples Health. Patients presenting with hip fracture are a large and heterogeneous group, with variations in age, mobility, living situation and comorbidities. Their outcomes in the different studies are dependent on case mix and the many other factors mentioned.

In addition, in Auckland the dependency levels of people in residential care have significantly increased over the last 20 years. These factors may account for some of the differences compared to audits from other New Zealand centres.

Review of previous data from Auckland City Hospital and from other New Zealand centres shows significant variability in process of care for older patients with hip fracture. Developing a standardised approach in New Zealand to future audits would help with comparisons.

Further research into the reasons for delay to operation and long length of stay in Older Peoples Health is needed to improve the process of care. The application of quality improvement principles and ongoing audit of the whole patient journey is needed to allow continuous improvement of the Ortho-Geriatric Service.

Competing interests: None.

Author information: Lucy Fergus, Advanced Trainee in Geriatric Medicine, Older Peoples Health; Greer Cutfield, Nurse Specialist, Fracture Neck of Femur, Orthopaedics; Roger Harris, Geriatrician, Older Peoples Health; Auckland City Hospital, Auckland

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Correspondence: Dr Roger Harris; email rogerh@adhb.govt.nz

References:

1. NZHIS. Fractured neck of Femur Services in New Zealand Hospitals 1999-2000. 2002.
2. Fransen M, Woodward M, Norton R et al. Excess mortality or institutionalisation after hip fracture; men are at greater risk than women. JAGS 2002;50:685–690.
3. Bottle A, Aylin P. Mortality associated with delay in operation after hip fracture: observational study. BMJ 2006;332:947-51.
4. Elliot T, Wilkinson T, Hanger H et al. The added effectiveness of early geriatrician intervention on acute orthopaedics wards to orthogeriatric rehabilitation. N Z Med J 1996;109:72-3.
5. Thwaites T, Mann F, Gilchrist N, et al. Shared care between orthopaedic surgeons and geriatricians as a model of care for older patients with hip fractures. NZ Med J 2005;118(1214). http://www.nzmj.com/journal/118-1214/1438/content.pdf
6. Pande I, Scott D, O'Neill T et al. Quality of life, mortality and morbidity after low trauma hip fracture in men. Ann Rheum Dis 2006;65:87-92.
7. Acute management and immediate rehabilitation after hip fracture amongst people aged 65 years and over. NZGG June 2003.
8. Campling E, Devlin H, Hoile C et al. The Report of the National Confidential Enquiry into Perioperative Deaths 1991. NCEPOD, 1993.
9. Weatherall M. Case mix and outcome for patients with fracture of the proximal femur. NZ Med J 1993;106:451-2.
10. Tha HS, Armstrong D, Broad J, et al. Hip fracture in Auckland – contrasting models of care in two major hospitals. Internal Medicine Journal 2009;39:89-94.
11. Roche J, Wenn R, Moran C. Effect of comorbidities and postoperative complications on mortality after hip fracture in elderly people: a prospective observational cohort study. BMJ 2005 doi:10.1136/bmj.38643.663843.55 (published 18 November 2005).
12. Fisher A, Davis M, Rubenache S et al. Outcomes for older patients with hip fractures: the impact of orthopaedic and geriatric medicine cocare. J Orthop Trauma 2006;20:172–180.

13. Menke H. Predictive value of ASA classification for the assessment of the perioperative risk. International Surgery 1993;78(3):266-70.
14. Black D, Cummings S, Karpf D et al. Randomised trial of the effect of alendronate on risk of fracture in women with existing vertebral fractures. Lancet 1996;348:1525-41.
15. Black D, Thompson D, Bauer D et al. Fracture risk reduction with alendronate in women with osteoporosis: fracture intervention trial. J Clin Endocrinol Metab 2000;85:4118-24.
16. Chapuy M, Arlot M, Duboeuf F et al. Vitamin D3 and calcium to prevent hip fractures in elderly women. NEJM 1992;327:1637-42.
17. Wong P, Spencer D, Mc Elduff P et al. Secondary screening for osteoporosis in patients admitted with minimal – trauma fracture to a major teaching hospital. Intern Med J 2003;33:505-20.
18. Teede H, Jayasuriya I, Gilfillan C et al. Fracture prevention strategies in patients presenting to Australian hospitals with minimal-trauma fractures: a major treatment gap. Intern Med J 2007;37:674-679.
19. Sidwell A, Wilkinson T, Hanger H et al. Secondary prevention of fractures in older people: evaluation of a protocol for the investigation and treatment of osteoporosis. Intern Med J 2004;34:129-32.
20. Boyd M, Connolly M, Kerse N, Foster S, et al. Changes in Aged Care Residents' Characteristics and Dependency in Auckland 1988 to 2008; Findings from OPAL 10/9/2008 - Older Persons' Ability Level Census. ISBN 978-0-473-15974-0 (paperback), ISBN 978-0-473-15975-7 (electronic). 2008.

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Prevalent dietary supplement use in older New Zealand men

Catherine J Bacon, Mark J Bolland, Ruth W Ames, Amanda T Y Siu, Barbara H Mason, Anne M Horne, Andrew Grey, Ian R Reid

Abstract

Aims Because of a lack of recent data from New Zealand older men, we examined dietary supplement use in this demographic.

Methods We surveyed men aged >40 years who were participating in a trial of calcium supplementation on bone and cardiovascular outcomes.

Results Forty-seven percent reported using at least one supplement and 30% of users took more than two different supplements. Amongst users, median monthly expenditure on these products was NZ$20 (interquartile range: $10–$45). The most common supplements used were vitamins or minerals (49%), followed by nutritional oils (22%) (including fish oils, 13%) and glucosamine/chondroitin preparations (13%). Supplements were mainly taken for reasons of non-specific prophylaxis or health maintenance (58% of reasons), although 21% of reasons cited treatment or symptom alleviation for a medical condition. Daily requirements for vitamins A, D and E were exceeded, from supplement intake alone, by 12%, 10% and 40% of supplement users respectively.

Conclusions Many older New Zealand men spend substantial amounts of money on dietary supplements despite uncertain health benefits. Health professionals should remain alert to supplement use by their patients, including males.

Dietary supplements include a vast quantity and variety of over-the-counter pills, liquids or powders containing vitamins and minerals, herbal and other botanical products, amino acids, a range of other enzymatic or potentially nutritive substances, and mixtures of these.

Around the turn of the millennium, New Zealand surveys reported a prevalence of regular dietary supplement use of 18 – 28%,[1-3] although use by both young and older males was 62–65% that of females.[1, 3] Longitudinal United States data supports an increasing use of dietary supplements by older people in the late 1990s,[4] and it is possible that there has been a similar rise in use in New Zealand in recent years.

Whilst many supplements are claimed to improve health or nutrition, there is often a lack of published evidence to support these benefits [5]. For those taking supplements, intakes of some nutrients may exceed recommended upper intake levels,[6, 7] a daily intake level judged safe for almost all individuals.[8] Furthermore, potentially harmful interactions may arise from concurrent use of herbal preparations and prescription medications,[4, 5] or toxicity from contaminants in supplements, including those derived from fish oils and other marine products.[9–11]

In a recent trial of calcium supplements conducted in middle-aged and older men living in Auckland,[12] we were surprised by the number of men who reported dietary

supplement use in a baseline questionnaire of medication use. Therefore, we asked participants to complete a more detailed questionnaire at a later visit.

We sought to determine the number of different supplements used, the number of doses taken each day and the estimated expenditure on these products. Dietary intakes of vitamins A, D, and E from supplements were calculated and compared with daily intake recommendations. We also investigated the importance of various information sources and the reason for using each product.

Methods

Participants—323 men took part in a two-year randomised controlled trial of calcium supplements investigating skeletal and cardiovascular endpoints, the methods and results for which have been previously detailed.[12, 13] Men were aged >40 years, were free of major medical conditions, had 25-hydroxyvitamin D >25 nmol/L, and were not taking therapy for hyperlipidaemia, osteoporosis, or vitamin D supplements at a dose of >1000 IU/day.

Baseline measurements—Smoking history and a record of physical activity were obtained at a screening visit prior to baseline, and measurements of height and weight were obtained at baseline.

Supplement questionnaire and analysis—Men were asked to complete a questionnaire describing their use of supplements, vitamins or similar, their reasons for taking each supplement and the importance of 11 listed and other participant-specified sources of information in their decision to take them. Importance was rated on a 5-point scale with 0 indicating that the source was of no importance in their decision and 4 indicating that it was very important. This questionnaire was completed between April 2006 and July 2007 when participants were in the second year of the study.

Baseline differences between supplement users and non-users were compared using t-tests. The number of different supplements listed and the number of daily doses taken by each man was quantified. A single dose was defined as one tablet, or the recommended dose for liquid preparations. Men were asked to estimate the monthly cost of each supplement.

Monthly cost data were incomplete for 21 men and, in these cases, were estimated from the cost of purchasing the same or similar products from online pharmacies on the Internet. To ascertain whether there were differences in supplement use between older and younger men, those above and below 60 years were compared using Mann-Whitney tests.

Intakes of vitamins A, D and E from supplements were computed using information provided by manufacturers. Vitamin A was expressed as retinol activity equivalents (RAE), which equates to the amount of precursor or preformed vitamin A that must be consumed to equal 1 mcg of retinol, calculated using the formula RAE = mcg retinol + 0.083 mcg beta-carotene.[8]

Statistical analyses were performed using SPSS version 15.0 software (SPSS Inc. Chicago, IL, USA).

Results

Participants and supplement use—305 men returned the supplement questionnaire. There were no significant differences in the general characteristics of the 142 men (47%) who reported taking supplements compared to those who did not (Table 1). Supplement users took a median of 2 different dietary supplements (Figure 1a) or 2 doses/day (range: 2 doses/week—10 doses/day). 16 (11%) men reported taking supplements prescribed by their doctor and 4 (3%) took only supplements prescribed by their doctor.

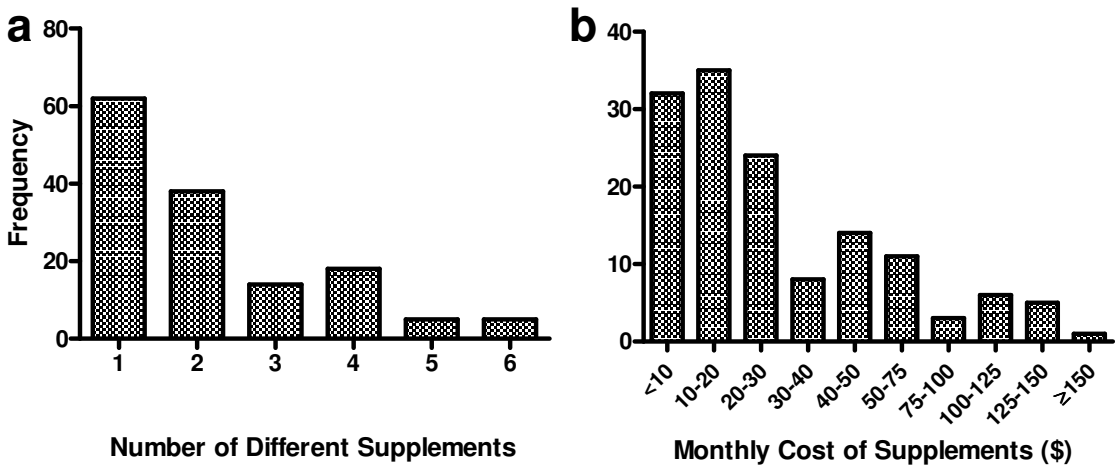
The median monthly estimated cost reported by participants who took supplements was NZ$20 (interquartile range: $10—$45; maximum $268). A fifth spent more than $50/month on supplements (Figure 1b). There was no difference between younger and older men in the number of different supplements, the total daily dose and the total monthly cost.

Table 1. General characteristics of respondents at baseline (n=305)

Variables	Supplement users* (n=142)	Non-users (n=163)
Age (years)	57.1±10.5	56.2±9.8
Weight (kg)	82±11	84±13
Body Mass Index (kg/m^2)	26±3	27±3
Physical Activity (MET.h/day)	32±5	32±6
Smoking status (%)		
Current	4	3
Previous	46	39

Data are mean±SD; * Positive response to questions asking whether men were currently taking prescription or non-prescription supplements, vitamins or similar.

Figure 1a and 1b. The number of different dietary supplements and monthly expenditure on dietary supplements for supplement users (n=142)



Sources of information—The information sources reported to be of greatest importance in decision making were scientific or medical publications, their own doctor, and health professionals other than their doctor, which all scored an average of 2.0-2.3 out of 4 on the level of importance. Magazines, news articles and people other than health professionals were also rated as moderately important (average score between 1.7 and 1.8). Older men (≥60 years) compared to those below this age attributed lesser importance to news articles (*P*=0.02), the Internet (*P*=0.01), health professionals other than their own doctor (*P*=0.006), and gyms, fitness centres or health clubs (*P*=0.006), and a greater importance to people other than health professionals (*P*=0.02).

Types of supplements and reasons for use—A total of 307 different products were taken. 49% were vitamins or minerals, either a single nutrient or combined multivitamin/multimineral preparation with or without other herbal ingredients.

Nutritional oils such as evening primrose or fish oils (22%, with 13% specifically branded as fish oil derivatives), preparations of glucosamine, chondroitin and/or methylsulfonylmethane (13%), and individual herbal products (8%) were also commonly taken.

Other products included bee pollen (3%), dietary fibre (1%) and co-enzyme Q10 (1%). One man reported taking a multivitamin/multimineral supplement formulated for horses. The most common reason provided for taking supplements was for non-specific health maintenance or prophylaxis (58%). Twenty-one percent cited treatment or relief of symptoms as the primary reason for use, 16% nutritional benefits, and 2% athletic performance.

Vitamin intake from supplements (Table 2)—Seventeen men (12% of supplement users) had a total intake of vitamin A from supplements alone in excess of the US recommended daily allowance (RDA). Because high intakes of beta-carotene are not toxic, the upper daily intake limit deemed tolerable (Upper Limit) for vitamin A (3000 mcg) applies to intake of preformed retinol only. The supplemental retinol intakes for two men were high enough that they would be very likely to exceed this amount if additional retinol intakes from food were taken into account. One individual combined two halibut liver oil tablets (total 3000 mcg), and the other took a total of 2940 mcg from supplements manufactured by a US company that are distributed in New Zealand through personal contacts.

For vitamin D, 14 men (10% of supplement users) exceeded a daily intake of 400 IU vitamin D from supplements alone, although only one exceeded the Upper Limit of 2000 IU/day (2400 IU/day).

Fifty-seven men (40% of supplement users) exceeded the US RDA for vitamin E from supplements alone, but none approached or exceeded the Upper Limit.

Table 2. Daily vitamin intakes from dietary supplement in men taking supplements containing these

Variables	n	Mean±SD	Median	75th Percentile	Maximum	US RDA* or AI	US UL*
Vitamin A (RAE[#])	80	504±645	275	750	3000	900	none set
Retinol (mcg)	68	476±197	197	750	3000	none set	3000
Vitamin D (IU)	85	226±363	100	400	2400	200-400	2000
Vitamin E (mg)	67	82±138	42	100	826	15	1000

* The US recommended daily allowance (RDA) is defined as the level of an intake sufficient to meet the nutrient need of almost all healthy men in this age group. Insufficient data is judged to exist to set this level for vitamin D. Instead, an adequate intake (AI), defined as the approximate average nutrient intake that appears to sustain a desired indicator of health, has been set. The tolerable upper intake level (UL) is defined as the maximum daily intake by an individual that is unlikely to pose risks of adverse health effects to almost all men in this age group.[8]

[#] Retinol activity equivalent (RAE) is the amount of precursor or preformed vitamin A that must be consumed to equal 1 mcg of retinol.[8]

Discussion

Almost half of men in our study reported taking dietary supplements, supporting previous surveys suggesting that many older New Zealand men take dietary supplements.[2, 3] This proportion is substantially higher than that of men >45 years reporting either regular or occasional use in New Zealand in the late 1990's (around 30%),[3] and higher than the prevalence reported in young New Zealand men (13%).[1] This may reflect increasing use amongst men, or may reflect the study population of healthy middle-aged and older men who volunteered for a clinical trial of a calcium supplement.

Men here had a low body mass index and low prevalence of smoking, both of which have been associated with increased supplement use [7]. The present study also suggests that many men spend substantial sums of money on these products. A fifth of men spent an estimated NZ$50/month or more on these products, and the true value may be higher since some estimates were based on the prices from online pharmacies which may be less expensive than other sources.

Although individual income data were not available, this figure represents approximately 2% of the average monthly income in New Zealand [Statistics New Zealand http://wdmzpub01.stats.govt.nz/wds/ReportFolders/ReportFolders.aspx (accessed December 8, 2009)]. This amount may have represented a higher percentage of income in this cohort, many of whom were retired.

The most commonly used supplements were multivitamin/multiminerals, followed by nutritional oils, over half of which were fish-derived, and glucosamine/chondroitin preparations.

Data from the 1997 New Zealand National Nutrition Survey show that almost all older men already meet recommended intakes of Vitamin A [3]. Similarly, when observed supplement intakes in our study were added to age- and gender-specific average vitamin E intakes from the national survey [3], all but five of the men taking supplemental vitamin E and 44% of all supplement users exceeded recommended levels of vitamin E.

Recommended levels of intake of vitamins and minerals are based on available evidence. Thus, by definition, there is no demonstrated benefit for healthy individuals taking levels of these nutrients in excess of the RDA. Furthermore, there is minimal evidence for specific benefits of vitamin supplementation in the literature.

Meta-analyses of antioxidant interventions (including vitamins A, C and E but excepting those providing selenium), report a lack of effect on cancer outcomes,[14, 15] and one large trial reported an increased mortality risk with high dose vitamin A (7500 ug retinol and 30 mg beta-carotene, total 9990 RAE).[16] The effects of glucosamine and/or chondroitin are also equivocal.

Recent systematic reviews of randomised trials report an effect of glucosamine sulphate, but not other salts, on osteoarthritic pain and disease progression, with most of the existing data for knee osteoarthritis.[17, 18] However, effects are considerably greater for trials that are industrially funded, particularly those of a single manufacturer.[17]

Similarly, evidence for the numerous reported benefits of fish oils is conflicting,[19-21] although there is growing evidence from several randomised controlled trials of a beneficial effect of omega-3 fatty acids, supplied by fish oils, on cardiovascular risk indices.[22-24]

Previous studies which combine food and dietary supplement consumption have noted nutrient intakes in excess of tolerable upper intake levels for a range of nutrients, including niacin, vitamin C, iron, magnesium and zinc.[6, 7]

Upper intake levels are conservative estimates of a sustained level of intake that can probably be tolerated by most individuals and, in the case of both vitamins A and D, have been criticised for being too low.[25, 26] Combined use of multivitamins and fish-liver oil products may result in potentially harmful intakes of pre-formed vitamin A or other fat soluble vitamins.

For a few individuals here, such use was associated with sizeable intakes of vitamins A and D. However, because the 3 men who approached or exceeded tolerable upper intake levels would be unlikely to exceed this level by a very large margin, even if intake from food was taken into account, the present data do not suggest a high likelihood of risk. Similarly, no men in this study approached the UL for vitamin E, and no adverse events have been noted in randomised trials of vitamin E doses around or below this level [27].

Men in the present study claimed that information from scientific or medical publications, their own doctor and other health professionals were the most important factors in their decisions about supplement use, though few products were actually prescribed by a physician.

Although the specific role that health professionals had in supplement use decisions is unclear, these findings may be in contrast with past studies which report that individuals are unlikely to seek medical advice or inform their doctors of their use of dietary supplements.[28, 29]

Regulations prevent dietary supplements being marketed as treatment or for alleviation of symptoms of specific medical conditions. It is therefore not surprising that most men noted non-specific prophylactic reasons for taking supplements. Compensating for dietary insufficiencies may be a justifiable reason, although relatively few men cited this.

Over one-fifth of reasons for taking a supplement cited symptom alleviation or treatment for an existing specific medical condition. Given the lack of evidence of a benefit arising from almost any ingredient in these products, it is alarming that so many men in the present study took dietary supplements for this reason.

In summary, many men who volunteered to take part in a clinical trial of calcium supplementation reported spending substantial amounts of money on dietary supplements. This is despite a lack of evidence supporting benefits of their use, and some evidence of associated risk. As a whole, this cohort purported to be most strongly influenced by health professionals and the results of scientific studies with respect to their decision to take supplements. Although these factors may not have ultimately driven men's behaviour, their high ratings suggest an acknowledgement of the authority of these sources of information.

So that sound and convincing advice may be provided, it is important that health professionals maintain up to date knowledge of available dietary supplements, evidence surrounding their use, as well as common claims made by their manufacturers and suppliers.

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Author information: Catherine J Bacon, Doctoral Graduate; Mark J Bolland, Senior Research Fellow; Ruth W Ames, Research Officer; Amanda T Y Siu, Research Student; Barbara H Mason, Research Officer; Anne M Horne, Research Officer; Andrew Grey, Associate Professor of Medicine; Ian R Reid, Professor of Medicine; Department of Medicine, University of Auckland, Auckland

Correspondence: Catherine Bacon, Clinical Bone Research, Department of Medicine, Level 3 Support Building, ACH, University of Auckland, Private Bag 92019, Auckland, New Zealand. Fax: +64 (0)9 3078970; email: c.bacon@auckland.ac.nz

References:

1. Allen T, Thomson WM, Emmerton LM, Poulton R. Nutritional supplement use among 26-year-olds. N Z Med J. 2000;113:274-7.
2. Norrish AE, Jackson RT, Sharpe SJ, Skeaff CM. Men who consume vegetable oils rich in monounsaturated fat: their dietary patterns and risk of prostate cancer (New Zealand). Cancer Causes Control. 2000;11:609-15.
3. Russell DG, Parnell W, Wilson N, et al. NZ Food: NZ People: Key Results of the 1997 National Nutrition Survey. In: New Zealand Ministry of Health; 1999.
4. Wold RS, Lopez ST, Yau CL, et al. Increasing trends in elderly persons' use of nonvitamin, nonmineral dietary supplements and concurrent use of medications. J Am Diet Assoc. 2005;105:54-63.
5. Garhart MT, Korman CF, Montgomery A, et al. Health Products for Seniors. "Anti-Aging" Products Pose Potential for Physical and Economic Harm. General Accounting Office Report to Chairman, Special Committee on Aging, U.S. Senate. In: General Accounting Office U, ed.: General Accounting Office; 2001.
6. Kim SH, Han JH, Keen CL. Vitamin and mineral supplement use by healthy teenagers in Korea: motivating factors and dietary consequences. Nutrition. 2001;17:373-80.
7. Archer SL, Stamler J, Moag-Stahlberg A, et al. Association of dietary supplement use with specific micronutrient intakes among middle-aged American men and women: the INTERMAP Study. J Am Diet Assoc. 2005;105:1106-14.
8. Grosvenor MB, Smolin LA. Nutrition. From Science to Life. Orlando, FL, USA: Harcourt College Publishers; 2002.
9. Fernandes AR, Rose M, White S, et al. Dioxins and polychlorinated biphenyls (PCBs) in fish oil dietary supplements: occurrence and human exposure in the UK. Food Addit Contam. 2006;23:939-47.
10. Saper RB, Phillips RS, Sehgal A, et al. Lead, mercury, and arsenic in US- and Indian-manufactured Ayurvedic medicines sold via the Internet [Erratum, JAMA 2008;300:1652]. JAMA. 2008;300:915-23.
11. Ross EA, Szabo NJ, Tebbett IR. Lead content of calcium supplements. JAMA. 2000;284:1425-9.
12. Reid IR, Ames R, Mason B, et al. Randomized controlled trial of calcium supplementation in healthy, nonosteoporotic, older men. Arch Intern Med. 2008;168:2276-82.

13. Reid IR, Ames R, Mason B, et al. Effects of calcium supplementation on lipids, blood pressure, and body composition in healthy older men: a randomized controlled trial. Am J Clin Nutr. 2010;91:131-9.
14. Bjelakovic G, Nikolova D, Simonetti RG, Gluud C. Antioxidant supplements for preventing gastrointestinal cancers.[update of Cochrane Database Syst Rev. 2004;(4):CD004183; PMID: 15495084]. Cochrane Database Syst Rev. 2008:CD004183.
15. Bardia A, Tleyjeh IM, Cerhan JR, et al. Efficacy of antioxidant supplementation in reducing primary cancer incidence and mortality: systematic review and meta-analysis. Mayo Clin Proc. 2008;83:23-34.
16. Omenn GS, Goodman GE, Thornquist MD, et al. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease. N Engl J Med. 1996;334:1150-5.
17. Vlad SC, LaValley MP, McAlindon TE, Felson DT. Glucosamine for pain in osteoarthritis: why do trial results differ? Arthritis Rheum. 2007;56:2267-77.
18. Reginster JY, Bruyere O, Neuprez A. Current role of glucosamine in the treatment of osteoarthritis. Rheumatology (Oxford). 2007;46:731-5.
19. Hooper L, Thompson RL, Harrison RA, et al. Risks and benefits of omega 3 fats for mortality, cardiovascular disease, and cancer: systematic review. BMJ. 2006;332:752-60.
20. Wang C, Harris WS, Chung M, et al. n-3 Fatty acids from fish or fish-oil supplements, but not alpha-linolenic acid, benefit cardiovascular disease outcomes in primary- and secondary-prevention studies: a systematic review. Am J Clin Nutr. 2006;84:5-17.
21. Riediger ND, Othman RA, Suh M, Moghadasian MH. A systemic review of the roles of n-3 fatty acids in health and disease. J Am Diet Assoc. 2009;109:668-79.
22. Marchioli R, Barzi F, Bomba E, et al. Early protection against sudden death by n-3 polyunsaturated fatty acids after myocardial infarction: time-course analysis of the results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI)-Prevenzione. Circulation. 2002;105:1897-903.
23. Wilkinson P, Leach C, Ah-Sing EE, et al. Influence of alpha-linolenic acid and fish-oil on markers of cardiovascular risk in subjects with an atherogenic lipoprotein phenotype. Atherosclerosis. 2005;181:115-24.
24. Kelley DS, Siegel D, Vemuri M, Mackey BE. Docosahexaenoic acid supplementation improves fasting and postprandial lipid profiles in hypertriglyceridemic men. Am J Clin Nutr. 2007;86:324-33.
25. Vieth R. Critique of the considerations for establishing the tolerable upper intake level for vitamin D: critical need for revision upwards. J Nutr. 2006;136:1117-22.
26. Kraemer K, Waelti M, de Pee S, et al. Are low tolerable upper intake levels for vitamin A undermining effective food fortification efforts? [Erratum, Nutr Rev 2008;66:610]. Nutr Rev. 2008;66:517-25.
27. Diplock AT. Safety of antioxidant vitamins and beta-carotene. Am J Clin Nutr. 1995;62:1510S-6S.
28. Albertazzi P, Steel SA, Clifford E, Bottazzi M. Attitudes towards and use of dietary supplementation in a sample of postmenopausal women. Climacteric. 2002;5:374-82.
29. Canter PH, Ernst E. Herbal supplement use by persons aged over 50 years in Britain: frequently used herbs, concomitant use of herbs, nutritional supplements and prescription drugs, rate of informing doctors and potential for negative interactions. Drugs Aging. 2004;21:597-605.

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Patterns of chronic pain in the New Zealand population

Clare Dominick, Fiona Blyth, Michael Nicholas

Abstract

Aim This study describes the prevalence and impact of chronic and recent pain in the New Zealand population and the groups most likely to report and use treatment for their chronic pain. Results are compared with international estimates.

Methods Data from the 2006/07 New Zealand Health Survey were analysed covering chronic pain, sociodemographic characteristics, chronic pain treatment use and health related quality of life. Prevalence estimates are presented. Chi-squared and logistic regression analyses were used to identify factors most strongly associated with chronic pain.

Results One in six (16.9%) New Zealanders reported chronic pain. Prevalence increased with age from 8.6% to 28.1%. People in the lowest two levels of three economic living standards categories had much higher adjusted odds (3.5 and 1.9) of reporting chronic pain than those with high economic living standards. Pacific and Asian peoples had much lower odds of reporting chronic pain compared with European/Other. Over a third (36%) did not use any treatment for their chronic pain while nearly half (48%) used some form of medical treatment. People with greater severity of recent pain, women and older age groups had much higher odds of using medical treatment for their chronic pain. A substantial minority did not report any treatment for their chronic pain. Higher numbers of chronic pain sites and greater severity of recent pain were associated with much lower scores across all the SF-36 physical and mental health domains.

Conclusions Patterns of chronic pain in the New Zealand are similar to those found internationally and indicate that chronic pain represents a major health issue in New Zealand.

Internationally chronic pain is recognised as a major health problem that has considerable impact individually, socially and economically.[1–3] It is viewed as meeting the criteria to be a condition in its own right, rather than a symptom of other conditions.[3,4] Chronic pain has received little attention compared with other long term conditions of similar prevalence and impact on individuals' function and health related quality of life.

Chronic pain is a complex biopsychosocial condition influenced by a wide range of psychosocial factors[5]. Pain is defined as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage' and chronic pain is generally viewed as pain that persists beyond the normal time of healing.[6,7]

Internationally, general population surveys have been used to investigate the prevalence of chronic pain and variation in overall prevalence estimates is apparent across countries ranging from 10% to 30%.[7–11]

Differences in prevalence estimates by sociodemographic characteristic are commonly found for age, gender, ethnicity and socioeconomic status.[7,9–13] Chronic pain prevalence increases with age.[10,12,14] Those in lower socioeconomic groups have been found to have higher chronic pain prevalence estimates, this includes individual and area level measures of socioeconomic difference.[8,12,15,16]

People with chronic pain have poorer health related quality of life and limitations in daily functioning. The severity of chronic pain and number of pain sites are associated with poorer physical and mental health and greater reductions in function.[17–22] Recent research has found an association between the severity of chronic pain (defined by both pain intensity and pain related disability) with increased risk of mortality.[23]

The prevalence and impact of chronic pain in the New Zealand population has remained largely unexamined since James' (1991) study,[24] due to a dearth of population level information. Although a more recent study conducted within the context of the World Mental Health Surveys estimated chronic pain prevalence in New Zealand, the operationalised definition diverged from other current chronic pain population surveys,[13,25] as it equated several diagnosed chronic conditions with chronic pain as part of the measure of chronic pain, rather than identifying chronic pain independent of underlying cause.

Given the international evidence showing the personal and societal impact of chronic pain, it is therefore pertinent to examine the current patterns of chronic pain within the New Zealand population and the relationship of these patterns with international evidence. The 2006/07 New Zealand health survey (NZHS) provided this opportunity as it included questions about chronic pain. The aim of this article was to describe the prevalence of chronic and recent pain in the New Zealand population, determine the strength of its association with a range of sociodemographic factors, describe treatment use for chronic pain, and describe the impact of chronic pain on health related quality of life for New Zealanders.

Methods

The data for this analysis came from the 2006/07 NZHS which is a nationally representative cross-sectional survey of 12,488 New Zealand adult residents, aged 15 years and over. The weighted survey response rate was 67.9% and participants included 3160 Māori, 1033 Pacific, 1513 Asian, and 8593 European/Other adults. The Ministry of Health commissioned survey was conducted from October 2006 to November 2007. Details of the sample, methods, and ethical approval were published by the Ministry of Health in 2008.[26,27] The Ministry of Health confidentialised the data provided for this analysis.

Respondents were asked whether they experienced chronic pain; to identify the site(s) of chronic pain; and for each site, to state the age of pain onset, the treatments they currently receive for the pain, and their attributed cause of the pain.

The definition of chronic pain used in the questionnaire is

> "pain that is present almost every day, but the intensity of the pain may vary. … pain that has lasted or is expected to last 6 months or more."

Respondents identified their pain sites from a show card list of nine and were able to nominate additional sites. The listed sites were head, neck, face jaw or joint below the ear, teeth or gums, back, chest, stomach, pelvic region, joints (fingers, wrists, elbows, shoulders, hips, and knees), Other. Three new site codes (legs, feet and arms) were derived from 'Other' nominations and included in the data set.

Data on severity of chronic pain was not available, however, the Medical Outcomes Study Short Form 36 (SF-36)[28] had an item asking respondents about the intensity of pain they had experienced in the

past 4 weeks. This has been used as a measure of recent pain severity. The number of reported chronic pain sites has also been used to differentiate groups within the chronic pain population, as the number of pain sites is associated with health related quality of life and functional outcomes[18–20].

In summary, three measures of pain were used in this analysis:

- Whether chronic pain was reported or not.
- The number of chronic pain sites (0, 1, 2, 3 or more).
- The intensity of bodily pain reported in the past four weeks of which two groupings are included (1) 'No to mild' versus 'moderate to very severe' recent pain and (2) 'No to moderate' versus 'severe or very severe' recent pain.

Sociodemographic characteristics included in the analysis were age, gender, ethnicity, economic living standards, employment status, whether living alone or not, and rural or urban residence. Survey respondents were able to nominate multiple ethnic identifications. Both multiple identification and prioritised ethnicity categorisations were used in analysis. Prioritised ethnicity was used for the logistic regression analyses. It includes only one ethnic category nominated by the respondent, according to a predetermined hierarchy. The four categories used for prioritised and multiple ethnic identification categories are Māori, Pacific, Asian, and European/Other, in order of prioritisation.

The Economic Living Standards Index (ELSI) has been used as the primary measure of socioeconomic status in this analysis. It is an individual measure of economic living standards which was developed and validated within the New Zealand population. It records restrictions in the ownership of possessions and social participation due to cost, day to day economising behaviour, and self-ratings of current economic position.[29]

The SF-36 was used as the measure of health related quality of life.[28] It has 36 items covering physical and mental health status in relation to eight health domains: physical functioning, role limitation (physical), bodily pain, general health perceptions, vitality (energy/fatigue), social functioning, role limitation (emotional), and general mental health. Responses to each of the SF-36 items are scored, and expressed on a 0–100 scale for each of the eight health domains.

Prevalence estimates and weighted population estimates (not sample frequency) along with their associated 95% confidence intervals are presented. Analyses were conducted with SAS version 9.2.[30] The surveyfreq, surveymeans and surveylogistic procedures were used with Taylor Series estimation. Results were weighted using the calibrated weight calculated by the Ministry of Health,[26,27] which took into account the inverse probability of selection and New Zealand population counts by age, gender, ethnicity and DHB area. Chi-squared analyses and logistic regression were used to identify the factors most strongly associated with chronic pain.

Two logistic regression models are presented. The first model incorporates all survey respondents and has chronic pain status as the dependent variable and sociodemographic characteristics as independent variables.

The second model considers only those respondents reporting chronic pain, and use of medical treatment for pain is the dependent variable. Sociodemographic characteristics and recent pain severity are the independent variables.

Odds ratio estimates are reported for each of the models and each of the odds estimates controls for all other factors included in the model. Results were checked using the Jackknife variance estimation method. Chronic pain site location is described using a simple count of nominations. Levels of missing data were very low (less than or equal to 1%) across all analyses included in this article.

Results

Overall, 16.9% (16.1–17.8) of the New Zealand adult population (aged 15 years and over) reported chronic pain and 22.6% (21.6–23.5) of the adult population had experienced moderate to severe recent pain. Nearly two-thirds (63.6%) of those who reported chronic pain reported moderate to severe recent pain (Table 1) while a fifth (21.4%) reported severe or very severe recent pain (table not presented).

Table 1. Chronic pain status by intensity of recent pain (moderate to very severe)

Chronic pain status	Recent Pain status	Weighted Frequency (NZ Pop)	Percent (NZ Pop)	95% CI	Row Percent	95% CI row
Chronic pain	None – Mild	192,182	6.2	(5.6–6.7)	36.4	(33.9–38.9)
	Moderate – Very severe	335,828	10.8	(10.1–11.4)	63.6	(61.1–66.1)
	Total	528,010	16.9	(16.1–17.8)	100	
No chronic pain	None – Mild	2,224,262	71.3	(70.3–72.3)	85.8	(84.9–86.7)
	Moderate – Very severe	368,071	11.8	(11.1–12.5)	14.2	(13.3–15.1)
	Total	2,592,332	83.1	(82.2–83.9)	100	(33.9–38.9)
Total	None – Mild	2,416,444	77.4	(76.5–78.4)		
	Moderate – Very severe	703,899	22.6	(21.6–23.5)		
	Total	**3,120,343**	**100**			

Chronic pain prevalence increased with age from a low of 8.6% for 15 to 24 year olds to a high of 28.1% for those aged 75 years and over. In New Zealand, unlike patterns internationally, chronic pain prevalence did not differ significantly between males and females except for the 65 to 74 year age group ($p<0.01$) (Table 2). The overall unadjusted prevalence for females was 17.7% (16.6–18.8) and for males, 16.1% (14.8–17.4).

Table 2. Prevalence of chronic pain by age group and gender

Age Group (years)	Females			Males		
	Percentage of age group	95% CI	Weighted frequency (NZ Pop)	Percentage of age group	95% CI	Weighted frequency (NZ Pop)
15–24	8.0	(5.8–10.2)	22,011	9.1	(6.2–12.1)	25,400
25–34	11.9	(9.5–14.2)	31,756	11.6	(8.6–14.6)	27,805
35–44	14.1	(12–16.3)	45,647	16.1	(13.3–18.8)	46,111
45–54	20.5	(17.6–23.5)	58,776	17.7	(14.5–21.0)	47,796
55–64	24.0	(20.7–27.3)	51,017	20.3	(17.0–23.7)	41,733
65–74	30.2	(26.1–34.3)	41,973	22.9	(19.1–26.7)	29,558
75+	30.0	(25.7–34.3)	35,694	25.6	(20.7–30.6)	22,732

Respondents who identified as European/Other were more likely to report chronic pain than those who did not identify as European/Other, while respondents who identified as Pacific or Asian were less likely to report chronic pain than those who did not identify with those ethnic groups (Table 3).

Results for Māori depended on the comparison group used in the analysis. Māori were equally as likely to report chronic pain as those who did not identify as Māori (Table 3). However, when the total population was used as a comparator and age and gender controlled, Māori had higher prevalence rates. In most analyses, prevalence rates for Māori were not significantly different from the prevalence in the European/Other population after controlling for other sociodemographic factors.

Table 3. Chronic pain prevalence by ethnic identification (all identifications)

Ethnic identification	Chronic pain status	Weighted frequency (NZ Pop)	Row %	95%CI row
Asian identification[1]	Chronic pain	26,705	9.6	(7.8–11.3)
No Asian identification	Chronic pain	501,305	17.6	(16.8–18.5)
European /Other ethnic identification[2]	Chronic pain	460,975	18.1	(17.1–19.0)
No European /Other identification	Chronic pain	67,035	11.8	(10.5–13.0)
Māori identification[3]	Chronic pain	61,412	17.3	(15.5–19.1)
No Māori identification	Chronic pain	466,599	16.9	(16.0–17.8)
Pacific identification[4]	Chronic pain	19,775	12.0	(9.6–14.4)
No Pacific identification	Chronic pain	508,235	17.2	(16.3–18.1)

[1] Asian versus No Asian identification significant difference p<0.0001

[2] European/Other versus No European/Other identification significant difference p<0.0001

[3] Māori versus No Māori identification not significant

[4] Pacific versus No Pacific identification significant difference p<0.0004

Chronic pain prevalence was strongly associated with economic living standards (ELS). The proportion of people who reported chronic pain increased as ELS decreased (p<0.0001) (Figure 1). This relationship was also found for those reporting moderate to severe recent pain compared with those reporting no or mild recent pain (p<0.0001).

Chi-squared analysis indicated that domiciliary status (living alone) and employment status (those not in work and not looking for work) were associated with chronic pain status (p<0.0001). The association with living alone did not remain significant when other sociodemographic factors were taken into account.

The number of chronic pain sites reported ranged from 0 to 9. Combining all nominations, the most frequently nominated sites were Joints (29.9% of all nominations), Back (24.1%), Neck (12%), Pelvic Region (8%), Head (6.9%), Stomach (5.8%), and Chest (3.9%).

Nearly two-thirds (64.7%) of those experiencing chronic pain reported only one pain site (Table 4). Higher numbers of sites were reported by older age groups (p<0.0001). Women reported greater numbers of chronic pain sites than men (p<0.0001). Respondents reporting greater numbers of sites were also more likely to report greater severity of recent pain (p<0.0001). Two-thirds (67%) of those reporting chronic pain had lived with chronic pain for 5 or more years and a quarter (27%) had lived with chronic pain for 40% or more of their lives.

Figure 1. Prevalence of chronic pain by Economic Living Standard Index

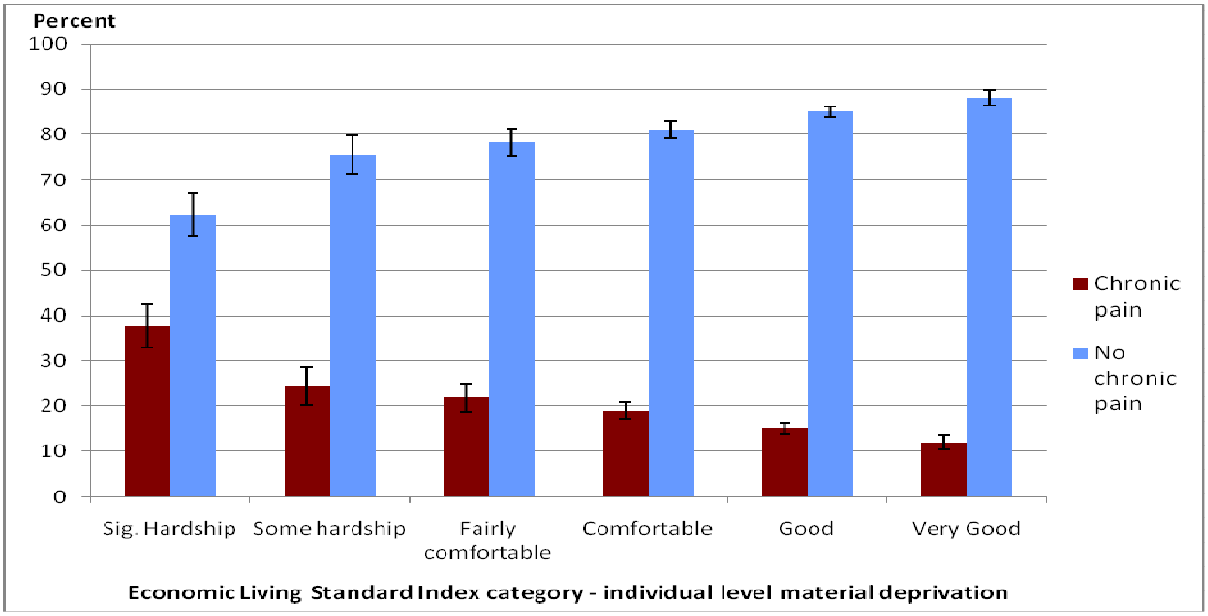


Table 4. Number of chronic pain sites

Number of chronic pain sites	Weighted frequency (NZ pop)	Percent (Chronic pain pop in NZ)	95% CI
1	341,731	64.7	(62.2–67.3)
2	103,569	19.6	(17.6–21.7)
3 or more	82,710	15.7	(13.7–17.6)
Total	**528,010**	**100**	

Table 5. Percentage of chronic pain population by attributed reason for their chronic pain (First pain site recorded)

Reason	Weighted frequency (NZ pop)	Percent (Chronic pain pop in NZ)	95% CI
Age-related	58,507	11.1	(9.5–12.6)
Health condition	146,375	27.7	(25.4–30.1)
Injury or accident	219,296	41.5	(39.0–44.1)
Work-related	21,782	4.1	(3.1–5.2)
Operation	16,037	3.0	(2.2–3.9)
Lifestyle or habit	14,207	2.7	(1.8–3.6)
Physical activity	6,642	1.3	(0.7–1.8)
Other	26,643	5.1	(4.0–6.1)
Not known	18,522	3.5	(2.5–4.5)

Survey respondents were asked what they thought caused their chronic pain. The most frequent attributions (concerning the first pain site recorded in the interview) were that their chronic pain was related to an injury or accident (42%), a health condition (28%) or age (11%) (Table 5). Men were more likely than women to attribute the

cause of their chronic pain to an injury or accident and women more likely than men to attribute it to a health condition.

Table 6. Odds ratio estimates for model of chronic pain in relation to sociodemographic characteristics

Effect for sociodemographic variables (adjusted for other variables in model)		Odds ratio Point Estimate	95% Wald CI	P value
Economic living standard (ELS)	Hardship vs Good/very good Comfortable vs Good/very good	3.5 1.9	(2.9-4.2) (1.7-2.2)	<.0001 <.0001
Age group	45–64 vs 15–44 65–99 vs 15–44	2.0 2.8	(1.8–2.4) (2.3–3.3)	<.0001 <.0001
Ethnicity	Asian vs European/Other Māori vs European/Other Pacific vs European/Other	0.5 0.9 0.5	(0.4–0.6) (0.8–1.1) (0.4–0.7)	<.0001 0.292 <.0001
Domiciliary status	Lives alone vs Lives with other(s)	1.0	(0.9–1.2)	0.6732
Gender	Females vs Males	1.0	(0.9–1.2)	0.8401
Employment	Looking for vs In work Other vs In work	1.0 1.2	(0.8–1.4) (1.1–1.4)	0.8774 0.0025
Urban domicile	Not major urban vs Major urban	1.1	(1.0–1.3)	0.0536

Logistic regression results showed that economic living standards (ELS), age and ethnicity were strongly associated with reporting chronic pain when other sociodemographic characteristics were controlled. Those with low ELS ('hardship' categories) had 3.5 higher odds of experiencing chronic pain than those with high ELS ('good' or 'very good' categories). Those with moderate ELS ('comfortable' categories) had 1.9 higher odds of experiencing chronic pain compared with those with high ELS (Table 6).

Those aged 45 to 64 years had twice the odds and those aged 65 years and over had 2.8 higher odds of reporting chronic pain compared with 15 to 44 year olds, when controlling for other sociodemographic factors. Using prioritised ethnicity categories, logistic regression results showed that those identifying as Pacific or Asian had lower odds of reporting chronic pain compared with European/Other and that the odds of Māori reporting chronic pain was the same as for European/Other. Those 'not in work and not looking for work' at the time of the interview had 1.2 higher odds of reporting chronic pain.

Respondents were asked about the treatments they currently used for their chronic pain. Considering the first pain site only, over a third (36%) of those with chronic pain did not use any treatments for their pain while 40% mentioned 'medical' treatments only. A further sixth (16.1%) used a range of other treatments and a smaller percentage (7.9%) used a combination of 'medical' and 'other' treatments (Table 7). Over half (53.8%) of those who reported 'moderate to very severe' recent pain had used some medical treatments for their chronic pain compared with just over a third (37.7%) of those who reported 'no to mild' recent pain ($p<0.0001$) (Table 7).

Those who reported 'severe or very severe' recent pain were more likely to have used some medical treatments (63.6%) for their chronic pain compared with those who reported 'no to moderate' recent pain (43.7%) ($p<0.0001$). A quarter (24.7%) of those reporting 'severe or very severe' recent pain were not using any form of treatment for their chronic pain.

Table 7. Severity of recent pain by current treatments for first chronic pain site recorded in interview

Severity of recent pain	Treatments for chronic pain site	Weighted frequency (NZ pop)	Percent (chronic pain pop)	95% CI	Row %	95%CI row
None to mild	Medical[1]	61,845	11.7	(10–13.4)	32.2	(28.1–36.3)
	Other[2]	35,986	6.8	(5.4–8.2)	18.7	(15.2–22.3)
	Medical and Other	10,664	2.0	(1.2–2.8)	5.5	(3.4–7.7)
	No treatment	83,688	15.8	(13.9–17.8)	43.5	(39.1–48)
	Total	192,182	36.4	(33.9–38.9)	100	
Moderate to very severe	Medical[1]	149,926	28.4	(26.1–30.7)	44.6	(41.5–47.8)
	Other[2]	48,940	9.3	(7.7–10.8)	14.6	(12.2–16.9)
	Medical and Other	30,821	5.8	(4.6–7.1)	9.2	(7.3–11.1)
	No treatment	106,141	20.1	(18.1–22.1)	31.6	(28.7–34.5)
	Total	335,828	63.6	(61.1–66.1)	100	
Total	Medical[1]	211,771	40.1	(37.6–42.6)		
	Other[2]	84,926	16.1	(14.1–18.1)		
	Medical and Other	41,485	7.9	(6.4–9.3)		
	No treatment	189,829	36.0	(33.5–38.4)		

[1] 'Medical' treatment categories include medicines/tablets/pills, injections, or waiting for surgery.

[2] 'Other' treatment categories in the survey data include Exercise or physiotherapy, Osteopathy, Chiropractor, Complementary or alternative treatments, Diet/diet control, Dietary supplements, Sleep/rest, Health treatment/hot baths, Improved footwear, Cream, or Other.

Logistic regression results (Table 8), with the dependent variable 'using medical treatment', showed that those who reported 'moderate to very severe' recent pain had 1.8 higher odds of using medical treatment compared with those who had experienced 'no to mild' recent pain when controlling for sociodemographic factors. Women had 1.8 higher odds of using medical treatment compared with men. Those aged 45 to 64 years and those aged 65 years and over had 1.8 and 1.7 higher odds respectively of using medical treatment compared with 15 to 44 year olds.

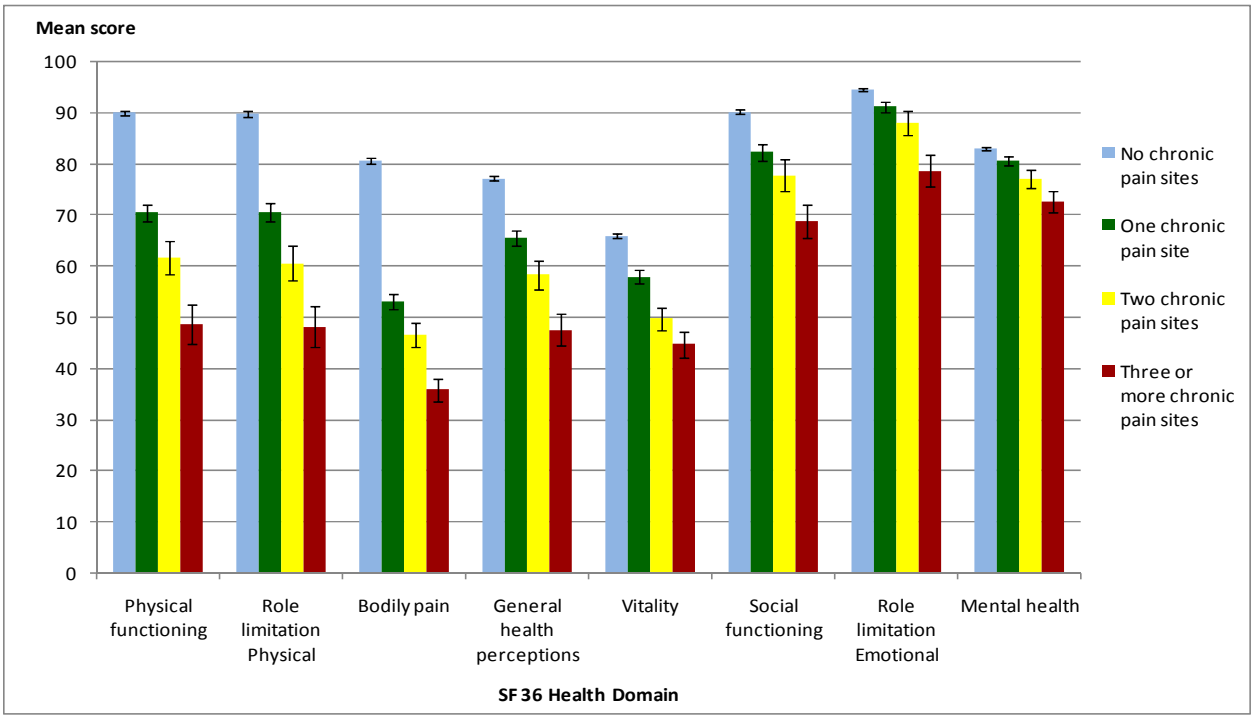
The odds of using medical treatment for chronic pain when reporting 'severe to very severe' recent pain were 2.1 higher than those who reported 'no to moderate' recent pain.

Significant differences were found between SF-36 mean scores for those reporting and those not reporting chronic pain ($p<0.001$). Respondents reporting chronic pain had significantly lower SF-36 scores (poorer health related quality of life) across all SF-36 domains in comparison with those not reporting chronic pain.

Table 8. Odds ratio estimates model of likelihood of using medical treatment for first chronic pain site (chronic pain population)

Effect for sociodemographic variables and recent pain (adjusted for other variables in model)		Odds Ratio Point Estimate	95% Wald CI	P value
Recent pain	Moderate to very severe vs None to mild	1.8	(1.4–2.2)	<.0001
Gender	Females vs Males	1.8	(1.5–2.3)	<.0001
Age group	45–64 vs 15–44 65–99 vs 15–44	1.8 1.7	(1.3–2.3) (1.2–2.4)	<.0001 0.0038
Employment	Looking for vs In work Other vs In work	1.5 1.3	(0.9–2.4) (1.0–1.8)	0.1338 0.0402
Ethnicity	Asian vs European/Other Māori vs European/Other Pacific vs European/Other	1.1 0.8 1.3	(0.7–1.8) (0.6–1.0) (0.8–2.3)	0.6151 0.0739 0.3173
Economic living standard (ELS)	Hardship vs Good/very good Comfortable vs Good/very good	0.9 0.9	(0.7-1.3) (0.7–1.1)	.073 0.2162
Urban domicile	Not urban vs Major urban	0.9	(0.7–1.1)	0.2197

Figure 2: Health-related quality of life (SF-36 domain mean score) by number of chronic pain sites



The number of chronic pain sites was also directly associated with health related quality of life. Higher numbers of chronic pain sites were associated with lower SF-36 scores across all physical and mental health domains (Figure 2).

The severity of recent pain was also associated with health related quality of life. Those reporting 'moderate to severe' recent pain had lower SF-36 scores than those

reporting ‘no to mild’ recent pain. This was the case for both those reporting and not reporting chronic pain ($p<0.0001$).

Discussion

Results from this analysis show that chronic pain affects one in six adult New Zealanders. This is slightly lower than estimates found in Australia and several European countries (19%),[8–10,31] but higher than that found in some Asian countries (<11%).[2] Internationally some variation in estimates is expected due to variation in the definitions of chronic pain and the methods used.[7,11] The NZHS defined chronic pain as pain lasting for six months whereas most of the comparison studies’ definitions used three months duration. NZHS results were weighted to accord with the New Zealand population demographics.

The use of these definitions and methods may explain the slightly lower prevalence estimate compared with Australian and European studies, but not with Asian countries. The lower prevalence estimate found for Asian ethnic groups is consistent with prevalence estimates found in Hong Kong[2] and Malaysia (unpublished report). Lower prevalence estimates for Pacific people were also found in the New Zealand Mental Health survey, although as the operationalised definition diverged from other population surveys of chronic pain,[25] the absolute value was higher than that found in this survey. These results in conjunction with those from other surveys suggest there may be more systematic variation in reporting of chronic pain across sociocultural groups which warrants more attention.

Overall prevalence in New Zealand did not vary significantly by gender, although there was a preponderance of women reporting chronic pain in older age groups, and women were more likely than men to report multiple pain sites. This contrasts with findings in Australia, Denmark and Norway where lower estimates were obtained for men (17.1%, 16%, 23.3%) compared with women (20%, 21%, 27.6%).[8,10,31] As expected, chronic pain prevalence in New Zealand increased with age.

Several significant relationships were found, but, as results derive from a cross-sectional survey, only associative and not causal relationships have been determined. Chronic pain was strongly associated with economic living standards (ELS). In line with evidence internationally, the odds of reporting chronic pain were much greater for people with low ELS compared with those with higher ELS. Associations between measures of socioeconomic status and health outcomes are well established. However, the contribution of social mechanisms to chronic pain outcomes is less well explored.[32]

In this analysis, recent pain severity was measured using a single item verbal descriptor scale of pain intensity and any interpretation needs to bear in mind there are a range of definitions of pain severity. This also limits comparison with other general population surveys. A fifth (21.4%) of respondents reporting chronic pain experienced severe or very severe recent pain at the time of the interview.

In New Zealand, two-thirds of those reporting chronic pain had experienced chronic pain for 5 or more years. The impact of chronic pain on health related quality of life was dramatic with much poorer health related quality of life associated with those reporting chronic pain.

Similar to results found internationally,[17–22] higher numbers of chronic pain sites and greater pain severity were associated with greater reductions in health related quality of life. These results support the proposition that number of chronic pain sites is a useful measure of population risk of poorer health outcomes.[33] Measurement, coding and population sociodemographic differences are likely contributors to the lower number of sites reported in this study compared with studies internationally.[33–35]

In the NZHS, chronic pain sites were recorded if a respondent had experienced pain every day for six months (allowing for varying intensity). In contrast, for example, the Ullenskar study recorded musculoskeletal pain sites experienced in the past seven days and the past 12 months.[33,35] In addition, chronic pain in joints was recorded only once in the NZHS even if multiple joints were affected. These were coded separately in the Ullenskar study and back pain had two codes (upper and lower back).

Joints, back and neck were the most frequently nominated sites in this study. Consistent with this analysis, an analysis of chronic pain site locations from an individual perspective showed that over half (57.6%) of those with chronic pain reported chronic pain in joints and just under half reported chronic pain in the neck or back (47.5%).[26]

While nearly half of those who reported chronic pain used some form of medical treatment (defined as medicines, pills, tablets, injections, or waiting for surgery) for their chronic pain, a third did not use any treatment.

Use of medical treatment did vary within the chronic pain population. People with greater severity of recent pain, women, and older age groups had much higher odds of using medical treatment for their chronic pain. A substantial minority of those experiencing severe recent pain did not use any form of treatment for their chronic pain.

The reasons for this pattern of treatment utilisation are uncertain. Individuals may have developed their own management approaches which do not include the use of health services or it might be that management is provided during health consultations for other reasons. Research in Australia showed that people reporting chronic pain were more likely to use health services and that higher use was associated with greater levels of pain related activity interference.[36] A planned analysis of overall health service use alongside the analysis of the treatment reported specifically for chronic pain should help clarify potential explanations.

These results show that chronic pain is a major health issue in New Zealand. Although people in all age groups are affected by chronic pain, the proportion of people affected by chronic pain will increase as the population ages in coming years. Recent New Zealand studies and commentary have highlighted the need for improvements in the policy and service delivery for chronic conditions. [37–39]

The recently released Australian National Pain Strategy highlighted the heavy burden of pain on the community, economy and health care services and called for improvements in its assessment and management.[40] With the recognition that chronic pain is a common chronic condition that meets the definition of a disease, it would be useful to examine the extent to which it is accorded priority for funding and services in New Zealand and the adequacy of those services in relation to population need.

Competing interests: None known

Author information: Clare Dominick,[1] Fiona Blyth,[2,3] Michael Nicholas[4]

[1]PhD Candidate, Pain Management and Research Institute, University of Sydney (based in Wellington, New Zealand)

[2]Head, Pain Epidemiology Unit, Pain Management and Research Institute, University of Sydney, Australia;

[3]School of Public Health, University of Sydney, Australia

[4]Associate Professor, Pain Management and Research Institute, University of Sydney, Australia

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Correspondence: Clare Dominick, PO Box 27-115, Wellington, New Zealand. Email: cdom1713@uni.sydney.edu.au

References:

1. Access Economics PTY Ltd. The high price of pain: the economic impact of persistent pain in Australia. Sydney: MBF Foundation; 2007.
2. Ng KF, Tsui SL, Chan WS. Prevalence of common chronic pain in Hong Kong adults. Clinical Journal of Pain 2002;18:275-81.
3. Siddall PJ, Cousins MJ. Persistent Pain as a Disease Entity: Implications for Clinical Management. Anesth Analg 2004;99:510-20.
4. Tracey I, Bushnell MC. How Neuroimaging Studies Have Challenged Us to Rethink: Is Chronic Pain a Disease? The Journal of Pain 2009;10:1113-20.
5. Turk DC, Okifuji A. Psychological factors in chronic pain: Evolution and revolution. Journal of Consulting and Clinical Psychology 2002;70:678-90.
6. IASP. Classification of chronic pain. Description of pain syndromes and definition of pain terms. Pain 1986;Supp.
7. Verhaak PF, Kerssens JJ, Dekker J, Sorbi MJ, Bensing JM. Prevalence of chronic benign pain disorder among adults: a review of the literature. Pain 1998;77:231-9.
8. Blyth FM, March LM, Brnabic AJM, Jorm LR, Williamson M, Cousins MJ. Chronic Pain in Australia: A prevalence study. Pain 2001;89:127-34.
9. Breivik H, Collett B, Ventafridda V, et al. Survey of chronic pain in Europe: prevalence, impact on daily life, and treatment. European Journal of Pain: EJP 2006;10:287-333.
10. Eriksen J, Jensen MK, Sjogren P, Ekholm O, Rasmussen NK. Epidemiology of chronic non-malignant pain in Denmark. Pain 2003;106:221-8.
11. Ospina M, Harstall C. Prevalence of chronic pain: an overview: Alberta Heritage Foundation for Medical Research; 2002 December 2002.
12. Sjogren P, Ekholm O, Peuckmann V, Gronbaek M. Epidemiology of chronic pain in Denmark: An update. European Journal of Pain: Ejp 2009;13:287-92.
13. Tsang A, Von Korff M, Lee S, et al. Common chronic pain conditions in developed and developing countries: gender and age differences and comorbidity with depression-anxiety disorders. Journal of Pain 2008;9:883-91.
14. Catala E, Reig E, Artes M, Aliaga L, Lopez JS, Segu JL. Prevalence of pain in the Spanish population: telephone survey in 5000 homes. European Journal of Pain: Ejp 2002;6:133-40.

15. Jordan KP, Thomas E, Peat G, Wilkie R, Croft P. Social Risks for disabling pain in older people: A prospective study of individual and area characteristics. Pain 2008;137:652-61.
16. Saastamoinen P, Leino-Arjas P, Laaksonen M, Lahelma E. Socio-economic differences in the prevalence of acute, chronic and disabling chronic pain among ageing employees. Pain 2005;114:364-71.
17. Gerdle B, Bjork J, Henriksson C, et al. Prevalence of current and chronic pain and their influences upon work and healthcare-seeking: a population study. Journal of Rheumatology 2004;31:1399-406.
18. Gureje O, Von Korff M, Kola L, Demyttanaere K, He Y, et al. The relation between multiple pains and mental disorders: Results from the World Mental Health Surveys. Pain 2008;135:82-91.
19. Karoly P, Ruehlman LS. Psychosocial aspects of pain-related life task interference: an exploratory analysis in a general population sample. Pain Medicine 2007;8:563-72.
20. Croft P. The question is not "have you got it"? But "how much of it have you got"? Pain 2009;141:6-7.
21. Smith BH, Elliott AM, Chambers WA, Smith WC, Hannaford PC, Penny K. The impact of chronic pain in the community. Family Practice 2001;18:292-9.
22. Kamaleri Y, Natvig B, Ihlebaek CM, Bruusgaard D. Does the number of musculoskeletal pain sites predict work disability? A 14-year prospective study. European Journal of Pain 2009;13:426-30.
23. Torrance N, Elliott AM, Lee AJ, Smith BH. Severe chronic pain is associated with increased 10 year mortality. A cohort record linkage study. European Journal of Pain 2010;14:380-6.
24. James FR, Large RG, Bushnell JA, Wells JE. Epidemiology of pain in New Zealand. Pain 1991;44:279-83.
25. Scott K, McGee MA, Schaaf D, Baxter J. Mental-physical comorbidity in an ethnically diverse population. Social Science & Medicine 2008;66:1165-73.
26. Ministry of Health. A Portrait of Health: Key results of the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health; 2008.
27. Ministry of Health. Methodology Report for the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health; 2008.
28. Ware JE, Kosinski M, Gandek B. SF-36 Health Survey: Manual and Interpretation Guide.: Lincoln RI Quality Metric Incorporated; 2005.
29. Jensen J, Spittal M, Krishnan V. ELSI Short Form: User Manual for a Direct Measure of Living Standards. Wellington: Centre for Social Research and Evaluation, Ministry of Social Development; 2005.
30. SAS Institute Inc. SAS/STAT®9.2 User's Guide Cary, NC: SAS Institute Inc.; 2008.
31. Rustoen T, Wahl AK, Hanestad BR, Lerdal A, Paul S, Miaskowski C. Gender differences in chronic pain--findings from a population-based study of Norwegian adults. Pain Management Nursing 2004;5:105-17.
32. Blyth FM. Chronic pain. Is it a public health problem? Pain 2008;137:465-6.
33. Natvig B, Ihlebaek CM, Kamaleri Y, Bruusgaard D. Number of pain sites - simple measure of population risk? In: Croft P, Blyth FM, van der Windt D, eds. Chronic Pain Epidemiology: From Aetiology to Public Health. New York: Oxford University Press; 2010.
34. Croft P, Dunn KM. Chronic pain syndromes: You can't have one without another. Pain 2007;131:237-8.
35. Kamaleri Y, Natvig B, Ihlebaek CM, Benth JS, Bruusgaard D. Number of pain sites is associated with demographic, lifestyle, and health-related factors in the general population. European Journal of Pain 2008;12:742-8.
36. Blyth FM, March LM, Brnabic A, J. M., Cousins MJ. Chronic pain and frequent use of health care. Pain 2004;111:51-8.

37. National Advisory Committee on Health and Disability. Meeting the Needs of People with Chronic Conditions: Hapai te whanau mo ake ake tonu. Wellington New Zealand: National Advisory Committee on Health and Disability; 2007.
38. Shipton E, Shipton E. The pain epidemic: some proposed solutions. The New Zealand Medical Journal 2005;118(1221). http://www.nzmj.com/journal/118-1221/1627/content.pdf
39. Taylor W. Musculoskeletal pain in the adult New Zealand population: prevalence and impact. The New Zealand Medical Journal 2005;118(1221). http://www.nzmj.com/journal/118-1221/1629/content.pdf
40. National Pain Summit initiative. National Pain Strategy: Pain Management for all Australians. Melbourne: Faculty of Pain Medicine; 2010.

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Physical activity among cancer survivors: a literature review

Ewa A Szymlek-Gay, Rosalina Richards, Richard Egan

Abstract

Aim Physical activity offers a variety of health benefits to cancer survivors, both during and post-treatment. The aim here is to review: the preferences of cancer survivors regarding exercise counselling and participation in a physical activity programme; adherence rates among cancer survivors to physical activity programmes; and predictors of adherence to exercise training.

Methods Two electronic databases, Ovid MEDLINE(R) 1950 to Present with Daily Update and SCOPUS, were used to undertake literature searches for studies examining exercise preferences of adult cancer survivors, and physical activity programmes for adults at any point of the cancer trajectory.

Results Studies suggest that, while physical activity levels are low among cancer survivors, most are interested in increasing their participation. Preferences and adherence to physical activity programmes differ across a range of demographic, medical, and behavioural variables, suggesting the importance of tailoring exercise programmes to patient-specific and disease-specific needs.

Conclusions Current evidence supports the benefits of physical activity for improving risk factors associated with cancer prognosis. Physical activity programmes developed for oncology patients and cancer survivors need to take into account the needs of the target population in order to optimise adherence, outcomes, and long-term behavioural changes in this population.

Impact of cancer in New Zealand

Cancer is a major public health problem in New Zealand with approximately 19,000 new cases diagnosed annually.[1] In 2005, the age-standardised incidence of cancer was 340.3 cases per 100,000 persons, with the highest incidence recorded for Pacific males (389.3 cases per 100,000), and the lowest for non-Māori, non-Pacific females (308.1 cases per 100,000).[1] The cancers of breast; colorectum and anus; prostate; malignant melanoma of skin; and trachea, bronchus and lung are the most common cancers in New Zealand accounting for 61% of the incident cancers.[1]

Although cancer remains the leading cause of death in New Zealand, accounting for a third of all deaths,[1] survival rates have improved over the last two decades.[2] The overall cumulative relative 5-year survival rate across all cancers and all disease stages is currently estimated at 61%,[3] although the chances of survival are greater if diagnosed with early-stage disease.[3]

The number of survivors to 5 years in New Zealand is estimated to be approximately 60,000,[3] which represents 1.5% of the New Zealand population. Continued advances in early diagnosis and treatment are likely to further increase the number of cancer survivors.

As the population of cancer survivors in New Zealand grows, it is important to acknowledge that surviving cancer is associated with several distinct health issues. Compared with persons who have not had cancer, cancer survivors have an increased lifetime risk of developing new primary cancers,[4–6] cardiovascular disease,[6–9] diabetes,[8,9] osteoporosis,[10] and functional decline.[8,11,12]

Furthermore, the risk of cancer recurrence is high among cancer survivors.[13–18] These risks are believed to result from cancer treatment, genetic predisposition, or common lifestyle factors.[11,19–21] Most of these adverse physiological and quality of life outcomes can be reduced by healthy lifestyle practices, including regular exercise.[8,9]

Physical activity

Despite methodological limitations and modest sample sizes, existing evidence strongly suggests that physical activity both during and post-treatment can improve cardio-respiratory fitness,[22–26] flexibility,[12] muscular strength,[26] physiological outcomes,[22] vigour,[22,26] cancer-related fatigue[23,26–30] and other cancer-related symptoms,[22,28] nausea,[12] physical well-being,[12] physical functioning,[23,26,28,30] anxiety,[12] and overall quality of life.[12,23,25,30,31] Furthermore, physical activity appears to minimise functional decline in cancer survivors,[32] improve immune system function,[33] maintain or minimise bone loss,[34,35] and reduce cancer-related chronic diseases.[8,9]

Evidence from prospective observational studies suggests that regular physical activity is associated with improved cancer prognosis, although data are few and limited to breast and colon cancer survivors. Specifically, breast cancer survivors who engage in at least 8–10 metabolic equivalent (MET)-hours a week of physical activity (approximately 3 hours of walking per week at a moderate pace) compared with less active survivors have a 40% to 50% reduced risk of death from breast cancer,[36,37] a 24% to 67% decreased risk of overall mortality,[36–40] and a 26% to 43% decreased risk of recurrence of breast cancer,[37] although this finding is not unanimous.[40]

Similarly, physically active colon cancer survivors (at least 9 MET-hours/week) appear to have a 43% to 61% lower risk of colon cancer mortality compared with less active survivors,[41] and a 29% to 63% lower risk of overall mortality.[41,42] Physical activity may also protect against the development of primary cancers,[43] although this is less clear in cancer survivors.

Physical activity has been shown to play a role in weight management as it appears to positively affect body composition[12,24,28] and facilitate weight loss[44] in oncology patients and cancer survivors. This is of importance because being overweight may increase the risk of cancer recurrence and decrease survival for many cancers.[45-51] Furthermore, recent results from some,[50,52] but not all,[53] large prospective studies have indicated that weight gain after cancer diagnosis may be associated with poorer prognosis.

Indeed, weight gain post-diagnosis in large cohorts of breast cancer survivors appeared to increase the risk of death from breast cancer by up to 78%,[50,52] and the risk of breast cancer recurrence by up to 53%.[52] Furthermore, for each 5-kg gain in weight, the risk of mortality from breast cancer may increase by 13%.[50] This is of particular concern because weight gain is common in breast cancer survivors.[54–56]

Whether weight reduction in overweight or obese cancer survivors would improve the disease outcomes is still under debate. Kroenke et al. and Nichols et al. found that post-diagnosis weight loss was not associated with breast cancer mortality[50,52] or recurrence[52] in breast cancer survivors.

In contrast, Caan et al. reported that breast cancer survivors who lost at least 10% of their body weight post-diagnosis had an increased risk of breast cancer recurrence, although this relationship was no longer significant once women who recurred within a year of study entry were removed from the analysis thus this finding should be interpreted with caution.[53] It is clear, however, that weight management via regular physical activity should be emphasised as a key part of the strategy to prevent the recurrence and cancer death in survivors.[57]

Preventing weight gain and achieving and maintaining a healthy weight in oncology patients and cancer survivors may also be important in reducing the risk for co-morbid conditions associated with excess weight for which this population is at particularly high risk.[6–9]

Physical activity may also reduce or avert cancer cachexia,[58] which is a common yet often undiagnosed multi-factorial syndrome that is characterised by loss of skeletal muscles and subcutaneous fat, fatigue, anorexia, abnormal metabolism, and decreased muscle strength.[59,60] Resistance exercise training in particular may attenuate or prevent muscle wasting in those affected by cancer cachexia, although the evidence for this is still limited.[58,61–63]

Several mechanisms have been proposed to explain the links between physical activity and cancer outcomes. Physical activity may improve cancer prognosis through reducing the amount of adipose tissue, which in turn may reduce circulating levels of sex hormones, decrease the production of inflammatory cytokines in adipose tissue, increase adiponectin levels, improve insulin resistance, reduce hyperinsulinaemia, and enhance immune function.[64–66]

Physical activity has also been shown to directly reduce systemic inflammation, improve immune function, reduce sex hormones production, improve insulin sensitivity and glycaemic control, thus positively affecting cancer prognosis without changes in body composition.[64]

Objectives

Current evidence supports the benefits of physical activity for improving the risk factors associated with cancer prognosis. Therefore, for long-term cancer survivors, regular physical activity should be a priority to improve post-treatment quality of life and help reduce the risk of cancer recurrence, second primary cancers, chronic diseases, and cancer-related mortality and overall mortality. For interventions emphasising regular physical activity to be effective, however, the specific needs of this population must be considered.

The aim of this article is therefore to review the following:

- The preferences of cancer survivors regarding exercise counselling and participation in a physical activity programme;
- Adherence rates among cancer survivors to physical activity programmes; and
- Predictors of adherence to exercise training. Current availability of exercise counselling and programmes for oncology patients and cancer survivors is also briefly discussed.

Methods

Two electronic databases, Ovid MEDLINE(R) 1950 to Present with Daily Update and SCOPUS, were used to undertake literature searches for studies examining exercise preferences of adult cancer survivors, and physical activity programmes for adults at any point of the cancer trajectory. The searches were conducted between 1 July 2009 and 1 December 2009. The terms used to identify relevant studies are shown in Table 1 below.

Table 1. Summary of literature review strategy

Keyword	Articles retrieved	
	Ovid MEDLINE(R)	SCOPUS
Neoplasm	217,040	1,581,834
Survivors	9,115	12,617
Exercise	49,100	209,656
Physical activity	57,571	67,057
Adult	3,301,584	4,358,443
Neoplasm AND Survivors AND Exercise	60	289
Neoplasm AND Survivors AND Exercise AND Adult	22	178
Neoplasm AND Survivors AND Physical activity	17	229
Neoplasm AND Survivors AND Physical activity AND Adult	9	162
Neoplasm AND Survivors AND (Exercise OR Physical activity)	76	400
Neoplasm AND Survivors AND (Exercise OR Physical activity) AND Adult	31	265

The searches were subsequently limited to studies published in the English language. In addition, reference lists of the retrieved original and review articles were searched in order to identify any other relevant studies.

Exercise preferences of cancer survivors

Several cross-sectional studies have examined exercise preferences of cancer survivors across a range of cancer types.[67–79] Although cancer survivors were aware of the many benefits of being physically active,[73,76,79] their levels of physical activity decreased during cancer treatment and remained low following treatment.[69,70,74–79]

Indeed, although 34–42% of survivors reported to engage in at least 150 minutes of moderate-to-vigorous activity before diagnosis,[70,78] only 7–16% retained this level of activity during treatment,[69,78] and 19–41% post-treatment.[70,74,75,77,78] Furthermore, 31–54% of survivors reported to be sedentary and not to engage in any type of physical activities.[67,70,74,75,77]

In female breast cancer survivors, this low participation in physical activities was due to soreness after surgery, lack of motivation, cost, work responsibilities, family commitments, health-related barriers (e.g., side effects from medication, other illness), psychological barriers (e.g., self-consciousness due to surgery), and uncertainty about which types of exercise were safe, and when it was safe to return to physical activity.[73,76,79]

Despite this apparent lack of engagement in physical activity, most studies reported that an overwhelming majority of cancer survivors would have liked to receive exercise counselling or information about participating in an exercise programme at some stage during their cancer experience.[67,69–72,74,75,77,78]

Furthermore, many cancer survivors felt that they should receive exercise counselling from either an exercise specialist or from a health professional such as a nurse, physician, or an oncologist,[69,71,72,74-76,79] and that face-to-face counselling was the preferred mode of counselling.[69,71,72,74,75] Moreover, most survivors indicated that they were interested in an exercise programme,[67,71,72,74,75,77,78] were able to participate in such a programme,[70-72,74,75,77,78] and most would have preferred to initiate an exercise programme after treatment.[69-72,77,78]

A high proportion of cancer survivors, regardless of the type of cancer they had, indicated that they would have preferred to exercise in the morning,[69,71,72,74,75,77,78] on weekdays,[75] at moderate intensity,[69,71,72,74,75,78] unsupervised,[69,72,74,75,78] in the company of friends, family or other cancer survivors,[69-72,74,75,77-79] and at home.[69-72,74,75,77] Walking was undoubtedly the most preferred type of exercise both in the summer and winter.[69-72,74-78]

Many survivors were interested in physical activity programmes that were scheduled in terms of day and time,[69,71,78] and many would have liked to perform the same type of activity each time they exercised.[69,72,75] However, not all these findings were consistent across all studies. The majority of cancer survivors in some studies stated that they would have preferred to exercise alone,[72,74,75] engage in unscheduled physical activity,[72,75] and perform different activities each time they exercised.[72,78]

Consistent with the literature in the general population,[80,81] physical activity preferences appeared to be associated with a range of demographic, medical, and behavioural variables. Age,[69,70,72,74,77] level of education,[68,69,71,72,74,75,77,78] current exercise behaviour,[69-72,75,77,78] body mass index (weight in kg divided by height in m^2),[68,71,72,78] type of treatment,[68,70-72] and household income [71,72,75,77] were the most commonly identified variables to moderate exercise programme and information preferences.

Adherence rates and predictors of adherence to exercise programmes among oncology patients and cancer survivors

Programmes—A number of randomised controlled trials have evaluated physical activity programmes offered to adults at any point of the cancer trajectory.[23,44,82–86] The programmes varied considerably in duration, ranging from 2 weeks[44] to 1 year,[23,86] although most programmes lasted between 3 and 6 months.[23,44,82–87]

The studies offered supervised exercise training,[23,44,82,83] home-based training,[23,44,85,86] or programmes including both supervised and home-based components.[23,44,84] Many programmes recommended that participants engage in moderate-intensity physical activity that increased progressively to at least 5 days a week for at least 30 minutes per day (i.e., an equivalent of at least 7.5 MET-hours/week).[23,44,82–86]

Most programmes focused on aerobic exercise,[23,44,84–86] some trials offered resistance training,[23,44] and in some studies cancer survivors received a combination of aerobic and resistance training.[23,44,82,83] The intensity of the aerobic training showed considerable variability with study participants required to exercise from at least 40% to no more than 85% of estimated maximum heart rate.[23,44,82–84,86]

Adherence to physical activity programmes—Although the uptake of exercise programmes was varied with only 63% (range 12% to 100%) of the approached patients agreeing to undertake the exercise intervention,[87] adherence rates to exercise programmes were good, ranging from 68% to 98% for supervised programmes,[23,44,82,83] 70% to 94% for home-based programmes,[23,44,85,86] and 81% for programmes that included both supervised and home-based components.[84]

The required level of intensity of aerobic training did not appear to affect adherence rates.[23,82–84] In general, the programmes were effective in that the study participants increased their physical activity levels from 1.5-4.9 MET-hours/week at baseline[84–86] to 6.2–16.8 MET-hours/week at the end of an exercise programme.[84–86]

Predictors of adherence to physical activity programmes—Lower body mass index,[82,84] a higher degree of readiness to change physical activity behaviour,[84] better self-efficacy scores at baseline,[86] higher physical activity levels before commencing an exercise programme,[86] higher aerobic fitness,[83] more advanced disease stage,[83] lower depression,[83] younger age,[82] and more positive attitude towards exercise[82] have been identified as predictors of better adherence to physical activity programmes. These findings, however, have not been observed consistently across studies.[82–84,86]

Availability of physical activity counselling and programmes for oncology patients and cancer survivors

Despite the desire for the availability of exercise programming for cancer survivors, relatively little exercise programming is available specifically for this population, either within the cancer-care setting or elsewhere.[88]

The limited programmes that have been developed for cancer survivors include written information such as the guidebook ‘Exercise for Health: an Exercise Guide for Breast Cancer Survivors’ in Canada,[89] as well as individualised or small-group structured programmes that are often subsidised such as the ‘Cancer Survivors Program’ in Australia[90] or ‘Pink Pilates’ in New Zealand.[91]

Recent surveys of oncologists[92] and oncology nurses[88] found that although most oncologists had positive attitudes towards recommending exercise to patients with cancer during treatment, and most oncology nurses had positive attitudes towards providing exercise rehabilitation services for patients with cancer, only 42% of cancer survivors indicated that exercise was discussed at their treatment consultation with their oncologists, and only 28% of discussions were initiated by the oncologist.[93] Furthermore, an exercise programme was recommended to only 28% of patients,[92] and few cancer-care hospitals provided an exercise programme for their patients.[88]

This lack of exercise discussions and programme availability was due to a lack of awareness and familiarity with the exercise literature as well as scarce resources.[88] In fact, the majority of oncology nurses surveyed reported their familiarity with the exercise oncology literature as 'none' or 'low', and nearly a fifth of oncology nurses were unaware that a body of research in this area existed.[88]

Discussion

Exercise during or after cancer treatment has been shown to improve the risk factors associated with cancer prognosis. Despite the known benefits of physical activity, exercise rates decrease drastically during cancer treatment and remain low even after treatment is completed.[69,79,94-98] Furthermore, about half of cancer survivors are sedentary and do not engage in any type of physical activities.[67,70,74,75,77]

Nevertheless, most patients with cancer and cancer survivors have been shown to be motivated to receive exercise advice, have interest in participating in an exercise programme, and have also indicated that they are interested in an exercise program designed specifically for them. Although, it appears that cancer survivors are unlikely to continue or initiate an exercise programme without a structured intervention.[69,79,94-97]

Several exercise behaviour change interventions for adult patients with cancer and cancer survivors have been shown to effectively increase overall activity levels.[23,44,82-86] Many study participants, however, remained inactive or did not achieve the recommended activity levels.[84,85] Furthermore, nearly 40% of oncology patients or survivors who were approached to participate in an exercise programme declined to undertake it.[87]

The low uptake of exercise interventions and low number of participants achieving the recommended activity levels might have been due to the fact that many programmes did not reflect the most frequently reported programme preferences among cancer survivors. The same physical activity can not be used across survivors of different tumour types, because cancer diagnosis, side effects associated with different treatments, different experiences of cancer survivors, cancer-site-specific barriers, patient position on the cancer spectrum, or patient demographics affect exercise behaviours and attitudes towards physical activity and therefore need to be taken into consideration when developing physical activity programmes specifically for oncology patients and cancer survivors.[99]

Tailoring of exercise programmes to patient-specific and disease-specific needs is thus essential for optimising adherence, outcomes, and long-term behavioural changes in oncology patients and cancer survivors.[100]

Application of behaviour change theories may be particularly useful during this process. Behaviour change theories may be used for understanding physical activity in various cancer survivor groups,[75,76,82,83,101,102] identifying the main beliefs about physical activity in cancer survivors that are necessary for developing physical activity interventions for this population,[75,76,82,83,101,102] and may be used for actually developing effective physical activity interventions for specific groups of cancer survivors.[85,86]

In conclusion, research efforts are needed to develop physical activity programmes specifically for oncology patients and cancer survivors. Such programmes should take into account the programme preferences of the target population.

It is evident that a number of factors influence the exercise preferences of cancer survivors. Whether the needs of New Zealand oncology patients and cancer survivors in relation to continuation or initiation of physical activity are similar to those of North American or European survivors is uncertain and requires investigation. Furthermore, there is a need to examine whether tailoring of exercise programmes is associated with improved long-term adherence and exercise outcomes in New Zealand patients with cancer and cancer survivors.

Author information: Ewa A Szymlek-Gay, New Zealand Science and Technology Postdoctoral Fellow, Department of Clinical Sciences, Pediatrics, Umeå University, Umeå, Sweden; Rosalina Richards, Research Fellow, Cancer Society Social and Behavioural Research Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin; Richard Egan, Research Fellow/Senior Teaching Fellow, Cancer Society Social and Behavioural Research Unit, Department of Preventive and Social Medicine, University of Otago, Dunedin.

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Correspondence: Dr Ewa Szymlek-Gay, Department of Clinical Sciences, Pediatrics, Umeå University, SE-901 85 Umeå, Sweden. Fax: +46 (0)90 123728; email: ewa.szymlek-gay@pediatri.umu.se

References:

1. Ministry of Health. Cancer: New Registrations and Deaths 2005: Revised edition. Wellington: Ministry of Health; 2009.
2. Ministry of Health. Mortality and Demographic Data 2005. Wellington: Ministry of Health; 2009.
3. Ministry of Health. Cancer Patient Survival. Covering the Period 1994 to 2003. Wellington: Ministry of Health; 2006.
4. Dong C, Hemminki K. Second primary neoplasms in 633,964 cancer patients in Sweden, 1958-1996. Int J Cancer. 2001;93:155-61.
5. American Cancer Society. Cancer Facts & Figures 2009. Atlanta: American Cancer Society; 2009.
6. Fosså SD, Vassilopoulou-Sellin R, Dahl AA. Long term physical sequelae after adult-onset cancer. J Cancer Surviv. 2008;2:3-11.

7. Carver JR, Shapiro CL, Ng A, et al. American Society of Clinical Oncology clinical evidence review on the ongoing care of adult cancer survivors: cardiac and pulmonary late effects. J Clin Oncol. 2007;25:3991-4008.
8. Demark-Wahnefried W, Aziz NM, Rowland JH, Pinto BM. Riding the crest of the teachable moment: promoting long-term health after the diagnosis of cancer. J Clin Oncol. 2005;23:5814-30.
9. Kintzel PE, Chase SL, Schultz LM, O'Rourke TJ. Increased risk of metabolic syndrome, diabetes mellitus, and cardiovascular disease in men receiving androgen deprivation therapy for prostate cancer. Pharmacotherapy. 2008;28:1511-22.
10. Stava CJ, Jimenez C, Hu MI, Vassilopoulou-Sellin R. Skeletal sequelae of cancer and cancer treatment. J Cancer Surviv. 2009;3:75-88.
11. Aziz NM. Cancer survivorship research: state of knowledge, challenges and opportunities. Acta Oncol. 2007;46:417-32.
12. Courneya KS. Exercise in cancer survivors: an overview of research. Med Sci Sports Exerc. 2003;35:1846-52.
13. Brewster AM, Hortobagyi GN, Broglio KR, et al. Residual risk of breast cancer recurrence 5 years after adjuvant therapy. J Natl Cancer Inst. 2008;100:1179-83.
14. Berman JM, Cheung RJ, Weinberg DS. Surveillance after colorectal cancer resection. Lancet. 2000;355:395-9.
15. Early Breast Cancer Trialists' Collaborative Group. Effects of chemotherapy and hormonal therapy for early breast cancer on recurrence and 15-year survival: an overview of the randomised trials. Lancet. 2005;365:1687-717.
16. Reed VK, Krishnan S, Mansfield PF, et al. Incidence, natural history, and patterns of locoregional recurrence in gastric cancer patients treated with preoperative chemoradiotherapy. Int J Radiat Oncol Biol Phys. 2008;71:741-7.
17. Martini N, Bains MS, Burt ME, et al. Incidence of local recurrence and second primary tumors in resected stage I lung cancer. J Thorac Cardiovasc Surg. 1995;109:120-9.
18. Pound CR, Partin AW, Epstein JI, Walsh PC. Prostate-specific antigen after anatomic radical retropubic prostatectomy: patterns of recurrence and cancer control. Urol Clin North Am. 1997;24:395-406.
19. Aziz NM, Rowland JH. Trends and advances in cancer survivorship research: challenge and opportunity. Semin Radiat Oncol. 2003;13:248-66.
20. Ganz PA. Late effects of cancer and its treatment. Semin Oncol Nurs. 2001;17:241-8.
21. Travis LB, Rabkin CS, Brown LM, et al. Cancer survivorship - genetic susceptibility and second primary cancers: research strategies and recommendations. J Natl Cancer Inst. 2006;98:15-25.
22. Schmitz KH, Holtzman J, Courneya KS, et al. Controlled physical activity trials in cancer survivors: a systematic review and meta-analysis. Cancer Epidemiol Biomarkers Prev. 2005;14:1588-95.
23. McNeely ML, Campbell KL, Rowe BH, et al. Effects of exercise on breast cancer patients and survivors: a systematic review and meta-analysis. CMAJ. 2006;175:34-41.
24. Kim CJ, Kang DH, Park JW. A meta-analysis of aerobic exercise interventions for women with breast cancer. West J Nurs Res. 2009;31:437-61.
25. Jones LW, Demark-Wahnefried W. Diet, exercise, and complementary therapies after primary treatment for cancer. Lancet Oncol. 2006;7:1017-26.
26. Adamsen L, Quist M, Andersen C, et al. Effect of a multimodal high intensity exercise intervention in cancer patients undergoing chemotherapy: randomised controlled trial. BMJ. 2009;339:b3410.
27. Kirshbaum MN. A review of the benefits of whole body exercise during and after treatment for breast cancer. J Clin Nurs. 2007;16:104-21.
28. Conn VS, Hafdahl AR, Porock DC, et al. A meta-analysis of exercise interventions among people treated for cancer. Support Care Cancer. 2006;14:699-712.

29. Cramp F, Daniel J. Exercise for the management of cancer-related fatigue in adults. Cochrane Database Syst Rev. 2008:CD006145.
30. Thorsen L, Courneya KS, Stevinson C, Fosså SD. A systematic review of physical activity in prostate cancer survivors: outcomes, prevalence, and determinants. Support Care Cancer. 2008;16:987-97.
31. Bicego D, Brown K, Ruddick M, et al. Effects of exercise on quality of life in women living with breast cancer: a systematic review. Breast J. 2009;15:45-51.
32. Morey MC, Snyder DC, Sloane R, et al. Effects of home-based diet and exercise on functional outcomes among older, overweight long-term cancer survivors: RENEW: a randomized controlled trial. JAMA. 2009;301:1883-9.
33. Fairey AS, Courneya KS, Field CJ, Mackey JR. Physical exercise and immune system function in cancer survivors: a comprehensive review and future directions. Cancer. 2002;94:539-51.
34. Irwin ML, Alvarez-Reeves M, Cadmus L, et al. Exercise improves body fat, lean mass, and bone mass in breast cancer survivors. Obesity. 2009;17:1534-41.
35. Schwartz AL, Winters-Stone K, Gallucci B. Exercise effects on bone mineral density in women with breast cancer receiving adjuvant chemotherapy. Oncol Nurs Forum. 2007;34:627-33.
36. Holick CN, Newcomb PA, Trentham-Dietz A, et al. Physical activity and survival after diagnosis of invasive breast cancer. Cancer Epidemiol Biomarkers Prev. 2008;17:379-86.
37. Holmes MD, Chen WY, Feskanich D, et al. Physical activity and survival after breast cancer diagnosis. JAMA. 2005;293:2479-86.
38. Pierce JP, Stefanick ML, Flatt SW, et al. Greater survival after breast cancer in physically active women with high vegetable-fruit intake regardless of obesity. J Clin Oncol. 2007;25:2345-51.
39. Irwin ML, Smith AW, McTiernan A, et al. Influence of pre- and postdiagnosis physical activity on mortality in breast cancer survivors: the health, eating, activity, and lifestyle study. J Clin Oncol. 2008;26:3958-64.
40. Sternfeld B, Weltzien E, Quesenberry CP, Jr., et al. Physical activity and risk of recurrence and mortality in breast cancer survivors: findings from the LACE study. Cancer Epidemiol Biomarkers Prev. 2009;18:87-95.
41. Meyerhardt JA, Giovannucci EL, Holmes MD, et al. Physical activity and survival after colorectal cancer diagnosis. J Clin Oncol. 2006;24:3527-34.
42. Meyerhardt JA, Heseltine D, Niedzwiecki D, et al. Impact of physical activity on cancer recurrence and survival in patients with stage III colon cancer: findings from CALGB 89803. J Clin Oncol. 2006;24:3535-41.
43. World Cancer Research Fund / American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington, DC: AICR; 2007.
44. Pinto BM, Floyd A. Methodologic issues in exercise intervention research in oncology. Semin Oncol Nurs. 2007;23:297-304.
45. Stevens RJ, Roddam AW, Spencer EA, et al. Factors associated with incident and fatal pancreatic cancer in a cohort of middle-aged women. Int J Cancer. 2009;124:2400-5.
46. Ma J, Li H, Giovannucci E, et al. Prediagnostic body-mass index, plasma C-peptide concentration, and prostate cancer-specific mortality in men with prostate cancer: a long-term survival analysis. Lancet Oncol. 2008;9:1039-47.
47. Rock CL, Demark-Wahnefried W. Nutrition and survival after the diagnosis of breast cancer: a review of the evidence. J Clin Oncol. 2002;20:3302-16.
48. Reeves GK, Pirie K, Beral V, et al. Cancer incidence and mortality in relation to body mass index in the Million Women Study: cohort study. BMJ. 2007;335:1134-9.

49. Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. Adults. N Engl J Med. 2003;348:1625-38.
50. Nichols HB, Trentham-Dietz A, Egan KM, et al. Body mass index before and after breast cancer diagnosis: associations with all-cause, breast cancer, and cardiovascular disease mortality. Cancer Epidemiol Biomarkers Prev. 2009;18:1403-9.
51. Carmichael AR. Obesity and prognosis of breast cancer. Obes Rev. 2006;7:333-40.
52. Kroenke CH, Chen WY, Rosner B, Holmes MD. Weight, weight gain, and survival after breast cancer diagnosis. J Clin Oncol. 2005;23:1370-8.
53. Caan BJ, Kwan ML, Hartzell G, et al. Pre-diagnosis body mass index, post-diagnosis weight change, and prognosis among women with early stage breast cancer. Cancer Causes Control. 2008;19:1319-28.
54. Demark-Wahnefried W, Rimer BK, Winer EP. Weight gain in women diagnosed with breast cancer. J Am Diet Assoc. 1997;97:519-26.
55. Irwin ML, McTiernan A, Baumgartner RN, et al. Changes in body fat and weight after a breast cancer diagnosis: influence of demographic, prognostic, and lifestyle factors. J Clin Oncol. 2005;23:774-82.
56. Saquib N, Flatt SW, Natarajan L, et al. Weight gain and recovery of pre-cancer weight after breast cancer treatments: evidence from the women's healthy eating and living (WHEL) study. Breast Cancer Res Treat. 2007;105:177-86.
57. Doyle C, Kushi LH, Byers T, et al. Nutrition and physical activity during and after cancer treatment: an American Cancer Society guide for informed choices. CA Cancer J Clin. 2006;56:323-53.
58. Ardies CM. Exercise, cachexia, and cancer therapy: a molecular rationale. Nutr Cancer. 2002;42:143-57.
59. Fearon KC, Voss AC, Hustead DS. Definition of cancer cachexia: effect of weight loss, reduced food intake, and systemic inflammation on functional status and prognosis. Am J Clin Nutr. 2006;83:1345-50.
60. Evans WJ, Morley JE, Argiles J, et al. Cachexia: a new definition. Clin Nutr. 2008;27:793-9.
61. Zinna EM, Yarasheski KE. Exercise treatment to counteract protein wasting of chronic diseases. Curr Opin Clin Nutr Metab Care. 2003;6:87-93.
62. Fearon KCH. Cancer cachexia: developing multimodal therapy for a multidimensional problem. Eur J Cancer. 2008;44:1124-32.
63. Al-Majid S, Waters H. The biological mechanisms of cancer-related skeletal muscle wasting: the role of progressive resistance exercise. Biol Res Nurs. 2008;10:7-20.
64. McTiernan A. Mechanisms linking physical activity with cancer. Nat Rev Cancer. 2008;8:205-11.
65. Kellen E, Vansant G, Christiaens M-R, et al. Lifestyle changes and breast cancer prognosis: a review. Breast Cancer Res Treat. 2009;114:13-22.
66. Fair AM, Montgomery K. Energy balance, physical activity, and cancer risk. Methods Mol Biol. 2009;472:57-88.
67. Demark-Wahnefried W, Peterson B, McBride C, et al. Current health behaviors and readiness to pursue life-style changes among men and women diagnosed with early stage prostate and breast carcinomas. Cancer. 2000;88:674-84.
68. Hong S, Bardwell WA, Natarajan L, et al. Correlates of physical activity level in breast cancer survivors participating in the Women's Healthy Eating and Living (WHEL) Study. Breast Cancer Res Treat. 2007;101:225-32.
69. Jones LW, Courneya KS. Exercise counseling and programming preferences of cancer survivors. Cancer Pract. 2002;10:208-15.
70. Jones LW, Guill B, Keir ST, et al. Exercise interest and preferences among patients diagnosed with primary brain cancer. Support Care Cancer. 2007;15:47-55.

71. Karvinen KH, Courneya KS, Campbell KL, et al. Exercise preferences of endometrial cancer survivors: a population-based study. Cancer Nurs. 2006;29:259-65.
72. Karvinen KH, Courneya KS, Venner P, North S. Exercise programming and counseling preferences in bladder cancer survivors: a population-based study. J Cancer Surviv. 2007;1:27-34.
73. Larsson IL, Jönsson C, Olsson AC, et al. Women's experience of physical activity following breast cancer treatment. Scand J Caring Sci. 2008;22:422-9.
74. Rogers LQ, Courneya KS, Verhulst S, et al. Factors associated with exercise counseling and program preferences among breast cancer survivors. J Phys Act Health. 2008;5:688-705.
75. Rogers LQ, Markwell SJ, Verhulst S, et al. Rural breast cancer survivors: exercise preferences and their determinants. Psychooncology. 2009;18:412-21.
76. Rogers LQ, Matevey C, Hopkins-Price P, et al. Exploring social cognitive theory constructs for promoting exercise among breast cancer patients. Cancer Nurs. 2004;27:462-73.
77. Stevinson C, Capstick V, Schepansky A, et al. Physical activity preferences of ovarian cancer survivors. Psychooncology. 2009;18:422-8.
78. Vallance JK, Courneya KS, Jones LW, Reiman T. Exercise preferences among a population-based sample of non-Hodgkin's lymphoma survivors. Eur J Cancer Care (Engl). 2006;15:34-43.
79. Whitehead S, Lavelle K. Older breast cancer survivors' views and preferences for physical activity. Qual Health Res. 2009;19:894-906.
80. Booth ML, Bauman A, Owen N, Gore CJ. Physical activity preferences, preferred sources of assistance, and perceived barriers to increased activity among physically inactive Australians. Prev Med. 1997;26:131-7.
81. Cohen-Mansfield J, Marx MS, Biddison JR, Guralnik JM. Socio-environmental exercise preferences among older adults. Prev Med. 2004;38:804-11.
82. Courneya KS, Friedenreich CM, Reid RD, et al. Predictors of follow-up exercise behavior 6 months after a randomized trial of exercise training during breast cancer chemotherapy. Breast Cancer Res Treat. 2009;114:179-87.
83. Courneya KS, Segal RJ, Gelmon K, et al. Predictors of supervised exercise adherence during breast cancer chemotherapy. Med Sci Sports Exerc. 2008;40:1180-7.
84. Latka RN, Alvarez-Reeves M, Cadmus L, Irwin ML. Adherence to a randomized controlled trial of aerobic exercise in breast cancer survivors: the Yale exercise and survivorship study. J Cancer Surviv. 2009;3:148-57.
85. Matthews CE, Wilcox S, Hanby CL, et al. Evaluation of a 12-week home-based walking intervention for breast cancer survivors. Support Care Cancer. 2007;15:203-11.
86. Pinto BM, Rabin C, Dunsiger S. Home-based exercise among cancer survivors: adherence and its predictors. Psychooncology. 2009;18:369-76.
87. Maddocks M, Mockett S, Wilcock A. Is exercise an acceptable and practical therapy for people with or cured of cancer? A systematic review. Cancer Treat Rev. 2009;35:383-90.
88. Stevinson C, Fox KR. Role of exercise for cancer rehabilitation in UK hospitals: a survey of oncology nurses. Eur J Cancer Care (Engl). 2005;14:63-9.
89. Vallance JK, Courneya KS, Taylor LM, et al. Development and evaluation of a theory-based physical activity guidebook for breast cancer survivors. Health Educ Behav. 2008;35:174-89.
90. Edith Cowan University, Health and Wellness Institute. Our Programs - Overview. Cancer Survivors Program. [Internet]. 2011 [updated 2011 January 19; cited 2011 May 30]. Available from: http://www.variowellness.org/programs/index.php
91. Pink Pilates. [Internet] 2011 [cited 2011 May 30]. Available from: http://www.pinkpilates.org.nz/home.
92. Jones LW, Courneya KS, Peddle C, Mackey JR. Oncologists' opinions towards recommending exercise to patients with cancer: a Canadian national survey. Support Care Cancer. 2005;13:929-37.

93. Jones LW, Courneya KS. Exercise discussions during cancer treatment consultations. Cancer Pract. 2002;10:66-74.
94. Vallance JK, Courneya KS, Jones LW, Reiman T. Differences in quality of life between non-Hodgkin's lymphoma survivors meeting and not meeting public health exercise guidelines. Psychooncology. 2005;14:979-91.
95. Courneya KS, Friedenreich CM. Determinants of exercise during colorectal cancer treatment: an application of the theory of planned behavior. Oncol Nurs Forum. 1997;24:1715-23.
96. Courneya KS, Friedenreich CM. Relationship between exercise during treatment and current quality of life among survivors of breast cancer. J Psychosoc Oncol. 1997;15:35-57.
97. Irwin ML, Crumley D, McTiernan A, et al. Physical activity levels before and after a diagnosis of breast carcinoma: the Health, Eating, Activity, and Lifestyle (HEAL) study. Cancer. 2003;97:1746-57.
98. Blanchard CM, Denniston MM, Baker F, et al. Do adults change their lifestyle behaviors after a cancer diagnosis? Am J Health Behav. 2003;27:246-56.
99. Craike MJ, Livingston PM, Botti M. How much do we know about the determinants of physical activity among prostate cancer survivors? Directions for future research to inform interventions. Aust N Z J Public Health. 2009;33:193-4.
100. Stull VB, Snyder DC, Demark-Wahnefried W. Lifestyle interventions in cancer survivors: designing programs that meet the needs of this vulnerable and growing population. J Nutr. 2007;137:243S-8S.
101. Keogh J, Shepherd D, Krägeloh C, et al. Predictors of physical activity and quality of life in New Zealand prostate cancer survivors undergoing androgen-deprivation therapy. N Z Med J. 2010;123:20-9.
102. Rogers LQ, McAuley E, Courneya KS, Verhulst SJ. Correlates of physical activity self-efficacy among breast cancer survivors. Am J Health Behav. 2008;32:594-603.

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Summary of guidance for the management of early bowel cancer

Anita Fitzgerald, Frank Frizelle, Mark Jeffery, Adrian Balasingam, Jan Casey, Judith Collett, Teresa Lynch, Denise Robbins, Adrian Secker, Tane Taylor, Clint Teague, Iain Ward, Jessica Berentson-Shaw

Abstract

Colorectal cancer is an important public health problem and one of the most common cancers registered in New Zealand. In 2009 the New Zealand Guidelines Group were commissioned to produce and evidence-based summary of current New Zealand and international data to inform best practice in the management of people with early bowel cancer. A guideline development team was convened, representing a range of stakeholder groups who met to discuss and agree on the recommendations for a clinical practice guideline. This article summarises the guideline methods and reports the recommendations from the Management of Early Bowel Cancer guideline, published in 2011.

Background

Colorectal cancer is an important public health problem; nearly one million new cases of colorectal cancer are diagnosed worldwide each year resulting in half a million deaths.[1] Like most cancers, colorectal cancer is more common among older people. In 2006, colorectal cancer was the most common cancer registered and the second most common cause of death from cancer in New Zealand, accounting for 14.8% of all cancer registrations and 14.7% of all deaths from cancer. Both registration and mortality rates fell between 1996 and 2006; male and female registration rates dropped 10.6% and 15.0% respectively, while mortality rates fell 28.9% for males and 16.8% for females.[2]

Improving early detection and diagnosis of cancer and improving access to timely and appropriate treatment are identified as goals of the New Zealand Cancer Control Strategy Action Plan 2005–2010.[3] The development and implementation of guidelines support the achievement of these goals by contributing to improvements in national consistency and quality in cancer services. In 2009 the New Zealand Guidelines group were commissioned to produce an evidence-based summary of current New Zealand and overseas data to inform best practice in the management of people with early colorectal cancer. The guideline,[4] published in 2011, is relevant and useful to all secondary and tertiary care practitioners involved in the care of people with early colorectal cancer.

The guideline covers the period from preoperative assessments through to treatment and includes recommendations for follow-up and specifically addresses the management of people with invasive adenocarcinoma of the colon or rectum. The guideline provides recommendations for secondary and tertiary care providers and assumes the patient has already been referred because of suspicious bowel symptoms

or has undergone initial testing in primary care. Guidelines on the referral of patients with suspected cancer are available on the NZGG website (www.nzgg.org.nz).

It should be noted that the management of people with more advanced colorectal cancer (including metastatic disease) at diagnosis or later; people with high-risk familial colorectal cancer syndromes; squamous cell carcinomas, and colorectal cancer screening were beyond the scope of this guideline. Squamous cell carcinomas were also excluded.

Methods

The New Zealand Guidelines Group (NZGG) convened a Guideline Development Team (GDT) in 2009, nominated for their knowledge of colorectal cancer, by a range of stakeholder groups including the Ministry of Health, the Royal Australian and New Zealand College of Radiologists, the New Zealand Society of Gastroenterology, The Royal College of Pathologists of Australasia, Cancer Voices consumer organisation, academic institutions, and District Health Boards. Maori and consumer representatives were actively involved in the group. The GDT considered the evidence for a number of clinical questions and formed recommendations based on the evidence provided by NZGG and their own knowledge of colorectal cancer.

A seeding guideline produced by the National Health and Medical Research Council in 2005[5] was adapted to the New Zealand context; where recommendations were considered current, these were adopted outright into the New Zealand guideline. A new set of clinical questions were developed where the GDT agreed that NHMRC recommendations were known to be out of date, had a controversial evidence base, or where practice was thought to differ between New Zealand and Australia. Recommendations arising from the new clinical questions were based on existing guidelines and systematic reviews with searches of the literature from 2004 onwards. Full methodological details can be found in the full text document available on the NZGG website (www.nzgg.org.nz).

Recommendations

The recommendations made in the guideline are graded according to the institution in which they were developed. Recommendations adopted from the NHMRC guideline retain the evidence level (I-IV), and strength of recommendation (not recommended – strongly recommended) that was applied at the time of writing in 2005 in Australia. The recommendations developed in New Zealand from 2009-2010 are graded according to the NZGG classification from A grade (the recommendation is supported by good evidence) to Good practice point (where no evidence is available, best practice recommendations are made based on the experience of the Guideline Development Team or feedback from consultation within New Zealand).

Ethnic disparities and cultural issues

- A patient navigator, care coordinator or support person should be involved to support patients and families/whānau following a diagnosis of colorectal cancer, and to assist in guiding them along the patient care pathway. (Good practice point)
- Service providers should ensure that information about colorectal cancer care and support services meets the needs of different ethnic groups and their families/whānau. (Good practice point)
- Māori-specific and Pacific-specific cancer services or service components should be provided where a need is identified. (Good practice point)

- Health systems planners and service providers should improve access to services for ethnic groups, for example, by developing and supporting outreach and community-based clinics. (Good practice point)
- Health systems planners should support and develop Māori and Pacific participation in the colorectal cancer care workforce at all levels. (Good practice point)
- Service providers should collect and report accurate, high-quality ethnicity data at all stages of the patient pathway to ensure that the effectiveness of health services in reducing disparities can be monitored. (Good practice point)
- Service providers should monitor practice, including review of patient experiences, to foster culturally competent, patient-centred care. (Good practice point)

Multidisciplinary Teams

- All people with colon cancer should be discussed at a Tumour Board meeting (B)
- All people with rectal cancer should be discussed at a Tumour Board Meeting (B)
- Every health practitioner involved in colorectal cancer care should actively participate in a multidisciplinary team. (Good practice point)
- The Tumour Board and multidisciplinary team involved in colorectal cancer care should provide culturally appropriate and coordinated care, advice and support. (Good practice point)
- The outcomes of Tumour Board and multidisciplinary team meetings should be communicated to the person with colorectal cancer and their general practitioner, and should be clearly documented in the medical records. (Good practice point)

Supportive and Rehabilitative Care

- Psychosocial care is important. Psychological interventions should be a component of care as they can improve the quality of life for patients with cancer (NHMRC evidence level I – strongly recommended)
- Supportive and rehabilitative care should be available to all people with colorectal cancer. (Good practice point)

Communication and Information Provision

- During consultation, practitioners should make available to people with colorectal cancer the level and amount of information that will be most effective in enabling them to understand their condition and treatment options. (Good practice point)
- People with colorectal cancer should be acknowledged as key partners in the decision-making about their cancer management. (Good practice point)

- Practitioners should provide people with colorectal cancer information about their diagnosis, treatment options (including risks and benefits) and support services. (Good practice point)
- Practitioners should give people with colorectal cancer information about managing bowel function, particularly diet, following surgery. (Good practice point)
- Practitioners should encourage people with colorectal cancer to take notes or record a consultation and have a support person present. (Good practice point)
- Practitioners should maintain a patient hand-held record, where available. (Good practice point)
- Service providers and practitioners should ensure that high-quality evidence-based information resources in a variety of formats and languages are available for people with colorectal cancer. (Good practice point)

Pre-operative assessments

- Preoperative assessment for colon cancer should include clinical examination, complete blood count, liver and renal function tests, carcinoembryonic antigen (CEA), chest x-ray and contrast enhanced computed tomography (CT) of the abdomen/pelvis/liver. (C)
- Preoperative assessment should include colonoscopy of the entire large bowel. Where complete examination is not possible, imaging of the proximal colon with CT colonography (or with barium enema if CT colonography is not available) is recommended. (C)
- If proximal parts of the colon are not directly visualised preoperatively, postoperative repeat colonoscopy should be undertaken within 12 months. (C)
- In selected cases, preoperative microsatellite instability (MSI)/immunohistochemistry may be helpful in guiding surgical management. (Good practice point)
- PET-CT scanning is not recommended as part of routine preoperative assessment of non-metastatic colon cancer. (C)
- Preoperative assessment for rectal cancer should include clinical examination, complete blood count, liver and renal function tests, carcinoembryonic antigen (CEA), chest x-ray and contrast-enhanced CT of the abdomen/pelvis/liver. (C)
- Preoperative assessments for rectal cancer should include MRI for identifying circumferential resection margin (CRM) involvement and local staging. (B)
- Preoperative assessment of possible T1 rectal cancers may include endorectal ultrasound (EUS) for local staging, as an alternative to MRI of the pelvis. (B)
- Endorectal ultrasound should not be used as the sole assessment to predict CRM involvement in people with rectal cancer. (B)

Management of Epithelial Polyps

- Adenomas with focal malignancy may be managed safely by endoscopic polypectomy provided strict criteria for patient selection and histopathological assessment are adhered to. In particular, adenomas with focal malignancy should be well or moderately differentiated and excision should be complete. (NHMRC evidence level III-2 – Recommended)

Preparation for surgery

- All patients who have a reasonable chance of a postoperative stoma should be prepared for this possibility. This includes a visit, where possible, by the stomal therapy nurse. (NHMRC evidence level III-2, Recommended)
- Bowel preparation is current standard practice before elective colorectal operations. However, recent randomised controlled trials have not demonstrated any conclusive benefit from this procedure. Accordingly, the previous guideline has been revised as follows:
    - Mechanical bowel preparation is not indicated in elective colorectal operations unless there are anticipated problems with faecal loading that might create technical difficulties with the procedure, for example, laparoscopic surgery, low rectal cancers. (NHMRC evidence level I, Not recommended)
- All patients undergoing surgery for colorectal cancer should receive prophylaxis for thromboembolic disease. (NHMRC evidence level I, Strongly recommended)
- Unfractionated heparin, low molecular weight heparin, and intermittent calf compression are effective in reducing the incidence of thromboembolism. (NHMRC evidence level II, Strongly recommended)
- Low molecular weight heparin has not been shown to be superior to low-dose heparin in colorectal surgical patients. (NHMRC evidence level II, Strongly recommended)
- All patients undergoing colorectal cancer surgery require prophylactic antibiotics. NHMRC evidence level II, Recommended)
- A single preoperative dose of intravenous cephalosporin and metronidazole, or gentamicin and metronidazole, is an effective regimen. (NHMRC evidence level I, Strongly recommended)
- Perioperative normothermia should be maintained. (NHMRC evidence level II, Recommended)

Elective surgery for colon cancer

- High ligation of the lymphovascular pedicle does not confer any oncological benefit. Resection where feasible should extend to the origin of segmental vessels. (NHMRC evidence level III-3, Equivocal)

- The no-touch isolation technique has no oncological benefit. (NHMRC evidence level II, Recommended)
- Segmental resection is equivalent to extended resection in outcome. (NHMRC evidence level II, Equivocal)
- Omental wrapping of anastomosis has no benefit. (NHMRC evidence level III-2, Strongly not recommended)
- In experienced hands, laparoscopic surgery for colon cancer has equivalent outcomes to conventional surgery. (NHMRC evidence level I, Recommended)
- Stapled functional end-to-end ileocolic anastomosis is recommended. (A)
- Elective surgery for colon cancer should be performed by a surgeon with specific training and experience in colorectal surgery, and with sufficient caseload to maintain surgical skills. (B)

Elective surgery for rectal cancer

- Local excision of T1 rectal cancer may be used in selected cancer patients according to the following guidelines:
    - mobile tumour <3 cm
    - T1 on endorectal ultrasound
    - not poorly differentiated on histology (biopsy). (NHMRC evidence level III-3, Equivocal)
- A distal distance of 2 cm (fresh) is recommended in most instances, or 1 cm fixed. (NHMRC evidence level III-2, Recommended)
- Sphincter-saving operations are preferred to abdominoperineal resection except in the presence of:
    - tumours such that adequate distal clearance (>2 cm) cannot be achieved
    - the sphincter mechanism is not adequate for continence
    - access to the pelvis makes restoration technically impossible (rare). (NHMRC evidence level III-3, Equivocal)
- For mid-to-low rectal tumours, the principles of extra fascial dissection and total mesorectal excision (TME) are recommended. (NHMRC evidence level III-2, Recommended)
- Where technically feasible, a colonic reservoir is recommended for anastomosis within 2 cm from ano-rectal junction. ((NHMRC evidence level II, Strongly recommended)
- Routine drainage should only be considered for rectal cancers. (NHMRC evidence level II, Equivocal)
- Elective surgery for rectal cancer should be carried out by a surgeon who has undergone a period of specialist exposure to this form of surgery during

surgical training and who has maintained satisfactory experience in the surgical management of rectal cancer. (B)

Emergency surgery

- Primary anastomosis should be considered as a colectomy, with an ileocolic or ileorectal anastomosis. (NHMRC evidence level III-2, Equivocal)
- Primary anastomosis could be considered for left-sided obstruction and may need to be preceded by on table colonic lavage. (NHMRC evidence level III-2, Equivocal)
- Primary resection of obstructing carcinoma is recommended unless the patient is moribund. (B)
- Colonic stenting for palliation of left-sided bowel obstruction in people with colorectal cancer is recommended, if endoscopic expertise can be readily accessed. (B)
- Colonic stenting as a bridge to surgery for left-sided bowel obstruction in people with colorectal cancer may be considered for an individual, if endoscopic expertise can be readily accessed. (C)
- People with colorectal cancer who have bowel obstruction and are being considered for colonic stenting should be invited to participate in randomised controlled trials, where these are available. (Good practice point)

Adjuvant therapy for colon cancer

- People with resected colon cancer should be considered for adjuvant therapy. (Good practice point)
- People with resected node positive colon cancer (Stage III) should be offered postoperative chemotherapy unless there is a particular contraindication, such as significant comorbidity or poor performance status. (A)
- People with resected node negative colon cancer (Stage II) with poor prognostic features may be offered postoperative chemotherapy. Discussion of risks and benefits of treatment should include the potential but uncertain benefits of treatment and the potential side effects. (C)
- For people with colon cancer who are to receive single agent postoperative chemotherapy, either capecitabine or bolus fluorouracil plus leucovorin are appropriate regimens. (B)
- For people with resected node positive colon cancer (Stage III) who are to receive postoperative chemotherapy, combination chemotherapy with oxaliplatin and a fluoropyrimidine is recommended. (A)
- Irinotecan should not be given as postoperative adjuvant chemotherapy for people with Stages I, II and III colon cancer. (A) Note: irinotecan is currently licensed in New Zealand for metastatic colorectal cancer only

Adjuvant therapy for rectal cancer

- Preoperative or postoperative adjuvant therapy should be considered by a multidisciplinary team for all people with rectal cancer. (Good practice point)
- Preoperative radiotherapy may lower the incidence of late morbidity compared to postoperative radiotherapy. (C)
- For people with rectal cancer who are at risk of local recurrence, either preoperative short-course radiotherapy or preoperative long-course chemoradiation is recommended. (B) Note: Short-course radiotherapy – 25 Gy in 5 fractions; long-course radiotherapy – 45–50.4 Gy in 25–28 fractions.
- Preoperative long-course chemoradiation is recommended for people with rectal cancer who have a low rectal cancer or a threatened circumferential resection margin. (B) Note: Long-course radiotherapy – 45–50.4 Gy in 25–28 fractions.
- Where people are receiving long-course radiotherapy (preoperative or postoperative), concurrent chemotherapy should be considered. (A)

Follow up after curative resection

- All people who have undergone colorectal cancer resection should be followed up intensively. (Good practice point)
- All people who have undergone colorectal cancer resection and develop relevant symptoms should undergo clinical assessment. (Good practice point)
- For people with colon cancer at high risk of recurrence (Stages IIb and III), clinical assessment is recommended at least every six months for the first three years after initial surgery and then annually for a further two years or when symptoms occur. (B)
- For people with colon cancer at lower risk of recurrence (Stages I and IIa) or for people with comorbidities restricting future surgery, clinical assessment is recommended when symptoms occur or by annual review for five years after initial surgery. (B)
- All people with colorectal cancer should have a colonoscopy before surgery or within 12 months following initial surgery. (B)
- For people with colon cancer at lower risk of recurrence (Stages I and IIa), follow-up colonoscopy every three to five years is recommended. (B)
- For people with rectal cancer, digital rectal examination (DRE), proctoscopy or sigmoidoscopy should be undertaken at three months, six months, one year and two years after initial surgery. Thereafter colonoscopy should be undertaken at three- to five-yearly intervals. (B)
- Follow-up should include physical examination and CEA. (B)
- All people with colorectal cancer Stages I to III should have liver imaging between years 1 and 3. (B)

- The use of faecal occult blood testing as part of colorectal cancer follow-up is not recommended. (B)
- Follow-up should be under the direction of the multidisciplinary team and may involve follow-up in primary care. (Good practice point)
- People with colorectal cancer should be given written information outlining planned follow-up (e.g. discharge report) at discharge from treatment, including what they should expect regarding the components and the timing of follow-up assessments. (Good practice point)

Synoptic reporting

- Pathology reporting of all colon and rectal cancer specimens should include structured (synoptic) reporting. (C)
- Reporting of investigations and procedures (colonoscopy, radiology, operation notes, oncology treatment records) relating to colorectal cancer in a synoptic format is recommended. (Good practice point)
- TNM staging and the data required to stage the patient should all be recorded to allow national and international comparisons. (NHMRC evidence level III-3, Equivocal)

Competing interests: None.

Note: The full text of the guideline is available on the New Zealand Guidelines Group website from July 1st 2011 www.nzgg.org.nz

Author information: Anita Fitzgerald, Assistant Research Manager, New Zealand Guidelines Group; Frank Frizelle, Professor of Colorectal Surgery, Academic Head of Department of Surgery, Christchurch Hospital, and Editor in Chief of the New Zealand Medical Journal; Mark Jeffery, Medical Oncologist, Oncology Service, Christchurch Hospital; Adrian Balasingam, Radiologist, Christchurch Radiology Group, Christchurch Hospital; Jan Casey, Consumer Representative; Judith Collett, Gastroenterologist, Clinical Advisor NZ Familial GI Cancer Registry, Visiting Gastroenterologist, Timaru, Greymouth and Southland Hospitals; Teresa Lynch, Project Manager, Surveillance Database Gastroenterology Day Unit, Gastrointestinal Investigative Unit, Christchurch Hospital; Denise Robbins, Consumer Representative; Adrian Secker, General Surgeon, Nelson Hospital; Tane Taylor, Consultant Taylor's Medical; Clint Teague, Pathologist, Aotea Pathology, Wellington; Iain Ward, Clinical Director, Radiation Oncology, Christchurch Hospital; Jessica Berentson-Shaw, Research Manager, New Zealand Guidelines Group

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Correspondence: Anita Fitzgerald, Assistant Research Manager, New Zealand Guidelines Group, Level 10, 40 Mercer Street, Wellington. afitzgerald@nzgg.org.nz

References:

1. Boyle P, Leon ME. Epidemiology of colorectal cancer. Br Med Bull. 2002;64:1–25.

2. Ministry of Health. Cancer: new registrations and deaths 2006. Wellington, New Zealand: Ministry of Health; 2010.
3. Cancer Control Taskforce. The New Zealand cancer control strategy: action plan 2005–2010. Wellington, New Zealand: Ministry of Health; 2005.
4. www.nzgg.org.nz
5. Australian Cancer Network Colorectal Cancer Guidelines Revision Committee. Guidelines for the prevention, early detection and management of colorectal cancer. Sydney, Australia: The Cancer Council Australia and Australian Cancer Network; 2005. Available from: www.nhmrc.gov.au/_files_nhmrc/file/publications/synopses/cp106/cp106.pdf

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Global polio eradication: progress, but determination and vigilance still needed

Stephen T Chambers, Nigel Dickson

Abstract

Aim To review recent events in the international effort to eradicate polio from earth and inform readers of recent changes in strategy that have implications for New Zealand

Method Review of recent literature and publications from World Health Organization (WHO).

Results After initial success in eradicating polio from Europe, the Western Pacific and the Americas, transmission of wild type polio in Nigeria, India, Pakistan and Afghanistan was never interrupted. These foci were the source of importations of polio into more than 40 countries, including Australia, that were previously polio-free. This has lead to a change in strategy by WHO and there are promising indications that polio is coming under control in these places.

Conclusion Importation of wild-type polio into New Zealand are still possible and it essential to maintain awareness amongst clinicians that this can happen, high immunisation levels, an effective National Poliomyelitis Response Plan for New Zealand, and ongoing surveillance of acute flaccid paralysis.

A case of wild type polio in Australia in June 2007 was a sharp lesson that polio is still a threat and not a mere text book curiosity from bygone days even in New Zealand.

A 22-year-old Pakistani student, who had been fully vaccinated with oral polio vaccine (OPV) as a child, had been studying in Melbourne and returned home for a holiday.[1] He toured some remote regions and developed fever, sweating, nausea, vomiting, pain in his low back and legs, and weakness of his left leg. After several days, all symptoms except the pain resolved and he returned to Melbourne where the weakness returned.

He was admitted to hospital where an MRI scan of his spine showed activity in the anterior horn cells suggestive of poliomyelitis which was confirmed when wild type polio virus was identified in his stools. The genotypic was similar to strains found in Pakistan and Afghanistan. He made a full recovery and no secondary cases were identified following an intensive public health response that included extensive testing and vaccination.[2]

This case reminds clinicians and public health physicians of the possibility of polio being seen in New Zealand, and is an opportune time to review the progress of the World Health Organization's (WHO's) towards global eradication of this disease.

The declaration of the WHO Western Pacific Region—including New Zealand—to be polio-free in October 2000 was a cause for celebration although it passed almost

unmarked here.[3] The Region includes recently endemic countries such as China and several others in Indochina where intensive efforts had been made to stop the circulation of wild type polio through immunisation of children. A key component had been national and subnational immunisation days which delivered oral polio vaccine(OPV) to a very large number of children over a few weeks. Some of these were on a massive scale and constituted one of the greatest public health interventions in history.[4]

Important difficulties that were overcome included maintaining the cold chain in tropical climates to preserve vaccine efficacy, and collection and culture of stools for isolation of virus in cases of acute flaccid paralysis to determine whether wild type polio virus transmission had been truly interrupted. As Europe and the Americas were also polio-free in 2000, there was reason to think the virus could be eliminated worldwide using the same strategy. Over the following decade however progress toward eradication stalled. Polio transmission was never stopped in Nigeria, parts of India, Afghanistan and Pakistan, and importations were detected in at least 40 previously polio-free countries.[5] See Table 1.

In 2010, there was a large outbreak in Tajikistan with nearly 500 cases.[6] This was brought under control through mass vaccination campaigns there and in surrounding countries which were also effected. This outbreak was not unexpected as Tajikistan had low vaccination rates, poor surveillance—hence unable to detect an outbreak early—and was close to endemic areas.

The WHO has recently reviewed this delayed progress, identified weaknesses with its previous approach, and embarked on a new strategic called the Global Polio Eradication Initiative Strategic Plan 2010–2012.[7] They is now more emphasis on local planning and engaging community leaders to overcome suspicion of the vaccination to maximise coverage; recognition that polio transmission can continue in regions with relatively low population densities; and, now that polio virus type 2 has been eliminated, utilisation of vaccines with improved immunogenicity against polio virus type 1 and 3. The latter are expected to greatly aid control in the residual reservoirs areas that have proved particularly resistant to previous efforts where vaccine can have relatively low immunogenicity. This approach has seen marked reductions in the number of cases in all the four countries that have never previously been free.

While it is possible that wild type polio virus all polio transmission could be eliminated over the next few years, important obstacles remain such as armed conflict, natural disasters, limited funding and other overwhelming priorities. Fortunately new leadership at WHO has put global eradication back on the agenda and ‘tranquility days’ to allow vaccination teams into areas of conflict have been negotiated. Although substantial funding has been given by many donor nations, and non-governmental organisations in particular the Bill and Melinda Gates Foundation and Rotary International, a shortfall of nearly a billion dollars still exists.[9]

Assuming circulating wild polio virus can be eliminated the strategy for final elimination of live polio and poliomyelitis from the earth will need careful planning. Currently live oral OPV is used in most countries and thought to cause 250-500 cases of vaccine associated paralytic polio (VAPP) annually, that has prompted several countries including New Zealand and Australia to change to injectable vaccine.

Table 1. Cases of poliomyelitis in selected countries documented by WHO[5] (as of Wednesday 15 June 2011)

Wild poliovirus (WPV) cases

Total cases	Year-to-date 2011	Year-to-date 2010	Total in 2010*
Globally	205	349	1349
• in endemic countries:	66	58	232
• in non-endemic countries:	139	291	1117

Countries	Year-to-date 2011			Year-to-date 2010			Total in 2010*	Date of most recent case
	WPV1	WPV3	Total	WPV1	WPV3	Total		
Pakistan	49		49	12	12	24	144	23-May-11
Afghanistan	4		4	2	8	10	25	09-May-11
Nigeria	8	4	12	1	2	3	21	09-May-11
India	1		1	4	17	21	42	13-Jan-11
Chad	65	3	68		14	14	26	14-May-11
DRCongo	55		55				100	09-May-11
Angola	4		4	8		8	33	27-Mar-11
Guinea		1	1					14-May-11
Côte d'Ivoire		4	4					12-May-11
Mali		3	3	2		2	4	20-Mar-11
Burkina Faso		1	1					26-Feb-11
Congo	1		1				441	22-Jan-11
Niger		1	1		2	2	2	19-Jan-11
Gabon	1		1					15-Jan-11
Uganda							4	15-Nov-10
Russian Federation							14	25-Sep-10
Liberia				1		1	2	08-Sep-10
Nepal				1		1	8	30-Aug-10
Kazakhstan							1	12-Aug-10
Tajikistan				239		239	457	04-Jul-10
Turkmenistan								28-Jun-10
Senegal				18				30-Apr-10
Sierra Leone				1				28-Feb-10
Total	**188**	**17**	**205**	**294**	**55**	**349**	**1349**	
Total in endemic countries	**62**	**4**	**66**	**19**	**39**	**58**	**232**	
Total outbreak	**126**	**13**	**139**	**275**	**16**	**291**	**1117**	

Data in WHO as of 15 Jun 2010 for 2010 data and 14 Jun 2011 for 2011 data.

*The 2010 total for Congo includes cases with inadequate specimens that have been exceptionally classified as confirmed polio based on their association with the WPV1 outbreak.

OPV can also spread from person to person where there is poor sanitation which occasionally allows the regeneration of neurovirulent strains; over the last 10 years there have been at least 15 such outbreaks. In addition there are rare immune suppressed individuals who excrete live vaccine derived poliovirus in whom the virus will persist lifelong. These factors necessitate the development of a cheap injectable vaccine that can be deployed worldwide over long periods to eradicate persisting vaccine derived strains.

While the signs are encouraging it is vital not to let the guard down too soon, and in New Zealand ongoing clinical and public health vigilance must be maintained in five specific areas:

- Clinicians must consider the possibility of poliomyelitis especially in people from parts of the world where wild virus is still circulating, and even among people who have been fully vaccinated. To investigate a suspected case clinicians should liaise closely with the local microbiologist and public health service.
- Public health authorities need to be prepared to implement the National Poliomyelitis Response Plan for New Zealand that has been developed by the Ministry of Health in the event of the importation of a case of wild polio.[8]
- High rates of immunisation coverage must be maintained to ensure that if wild polio is introduced herd immunity is high enough to preclude its spread. Historically childhood immunization rates have been poor; however recent initiatives such as the establishment of a national vaccine register have helped improve coverage. By august 2010 coverage for 90% for children ager 12-18 months and 93% of three year old children had been achieved (M. Bonné; personal communication).
- Epidemiological surveillance for acute flaccid paralysis, and the appropriate testing of faeces from these cases such as has been undertaken by the New Zealand Paediatric Surveillance Unit and ESR among paediatricians since 1997, needs to continue to confirm that there truly are no cases of polio among children here.[11]

Competing interests: None.

Author information: Stephen T Chambers, Department of Pathology, University of Otago, Christchurch—Infectious Diseases Physician Christchurch Hospital—and Chair of National Certification Committee for the Eradication of Poliomyelitis; Nigel Dickson, Co-Director New Zealand Paediatric Surveillance Unit, Department of Women's and Children's Health and Associate Professor of Epidemiology, Dunedin School of Medicine, University of Otago, Dunedin

Correspondence: Dr ST Chambers, Department of Infectious Diseases, Christchurch Hospital, PO Box 4345, Christchurch, New Zealand. Fax: +64 (0)3 3640952; Steve.Chambers@cdhb.govt.nz

References:

1. Stewardson AJ, Roberts JA, Beckett CL, et al. Imported case of poliomyelitis, Melbourne, Australia, 2007. Emerg Infect Dis. 2009;15:63-5.
2. Carnie JA, Lester R, Moran R, et al. Public health response to imported case of poliomyelitis, Australia, 2007. Emerg Infect Dis. 2009;15:1733-7.
3. Centers for Disease Control and Prevention (CDC). Certification of poliomyelitis eradication--Western Pacific Region, October 2000. MMWR Morb Mortal Wkly Rep. 2001;50:1-3.
4. Zhang Y, Yan D, Zhu S, et al. Type 2 vaccine-derived poliovirus from patients with acute flaccid paralysis in china: current immunization strategy effectively prevented its sustained transmission. J Infect Dis. 2010;202:1780-8.
5. WHO weekly epidemiological record http://www.who.int/wer/2010/
6. Tajikistan polio outbreak and regional response. WHO Epidemiological Brief. 23 June 2010. Geneva: WHO.
7. Global Polio Eradication Initiative Strategic Plan 2010–2012. http://www.polioeradication.org/Resourcelibrary/Strategyandwork/Strategicplan.aspx
8. Pallansch MA, Sandhu HS. The eradication of polio-progress and challenges. N Engl J Med. 2006;355:508-11
9. Tebbens RJ, Pallansch MA, Cochi SL, et al. Economic analysis of the global polio eradication initiative. Vaccine. 2010 Oct 25. [Epub ahead of print]
10. Ministry of Health. 2009. National Poliomyelitis Response Plan for New Zealand. Wellington: Ministry of Health
11. New Zealand Paediatric Surveillance Unit, Annual Report 2009. http://dnmeds.otago.ac.nz/departments/womens/paediatrics/research/nzpsu/index.html

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Quetiapine-associated cardiomyopathy

Sean Coffey, Michael Williams

Congestive heart failure due to dilated cardiomyopathy is an unusual cause of shortness of breath in young patients. Causes of dilated cardiomyopathy include myocarditis, familial cardiomyopathies, coronary artery disease, alcohol, pregnancy, and drugs, although it is most common not to find a specific aetiology.

Psychoactive medications, in particular clozapine, have been linked with the development of cardiomyopathy, possibly through the development of a preceding Type 1 hypersensitivity reaction leading to eosinophilic myocarditis.[1]

We report here two cases of cardiomyopathy associated with quetiapine. The study was approved by the Lower South Ethics Committee.

Case reports

Patient 1—The first patient, a 33-year-old male diagnosed with catatonic schizophrenia, had been treated with quetiapine for 4 years. He had been stabilised on a dose of 1000 mg daily for 2 years. Eight weeks prior to presentation he had an influenza-like illness. He presented with a 2-week history of shortness of breath, ankle swelling and orthopnoea. On examination he was tachycardic with a heart rate of 126 beats per minute, jugular venous pressure 10 cm above the sternal angle, crepitations on lung auscultation, and hepatomegaly.

An echocardiogram showed globally severely impaired left and right ventricular systolic function, with a left ventricular (LV) end diastolic dimension of 6.5 cm and an ejection fraction of 14%. Coronary angiography showed no obstructive epicardial coronary artery disease. A previous echocardiogram performed 10 years previously had shown normal ventricular function. Despite extensive investigation for infectious and metabolic causes, no underlying cause for his cardiomyopathy was able to be determined.

A diagnosis of dilated cardiomyopathy due to quetiapine was made, and his quetiapine withdrawn. He symptomatically improved after treatment with cilazapril, carvedilol, furosemide, digoxin and spironolactone, and was discharged 9 days after admission. However his mental status deteriorated rapidly, leading to admission 10 days later to an acute psychiatric unit.

Ziprasidone therapy was started but had to be withdrawn after QT interval prolongation. Amisulpride therapy was then commenced. Four days later, 36 days after his initial presentation, he was found dead in the morning, with no external cause of death found. The cause of death was thought to be a malignant arrhythmia related to his underlying cardiomyopathy.

Patient 2—The second patient, a 28-year-old woman presented with rapid onset of shortness of breath starting 2 days previously. She had been taking quetiapine 800 mg daily and venlafaxine 150 mg daily for 2 years as treatment for depression and possible borderline personality disorder. On examination she was obese and tachycardic but without clinical signs of congestive heart failure. She was initially investigated for a pulmonary embolus, and was anticoagulated after a CT pulmonary angiogram was possibly consistent with pulmonary emboli.

However her dyspnoea continued to progress and a subsequent echocardiogram showed globally severely impaired LV systolic function, with a LV end diastolic dimension of 6.5 cm and an ejection fraction of 15%. Pharmacological stress myocardial perfusion scanning showed no inducible ischaemia and the results were interpreted as being consistent with a non-ischaemic dilated cardiomyopathy.

Detailed testing failed to show any infectious nor metabolic cause for her cardiomyopathy. A diagnosis of quetiapine induced cardiomyopathy was made, and the quetiapine was stopped. She responded well to initial treatment with quinapril, carvedilol, furosemide and spironolactone, with subsequent echocardiograms showing an improvement in ejection fraction to 35%.

Discussion

The first case described highlights the difficulty in managing the mental health of patients with severe cardiomyopathy where there are concerns about chronic therapy being related to presentation with heart failure. Withdrawal of the psychoactive medication led to a rapid decline in the patient's mental status.

A common alternative for patients who have failed therapy with quetiapine is clozapine, which is contraindicated in the setting of a pre-existing cardiomyopathy. Many alternative agents are associated with QT interval prolongation, as we found in this case, complicating suitable therapeutic options.

These two case reports add to the small worldwide literature on quetiapine-induced heart disease. There has been one case report previously describing quetiapine-associated myocarditis[2] and one describing quetiapine-associated cardiomyopathy.[3] Another case report described a fatal dilated cardiomyopathy thought to be due to methylphenidate, but may also have been due to quetiapine.[4]

As quetiapine and clozapine are both benzazepine derivatives with similar chemical structures,[5] given the cardiac toxicity associated with clozapine, there is a clear possibility that quetiapine could have a similar effect. Clozapine is usually viewed as leading to myocarditis in the initial stages of treatment, but a recent New Zealand case series showed that 20% were diagnosed after a month of treatment and 12% diagnosed after a year.[6]

Dilated cardiomyopathy would necessarily present later than a causative myocarditis. There were cases of cardiomyopathy associated with clozapine occurring after 3 years of treatment, with a mean duration of treatment of 12 months.[1]

In conclusion, we have presented two cases of quetiapine-associated cardiomyopathy, one of whom died. Along with the five previously reported cases,[2–4,7] this may indicate an adverse drug reaction signal that warrants further evaluation.

Although quetiapine was not implicated in a data mining study examining cardiotoxicity associated with antipsychotic drugs, the study relied on adverse drug reactions being reported to a pharmacovigilance centre.[7]

It is accepted that adverse reactions are widely underreported. We hope that this paper will prompt such reports to facilitate further analysis.

Author information: Sean Coffey, Registrar, Department of Cardiology, Dunedin Hospital and Clinical Lecturer, Department of Medicine, University of Otago, Dunedin; Michael Williams, Cardiologist, Department of Cardiology, Dunedin Hospital and Clinical Associate Professor, Department of Medicine, University of Otago, Dunedin

Correspondence: Dr Michael Williams, Cardiology Department, Dunedin Hospital, 201 Great King Street, Dunedin 9016, New Zealand. Fax: +64 (0)3 4747655; email: michael.williams@otago.ac.nz

References:

1. Kilian JG, Kerr K, Lawrence C, Celermajer DS. Myocarditis and cardiomyopathy associated with clozapine. Lancet. 1999;354:1841-5.
2. Roesch-Ely D, van Einsiedel R, Kathofer S, et al. Myocarditis with quetiapine. Am J Psychiatry. 2002;159:1607-8.
3. Bush A, Burgess C. Fatal cardiomyopathy due to quetiapine. N Z Med J. 2008;121(1268). http://www.nzma.org.nz/journal/121-1268/2909/
4. Nymark TB, Hovland A, Bjørnstad H, Nielsen EW. A young man with acute dilated cardiomyopathy associated with methylphenidate. Vasc Health Risk Manag. 2008;4:477-9.
5. Booth RG. Psychotherapeutic drugs: antipsychotic and anxiolytic agents. In: Lemke T, Williams DA, editors. Foye's Principles of Medicinal Chemistry. 6th ed. Philadelphia: Lippincott Williams and Wilkins; 2007, p601-630.
6. Hill GR, Harrison-Woolrych M. Clozapine and myocarditis: a case series from the New Zealand Intensive Medicines Monitoring Programme. N Z Med J. 2008;121(1283). http://www.nzma.org.nz/journal/121-1283/3288/
7. Coulter DM, Bate A, Meyboom RH, et al. Antipsychotic drugs and heart muscle disorder in international pharmacovigilance: data mining study. BMJ. 2001;322:1207-9.

THE NEW ZEALAND MEDICAL JOURNAL
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In-situ and lobular, but not as we know it

Paul Samson, Richard Harman

A 54-year-old woman presented with suspicious micro-calcifications in the right breast upper outer quadrant on a screening mammogram (Figure 1). Relevant family history included breast cancer diagnosis in a maternal grandmother in her late 40s and in the patient's mother in her 70s. Approximately 6 years prior to presentation the patient had undergone stereotactic biopsies of micro-calcifications in right breast upper outer quadrant.

Histology at that stage revealed benign fibrocystic change with micro-calcification. Stereotactic core biopsies in the new 9 mm area of radiologically indeterminant micro-calcification now showed features of pleomorphic lobular carcinoma in-situ (PLCIS). Subsequent hook-wire localisation excision of this area found a residual 3.5 mm area of low grade PLCIS (Figure 2), with clear resection margins.

Figure 1. Screening mammogram work-up view showing 9 mm cluster of micro-calcifications in right breast upper outer quadrant

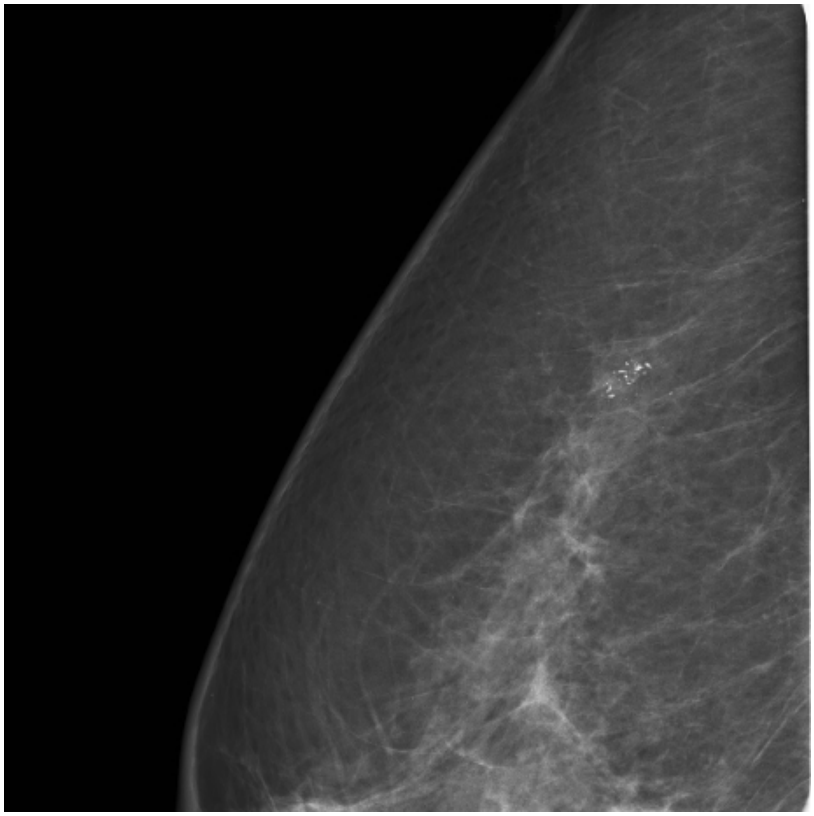
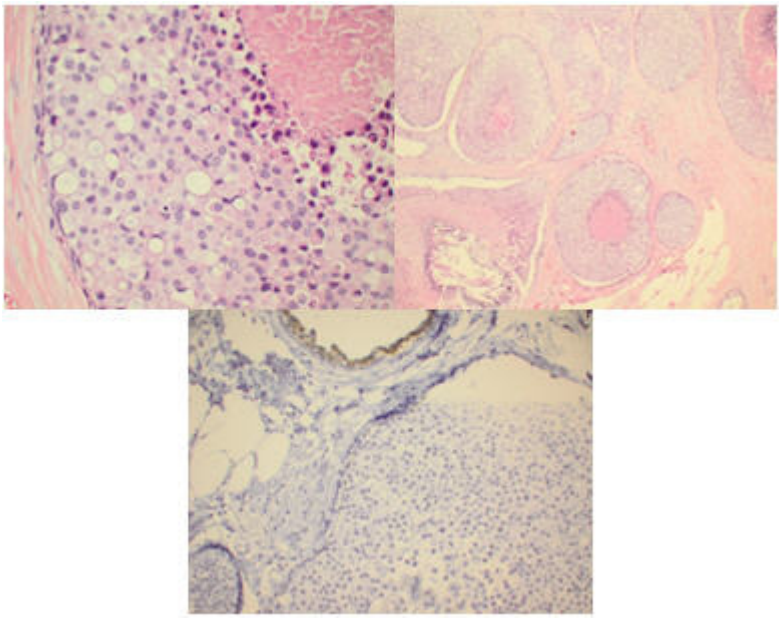


Figure 2. (left pane) Pleomorphic epithelial cells with eccentric nuclei and abundant cytoplasm; (right pane) Some duct spaces show central comedo-type necrosis and micro-calcification; (lower pane) The atypical cells are negative staining for E-cadherin



PLCIS, originally described in 1996, consists of lobular cells with marked pleomorphism and large eccentrically placed nuclei.[1] These cells are cytologically more discohesive than in classic LCIS (CLCIS). Additional findings of central necrosis, calcifications and negative staining for E-cadherin are frequently present.

Central necrosis and calcifications are otherwise rarely seen in classic LCIS, but are common in ductal carcinoma in-situ (DCIS).[2] PLCIS may be associated with an infiltrating pleomorphic carcinoma which has similar cytologic appearance and a poor prognosis. Compared with CLCIS, PLCIS shows significantly higher Ki67 index, lower oestrogen receptor and progesterone receptor expression, and higher incidence of HER2 gene amplification.[3] The histologic features, biomarker profile, and genomic instability suggest a more aggressive behaviour of this form of LCIS.[4] Multi-focal disease is also reported.[3]

There is a lack of high quality, extensive follow-up in patients with PLCIS. Current recommendations are to treat these patients more like patients with DCIS than with LCIS.[5] Patients thus require excision to clear margins with consideration of post-operative irradiation. Axillary lymph node biopsy or dissection is not necessary in the management of LCIS. If however a pleomorphic invasive component is present, axillary staging should be performed, followed by systemic therapy where indicated.

The benefit of selective oestrogen receptor modulators (SERMs) in patients with PLCIS is undefined, as subset analysis of patients with PLCIS is not possible from the major breast cancer chemoprevention trials.[6] LCIS is not typically tested for hormone receptor expression.

Despite the lack of data in PLCIS, the current recommended approach parallels that for pure DCIS with mastectomy or lumpectomy plus radiotherapy, followed by a SERM.

In summary, pleomorphic LCIS is a less known entity that requires more aggressive treatment than with classic-type LCIS. Multidisciplinary discussion involving radiologists, pathologists, breast surgeons and oncologists is indicated in optimal management of these patients.

Author information: Paul Samson, Breast & Endocrine Fellow; Richard Harman, Breast & Endocrine & Melanoma Surgeon; Department of General Surgery, North Shore Hospital, Auckland

Correspondence: Paul Samson, Department of General Surgery, North Shore Hospital, Private Bag 93 503, Takapuna, North Shore City 0740, New Zealand. Email: teamsamson@hotmail.com

References:

1. Frost AR, Tsangaris TN, Silverberg SG. Pleomorphic lobular carcinoma in situ. Pathol. Case Rev. 1996;1:27.
2. Georgian-Smith D, Lawton TJ. Calcifications of lobular carcinoma in situ of the breast: radiologic-pathologic correlation. Am J Roentgenol. 2001;176:1255.
3. Chen YY, Hwang ES, Roy R, et al. Genetic and phenotypic characteristics of pleomorphic lobular carcinoma in situ of the breast. Am J Surg Pathol. Nov 2009;33(11):1683-94.
4. Sneige N, Wang J, Baker BA, et al. Clinical, histopathologic, and biologic features of pleomorphic lobular (ductal-lobular) carcinoma in situ of the breast: a report of 24 cases. Mod. Pathol. 2002;15:1044-1050.
5. Sabel MS. Lobular carcinoma in situ. UpToDate [updated September 2009]. Available from: www.utdol.com
6. Anderson BO, Calhoun KE, Rosen EL. Evolving concepts in the management of lobular neoplasia. J Natl Compr Canc Netw. 2006;4:511.

THE NEW ZEALAND MEDICAL JOURNAL
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Boutonnière

Thattungal M Anoop, Puthukuli N Mini, Puthiyaveettil K Jabbar

Clinical

A 60-year-old lady presented with arthritis and generalised weakness. She had experienced frequent exacerbations and remissions of arthritis and had developed progression of hand and foot deformities over the last 6 years. Figures 1 and 2 are clinical images of the hand. *What are the findings and what is the diagnosis?*

Figure 1

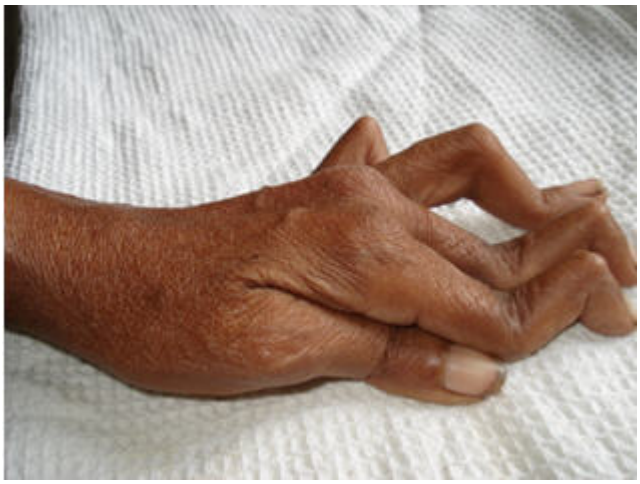
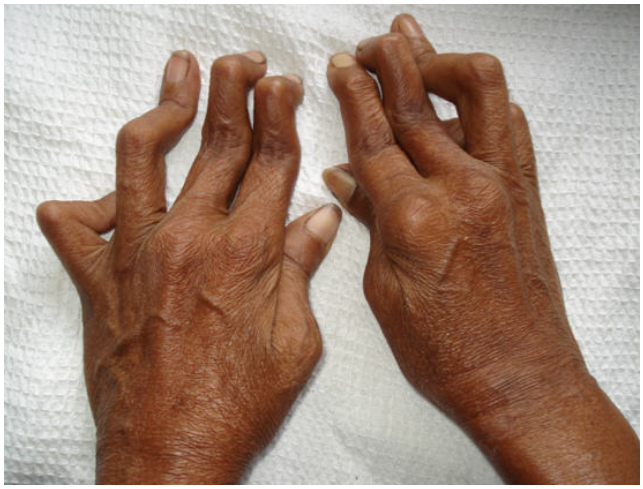


Figure 2



Answer

Examination of hands (Figures 1 and 2) showed ulnar deviation of fingers, hyperextension of the proximal interphalangeal joint with flexion of the distal interphalangeal joint (swan-neck deformity), flexion of the proximal interphalangeal joint with hyperextension of the distal interphalangeal joint (boutonnière/button-hole deformity) and Z-shaped deformity of the thumb. No extra-articular manifestations were present.

Laboratory data revealed haemoglobin of 9 gm/dl, ESR 120 mm/hour, total leucocyte count 8100/mm^3 with normal differential count. Blood sugar 110 mg/dl, creatinine 1.2 mg/dl; serum uric acid 5 mg/dl. Rheumatoid factor was positive titre 390 IU/ml. CRP was elevated at 6 mg/dl. ANA was negative.

Rheumatoid arthritis is a progressive, disabling disease which can lead to long-term deformity and disability.

In rheumatoid arthritis patients, the metacarpophalangeal, proximal interphalangeal, and wrist joints are involved earlier and more frequently than any other joint of the body; this involvement is collectively defined as 'rheumatoid hand disease'.

Permanent damage to the joints occurs at a very early stage in the disease. So establishing the most effective therapy with early arthritis, may prevent deformities in future.

Once the patient develops established joint deformities, damage is usually irreversible.

Author information: Thattungal M Anoop, Puthukuli N Mini, Puthiyaveettil K Jabbar; Dept of Internal Medicine, Kottayam Medical College, Kottayam, Kerala, South India

Correspondence: Dr TM Anoop, Dept of Internal Medicine, Kottayam Medical College, Kottayam, Kerala, South India – 686008. E-mail: dranooptm@yahoo.co.in

THE NEW ZEALAND MEDICAL JOURNAL
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Do senior medical students know enough clinical anatomy?

Background—A sound knowledge of clinically relevant anatomy is essential for safe and effective medical practice.[1] Anatomy teaching to medical students has decreased as a consequence of curricular changes. It is estimated that there has been an 80% reduction in anatomy teaching in Australasian medical schools since the introduction of problem-based medical graduate programmes, with more than 90% of all anatomy teaching occurring in the early years of medical courses.[2] Whilst this reduction was to some extent an appropriate response to unnecessary rote learning of clinically marginal information, the concern is that the pendulum has swung too far in the opposite direction and students are failing to learn sufficient clinical anatomy. Anatomical ignorance among junior doctors has been linked to an increase in medicolegal claims,[3,4] and has not escaped media attention.[5] We attempted to gauge knowledge of clinical anatomy among senior medical students at the Dunedin School of Medicine.

Methods—The authors drafted 32 clinical anatomy questions which were reviewed by nine specialists in medicine, general surgery, and orthopaedics. The 20 questions given the greatest priority formed the survey tool. With University ethical approval, all fourth, fifth and trainee intern (TI) year medical students at Dunedin School of Medicine in 2010 were invited to participate. All had been exposed to the same anatomy curriculum. During a whole class session, students individually answered the 20 questions (1 minute per question). Scores were collated and analysed using PASW Statistics 18.0®, and differences between cohorts tested using ANOVA.

Results—Participation rates in years 4 and 5 were 77% and 82%, respectively but due to concurrent electives, the TI response rate was just 38%. Out of a maximum score of 39, the mean score of 5th year medical students was 25.1, which was significantly higher than 4th year (23.1) and TI (23.6) students (P=0.03). The proportion of 4th year, 5th year, and TI students who scored less than 50% was 20%, 12%, and 15%, respectively. All cohorts scored less than 50% in four questions: the long thoracic nerve and winging of the scapula; the scaphoid bone and tenderness in the anatomical snuff box; structures palpable on digital rectal examination; and vulnerability of the spinal accessory nerve during lymph node biopsy in the posterior triangle of the neck (Table 1).

There was no significant difference between the three years in scores relating to seven 'medical anatomy' questions (mean 4th year score 10.2, 5th year 10.9, TI 9.8; maximum score 12.5; P=0.07) but scores for six 'orthopaedic anatomy' questions were significantly higher among fifth year and TI students (mean 4th year score 3.1, 5th year 5.1, TI 5.8; maximum score 10.5; P<0.01) and scores for seven 'general surgical anatomy' questions declined progressively (mean 4th year score 9.6, 5th year 8.1, TI 7.8; maximum score 16; P<0.01) (Figure 1).

Table 1. Mean scores for 20 clinical anatomy questions

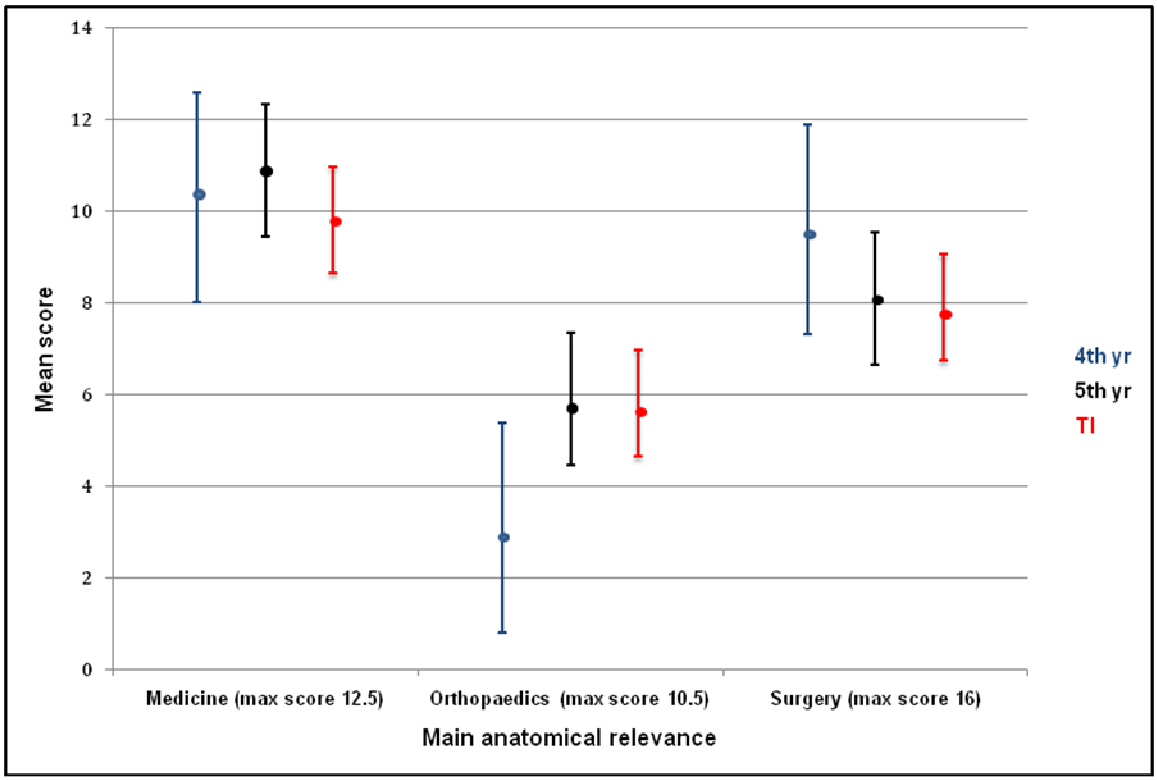
Variables	Q1 Lumbar puncture	Q2 Pleural tap	Q3 Cranial nerve III palsy	Q4 Chest radiograph	Q5 Chest surface anatomy	Q6 Bronchial foreign body	Q7 Limb reflex	Q8 Rotator cuff muscles	Q9 Long thoracic nerve	Q10 Scaphoid fracture
4th year	2.0	1.4	0.7	0.6	1.5	1.8	0.7	1.5	0.3	0.4
5th year	2.6	1.3	0.7	0.6	1.3	1.7	1.0	1.9	1.1	0.9
TI year	1.4	1.6	0.8	0.8	1.3	1.6	0.9	1.7	1.0	1.0
Total mean (SD)	2.2 (0.8)	1.4 (0.8)	0.7 (0.4)	0.7 (0.4)	1.4 (0.5)	1.7 (0.5)	0.8 (0.5)	1.7 (0.5)	0.7 (0.7)	0.7 (0.4)
Max score	3	2	1	1	2	2	1.5	2	2	2
Category	Medicine	Medicine	Medicine	Medicine	Medicine	Medicine	Medicine	Orthopaedics	Orthopaedics	Orthopaedics
	Q11 Knee ligaments	Q12 Radial nerve	Q13 Axillary nerve	Q14 Femoral nerve	Q15 Appendix	Q16 Digital rectal examination	Q17 Great saphenous vein	Q18 Duodenal ulcer	Q19 Accessory nerve	Q20 Abdominal CT scan
4th year	0.6	1.3	0.2	1.5	1.1	0.8	0.8	0.9	1.0	3.1
5th year	0.9	1.4	1.0	1.8	1.0	0.6	0.6	1.1	0.4	2.3
TI year	0.9	1.4	0.9	1.1	0.9	0.9	0.7	1.0	0.5	2.6
Total mean (SD)	0.8 (0.3)	1.4 (0.6)	0.8 (0.5)	1.6 (0.6)	1.0 (0.6)	0.7 (0.4)	0.7 (0.7)	1.0 (0.6)	0.6 (0.8)	2.7 (0.7)
Max score	1	2	1.5	2	2	2	2	2	2	4
Category	Orthopaedics	Orthopaedics	Orthopaedics	Surgery	Surgery	Surgery	Surgery	Surgery	Surgery	Surgery

Unshaded columns: no significant difference between mean cohort scores

Lighter shaded columns: significant difference between cohort scores but mean score >50% of maximum

Darker shaded columns: significant difference between cohort scores and mean score <50% of maximum

Figure 1. Mean clinical anatomy scores of student cohorts



Discussion—The knowledge of clinical anatomy among our senior medical students varied but mean scores for each year were between 59% and 64% in this straightforward test of clinical anatomy. If a minimum standard is arbitrarily set at 50% of the maximum score, then 15% of TIs failed to reach this standard. In a UK survey, 61% of clinicians felt that junior doctors had an inadequate knowledge of anatomy for safe clinical practice.[6] Only one-third of senior medical students at the University of Auckland considered that they knew enough clinical anatomy to practice safely.[7] Currently, there is no consensus on what constitutes an appropriate level of clinical anatomy for medical students and junior doctors.[2,8]

Our results suggest that some clinical anatomy topics are retained better than others. Of concern is the apparent progressive decline in 'general surgical anatomy' knowledge. This may reflect that anatomy education in New Zealand (and the vast majority of other medical schools) is almost exclusively delivered in the early years of medical training.[2] Knowledge of 'orthopaedic anatomy' appears to be reasonably well retained which may be related to orthopaedic teaching and/or the small clinical anatomy input into orthopaedic runs. However, a broader range of questions may have resulted in a different conclusion. In a survey of senior medical students and junior doctors in the UK only one-third could correctly name five or more human carpal bones.[9] Mean scores for two of our six questions relating to cranial or peripheral nerves were below 50% of the maximum score, suggesting particular weaknesses in this area and echoing the findings of another study.[6]

In our opinion, a modest amount of clinical anatomy teaching should be integrated into the later years of medical undergraduate training and not essentially restricted to second and third years. Such teaching fits well with the interpretation of modern imaging and could be incorporated relatively seamlessly into existing medical and surgical teaching blocks, a proposal strongly supported by senior medical students,[10] newly qualified doctors,[8] and experienced clinicians.[6] This would however require appropriate resourcing and the availability of clinical anatomists.

Sultan Al-Shaqsi
Fourth-year Medical Student
Dunedin School of Medicine

Mark D Stringer
Professor of Anatomy
Otago School of Medical Sciences
University of Otago, Dunedin
mark.stringer@anatomy.otago.ac.nz

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References:

1. Ahmed K, Rowland S, Patel V, et al. Is the structure of anatomy curriculum adequate for safe medical practice? Surgeon. 2010 ;8:318-24.
2. Craig S, Tait N, Boers D, McAndrew D. Review of anatomy education in Australian and New Zealand medical schools. ANZ J Surg. 2010;80:212-6.
3. Ellis H. Medico-legal litigation and its links with surgical anatomy. Surgery. 2002;20:i.
4. McKeown P, Heylings D, Stevenson M, et al. The impact of curricular change on medical students' knowledge of anatomy. Med Educ. 2003;37:954-61.
5. Cresswell A. Doctors fail basic anatomy. The Weekend Australian. 2006 6 May 2006.
6. Waterston S, Stewart I. Survey of clinicians' attitudes to the anatomical teaching and knowledge of medical students. Clin Anat. 2005;18:380-4.
7. Insull PJ, Kejriwal R, Blyth P. Surgical inclination and anatomy teaching at the University of Auckland. ANZ J Surg. 2006;76:1056-9.
8. Fitzgerald J, White M, Tang S, et al. Are we teaching sufficient anatomy at medical school? The opinions of newly qualified doctors. Clin Anat. 2008;21:718-24.
9. Spielmann P, Oliver C. The carpal bones: a basic test of medical students' and junior doctors' knowledge of anatomy. Surgeon. 2005;3:257-9.
10. Dawson AG, Bruce SA, Heys SD, et al. Student views on the introduction of anatomy teaching packages into clinical attachments. Clin Anat. 2009;22:267-72.

THE NEW ZEALAND MEDICAL JOURNAL
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Spiriva (Respimat) increases mortality by 52% in patients with COPD—time to take action

The study by Singh and co-authors from John Hopkins University Medical School published in a recent issue of *BMJ*[1] has now clearly demonstrated and quantified the increased risk of cardiovascular mortality associated with the use of Respimat Soft Mist Inhaler containing tiotropium bromide (Spiriva) in chronic obstructive pulmonary disease (COPD).

The study was conducted using robust statistical and clinical methodology and the results will have worldwide clinical implications as Spiriva is currently approved for use in 55 countries. Singh et al compared Spiriva to placebo and reported a 52% (RR: 1.52; 95%CI: 1.06–2.16) increase in mortality with the 5 µg/day dose which is the recommended dosage of Spiriva use in New Zealand. However, it is not unrealistic that many COPD patients in New Zealand are using 10 µg/day and therefore further increasing their risk of mortality as reported in the study by 115% (RR: 2.15; 95%CI: 1.03–4.51).

Studies have reported much higher peak plasma concentrations with the use of Spiriva than the powered formulations.[2] Nevertheless, the concern with this class of medication (anticholinergics) in patients with COPD is not new with the Lung Health study published in 2002[3] reporting increased cardiovascular mortality with anticholinergic medications. In our study published this year[4] we looked at the effectiveness of tiotropium in Spiriva compared to tiotropium contained in other inhaler devices.

This study was conducted due to claims by the pharmaceutical industry that Spiriva provided greater effectiveness compared to other inhaler devices. We found no differences between any of the devices containing tiotropium. Although mortality was not reported adequately in our study due to insufficient data we did raise this concern of increased cardiovascular mortality which was consistently being reported in literature and called for urgent investigation. The Singh et al trial has now provided this conclusive evidence and we urgently need to take appropriate action.

The safety of tiotropium has been the subject of lengthy investigations by the US Food and Drug Administration (US FDA) including an advisory committee hearing held in 2009. However, these US FDA investigations looked at the safety of tiotropium powder and not the Respimat Soft Mist Inhaler. It is not surprising why the US FDA has as yet not approved Spiriva in the United States and now given the evidence from the latest study it seems highly unlikely that Spiriva will ever get approval in the United States. This does very much remind us all of the world-wide approval of thalidomide which also never gained approval in the United States.

Although in New Zealand there is a precautionary warning in the package insert, Medsafe now needs to update their warning to all prescribers throughout the country and urgently revise the Spiriva product datasheet to make the use of Spiriva a contraindication in patients with pre-existing arrhythmias who are at the highest risk

of death. Medsafe should also request the manufacturer of Spiriva to send a “Dear Doctor” letter to all prescribers in New Zealand.

Dr Felix S F Ram
Senior Lecturer in Clinical Pharmacology
School of Health and Social Sciences
Massey University, Auckland
fsfram@yahoo.co.uk

References:

1. Singh S, Loke YK, Enright PL, Furberg CD. Mortality associated with tiotropium mist inhaler in patients with chronic obstructive pulmonary disease: systematic review and meta-analysis of randomised controlled trials. BMJ 2011, June 14;342:d3215. doi:10.1136/bmj.d3215.
2. van Noord JA, Cornelissen PJ, Aumann JL, Platz J, Mueller A, Fogarty C. The efficacy of tiotropium administered via Respimat Soft Mist Inhaler or HandiHaler in COPD patients. Respir Med. 2009 Jan;103(1):22-9.
3. Anthonisen NR, Connett JE, Enright PL, Manfreda J; Lung Health Study Research Group. Hospitalizations and mortality in the Lung Health Study. Am J Respir Crit Care Med. 2002 Aug 1;166(3):333-9.
4. Ram FS, Carvallho CR, White J. Clinical effectiveness of the Respimat inhaler device in managing chronic obstructive pulmonary disease: evidence when compared with other handheld inhaler devices. Int J Chron Obstruct Pulmon Dis. 2011;6:129-39.

THE NEW ZEALAND MEDICAL JOURNAL
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Response to 'lifetime cardiovascular risk' letter

I'm delighted to have the opportunity to respond to the letter by Dr Ben Gray about lifetime cardiovascular risk.[1] I would like to respectfully point out that there were several errors in his letter quoting the PROSPER trial.[2] This trial was not undertaken in men over 75, it was conducted in men and women aged 70 to 82 years, and it did not show a reduction in mortality from cardiovascular disease, nor a significant increase in cancer deaths.

Not withstanding these errors, Dr Gray raises an important issue about substitution of deaths from cardiovascular disease for cancer deaths. It has long been recognised that if you decrease the incidence of a disease that the incidence of another disease may increase. However, the contribution of a decline in heart disease mortality to an increase in cancer mortality is small.[3]

The use of lifetime risk assessment would inform many individuals who in middle age are falsely reassured by a low New Zealand 5-year risk assessment, that their risk of dying from heart disease is 50% and their life expectancy is reduced by 10 years. These outcomes may well be changed by modifying their lifestyle.[4,5]

I would argue that it would be better to live these 10 years and to face whatever other illnesses might occur. Treatment for these illnesses may undergo enormous improvements over that time. Furthermore one doesn't know whether one will develop cancer but we can quantify the risk and reduce the occurrence of cardiovascular disease. Besides causing death, cardiovascular disease, may cause devastating strokes and myocardial infarction. The latter may be associated with severe heart failure symptoms and/or arrhythmias requiring medications, which may have major side effects. In addition angioplasty or surgery may be necessary.

Changes in lifestyle are difficult to achieve and need to be maintained over many years to prevent cardiovascular disease which develops over decades. I would expect attempts to change lifestyle at the age of 60, when the 5-year risk becomes 10%, would be doomed to fail.

I believe that assessing and informing people about their lifetime risk is much more likely to change lifestyle and outcomes than nihilism.

Not using lifetime risk may be appropriate when life expectancy is about 5 years, but not always.

As George Burns (1896–1996) said:

'If you live to a 100 years you've got it made. Very few people die past that age'.

Professor Harvey White
Director of Coronary Care & Green Lane Cardiovascular Research Unit
Green Lane Cardiovascular Service
Auckland City Hospital
Auckland, New Zealand

References:

1. Gray B. Lifetime cardiovascular risk [letter]. N Z Med J 2011;124(1336):96. http://www.nzmj.com/journal/124-1336/4719/content.pdf
2. Shepherd J, Blauw GJ, Murphy MB, et al. Pravastatin in elderly individuals at risk of vascular disease (PROSPER): a randomised controlled trial. Lancet 2002;360:1623-1630.
3. Llorca J, Delgado-Rodriguez M. Competing risks analysis using Markov chains: impact of cerebrovascular and ischaemic heart disease in cancer mortality. Int J Epidemiol 2001;30:99-101.
4. Lloyd-Jones DM, Leip EP, Larson MG, et al. Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. Circulation 2006;113:791-798.
5. Stewart RA, White HD. Reducing the lifetime cardiovascular risk of New Zealanders. N Z Med J 2007;120:U2708. http://www.nzmj.com/journal/120-1261/2708/content.pdf

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Warning Notice

This notice appeared in NZMJ May 1912;9(42):138.

There being some difficulty between the Lodges and their Doctors in the North Otago district, comprising the townships of Kurow, Duntroon and Ngapara and the surrounding neighbourhood, members of the Association are advised to make enquiries concerning the existing conditions from the Secretary of the Division (Dr. E. J. O'Neill, Dunedin), or from the General Secretary in Wellington before accepting any position offered by the lodges in this neighbourhood.

The same warning also applies to Kaikoura South, where there is also a disagreement with the Lodges. Intending applicants should apply for information from the Secretary of the Division in Christchurch, or from the General Secretary, Wellington.

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Pharmacovigilance—New Zealand the leader

Pharmacovigilance involves keeping an eye on drug interactions involving adverse events, and the WHO has a database called VigiBase. This receives continuous submission of 'individual case safety reports' – ICSRs – from members of the WHO Programme for International Drug Monitoring.

We note that most submissions seem to be made from the developed countries but the WHO aims to extend their reach. A figure in a recent report shows the top 20 countries in terms of their reporting rates per million inhabitants per year, during the last 5 years. New Zealand is still at the top. Must be good.

Uppsala Reports 2011;April:4–5.

Bacterial contamination of doctors' wrists?

The Department of Health in England introduced guidelines in 2007 banning healthcare workers from wearing white coats or other long-sleeved garments as part of efforts to reduce nosocomial bacterial transmission. So the traditional long-sleeved white coat disappeared from the wards in England and probably most other places, including New Zealand.

White coat devotees asked for evidence, but none was forthcoming—until now. This report summarises a study that showed no significant difference in bacterial or in methicillin resistant *Staphylococcus aureus* (MRSA) contamination between doctors in the short sleeved uniform and those in the long sleeved coats (J Hosp Med 2011, doi:10.1002.jhm864). Furthermore, bacterial contamination was found after only 3 hours wear of freshly laundered uniforms.

So long white coat sleeves are no more evil than short sleeves. It would seem that frequent hand cleansing would be appropriate, whatever uniform is favoured.

BMJ 2011;342:d1079.

Rehabilitation after stroke: would body-weight-supported treadmill exercise help?

This paper starts with the proposition that locomotor training, including the use of body-weight support in treadmill stepping may improve the rate of recovery from a stroke. The study aims to elucidate whether it does and whether it should be started early or late (i.e. at 2 or 6 months after the stroke). 408 patients with walking impairment 2 months after their stroke were randomised to 3 groups. One group received training on a treadmill with the use of body-weight support 2 months after the stroke had occurred (early locomotor training), the second group received this training 6 months after the stroke had occurred (late locomotor group), and the third group participated in an exercise programme at home managed by a physical therapist 2 months after the stroke (home-exercise programme).

Neither early or late treadmill intervention was found to be superior to home managed exercise managed by a physiotherapist.

N Engl J Med 2011;364:2026–36.

Coronary-artery bypass (CABG) surgery in patients with left ventricular dysfunction

CABG is a well established treatment for patients with angina, extensive coronary artery disease and reasonably well preserved left ventricular function. However its place in patients with coronary artery disease and heart failure has not been clearly established.

Hence this study in which 1212 patients with an ejection fraction of less than 35% and coronary disease amenable to coronary-artery bypass grafting were randomly assigned to receive optimal medical therapy for heart failure and coronary disease (602 patients) or to receive optimal medical therapy plus CABG (610 patients).

At 5 years there was no difference between either cohort in the rate of death from any cause. An editorial reviewer agrees that the use of CABG surgery in such patients is inappropriate.

N Engl J Med 2011;364:1607–16.

Radial versus femoral access for coronary angiography in patients with acute coronary syndromes

Apparently these alternatives are a source of ongoing controversy amongst interventional cardiologists. This randomised study enrolled 7021 patients from 158 hospitals in 32 countries. They randomised to either radial or femoral access and a number of relevant end points were assessed.

Both interventions resulted in similar rates of the composite of death, myocardial infarction, stroke, or non-CABG-related major bleeding. Large haematomas, pseudoaneurysms, and patients' preference were all in favour of the transradial approach.

The length of stay in hospital was 4 days in both cohorts. The authors of the study conclude that both routes are safe and effective and the lower rate of local vascular problems is a significant plus for the radial approach.

Lancet 2011;377:1409–20.

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Jacob Madison Beck-Jaffurs

09/01/1987 – 11/03/2011

Most obituaries in medical journals are about distinguished doctors, reflecting on the contributions of a full career. This is not one of those stories. It is however, a story well worth telling. It is the story of a young man, fit, vibrant, intelligent and compassionate. A young man that had yet to complete medical school—in his final year as a Trainee Intern at the University of Auckland—when he died in a diving accident. A young man who had not yet had the chance to make his mark in medicine or even choose his specialty.

Although he was too humble to ever think of himself as a life changer, Jacob had a profound effect on those he met. He brought a rare balance of serenity and intense joy to the pressures and pace of a hectic world. He was passionate about medicine, and he relished learning how to make sick and injured people well.

Jacob's striking quality was a unique pragmatic altruism, which left every room brighter for his presence, and drove him to create the nationwide charitable foundation *Professional Pathways Trust* which continues to better the lives of hundreds of young New Zealanders today.

Jacob Beck-Jaffurs was born in the USA, and moved with his family to Whangarei as an 11-year-old boy. His father, Chip, is an emergency physician and his mother, Barbara, a lawyer.

Jacob grew up surrounded by people who encouraged and inspired him to excel. He lived life enthusiastically, and his drive for excellence went hand in hand with enjoying everything he did to the fullest. He was a waterman who honed his surfing skills at the Raglan Surf Academy and competed nationally. Later, he became an avid freediver, exploring New Zealand's coastline to spearfish or simply to watch the majesty of the depths play out before him. A keen athlete, he played soccer and volleyball competitively, and was training as a boxer. He loved to snowboard, to climb mountains, to explore remote destinations and generally to participate in any playful activity or wilderness adventure that would get him into the open air.

After completing high school, Jacob was accepted into medical school and maintained consistently good grades throughout. He took a year out from his studies after his

third year to work, travel and surf on every windswept beach he could find in Central America. Jacob managed to achieve everything he put his mind to, whether it involved the discipline of sporting competition, or locking himself in the library for weeks at a time to study for exams, the worst kind of torture for an active young man so in love with the outdoors.

As a man blessed with opportunity, Jacob saw the disparities in opportunities available to other students. Unselfish and empathetic, Jacob looked at his classmates from poor rural high schools and reflected on the different trajectories he had followed from many of those peers. Never one to look at a problem without seeking a solution, Jacob, in his 4th year of medicine, and his lifelong friend, lawyer Michael Forster, founded the Professional Pathways Trust. Jacob spent every hour between hospital commitments and surf breaks working to develop this charity into what has become one of the largest not-for-profit mentoring services in New Zealand high schools.

Professional Pathways provides mentoring to students from low-decile high schools to guide them into professional careers such as medicine, law and engineering. Young professionals volunteer to give friendly, empathetic advice and mentoring to students who might not otherwise have a connection with the professional world. Jacob believed that exposure and encouragement can make a profound difference in the lives of young people and he wanted to see some of these students expand their dreams and achieve greater things than they had believed themselves capable of.

When meeting with students from various high schools, Jacob had them spellbound. They were mesmerized by this energetic young man who was able to conclusively demonstrate that you could look like you just left the beach and be a doctor. He would sit and listen, quietly acknowledging their fears and feelings of inadequacy and boosting their confidence by working through actual medical school exam papers and proving that common sense, commitment and a keenness to learn are the only tools required to survive medical school.

A scholarship has been established in Jacob's memory, which will be awarded annually to an exceptional student who has received mentoring through the Professional Pathways programme. You can learn more about this scholarship, or Jacob’s charity, at www.professionalpathways.org Better yet, make a contribution, or get involved.

The years I knew Jake were too short, but the impact he made on my life and on the lives of hundreds of others, will last forever. He was an athlete, a doctor, a professional and a loyal friend. The Jacob I knew was selfless and modest, quietly going about the business of bettering himself and supporting others without ever seeking recognition. He walked through this life with a perpetual grin on his face, forever chuffed with the simplest things, like a sunny lunch break in the Auckland Domain or an uncrowded beach. The high expectations he demanded of himself inspired everyone around him and I can only hope that my medical practice in the future will be worthy of his example.

Jacob lived with dignity, passion and grace and he died doing something he loved. He will graduate posthumously with the class of 2011 in November. Jacob was just 24 years old.

We will miss him forever.

Many thanks to Chip Jaffurs, Barbara Beck and Michael Forster for their support and contributions to this obituary.

Rachel Lister wrote this obituary (Trainee Intern at University of Auckland & Trustee at Professional Pathways Trust).

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David Idwal Tavener-Smith

16 September 1954 – 27 May 2011; BA (Natal) MBBCh (Wits) DTM&H (Wits) FFRad (D)(SA)

Senior Consultant Radiologist: Tauranga Hospital
Artist, Photographer, Radiologist, Teacher, Fish Botherer Extraordinaire

David was a larger than life, universal man. He died unexpectedly, at the age of 56, in Tauranga. The large crowd gathered at his farewell, mirrored some of the important facets of David's life: family from South Africa and Australia, friends, neighbours, past and present colleagues in the medical world, kayakers, mountain bikers and "fellow fishing fanatics".

There were many commendations about David at his farewell memorial service. Dr Phil Borrie, senior consultant radiologist at Tauranga Hospital, colleague and long-standing friend, paid tribute to a remarkable man (*his words from the Eulogy are in italics).*

"Stubborn, thorough, gruff, caring, forthright, methodical, eloquent, humorous, contained, fisherman extraordinaire, gardener, organised, direct, interested, interesting, knowledgeable, kind, independent, pedantic…", encapsulates David.

David was a multi-dimensional man, a polymath who was proficient in the arts and sciences and quite exceptional in his "ways with a trout".

During the memorial service it was said: "His was an unusual blend of skills—a radiologist with a Fine Arts background. He always said radiology was the artistic side of medicine where he got to view pictures all day: a curious blend of Photoshop and X-rays". David's passion was trout fishing, a man described as "*…one of New Zealand's most accomplished fly fishermen".*

David spent his formative years in Zambia, his school years in Northern Ireland and his university and early career years in South Africa. He reached New Zealand in the early 1990s. David loved what he saw in New Zealand and after a nomadic journey as a locum diagnostic radiologist in New Plymouth then Whangarei, he decided to put down roots in Tauranga, in 1994. He took to life at 'The Bay of Plenty', with alacrity claiming "plenty of sun, plenty of fish, plenty of time off".

David was *"…passionate about Radiology, best diagnosis and also teaching of registrars, particularly teaching of the Barium studies". He was "methodical and pedantic at times and any house surgeon must surely have all the facts – all the W's would come out …*

- You want what?
- What for?
- When did this happen?

- What do the lab results show?
- What do you expect the test will reveal?
- When do you want the examination done?"

David was known for his razor sharp wit, dry humour and enthusiasm for the challenge of a good discussion. He would converse eloquently on a huge range of topics. His bookshelves bulged with a diversity of literature from radiological text, art, music, plants and novels to chaos theory.

He had a dedicated "fishing room" filled with the tricks of the trout whisperer's trade. The intricately tied flies combined the skills of an entomologist with those of an artist. He had a profound love of a quiet river bend, the natural landscape and the thrill of white water.

Although he never married (describing himself as "*not for domestic use*") he leaves behind his long-time partner, Gail Paskin. He is survived by his parents Maureen and Ron and brother Owen and his family in South Africa and brother Gwyn and his family in Australia. He was devoted to his family and they are so proud of his remarkable life and legacy.

David was a genuine Renaissance man. His knowledge was broad and deep and he had achieved excellence in Medicine, Art and Fishing. He lived his life so well.

Anne Tavener-Smith (Durban, South Africa) compiled this obituary and Dr Phil Borrie (Radiology Department, Tauranga) wrote of David in his eulogy.

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Denis Wordsworth Feeney

27 June 1920 – 24 May 2011

Denis was born in Wanganui, the third of three boys. His father James was a stock and station agent in Wanganui, and his mother Olive was the daughter of Charlie Wordsworth, one of the survivors of a shipwreck in 1876 on the Crozet Islands between the Cape of Good Hope and Antarctica.

In the Depression, Denis won a scholarship to Silverstream Boarding School and went on to attend Teachers College, where his training was interrupted by WW2. He enlisted with the British Royal Navy, working on the minesweeper HMS Waterwitch in the Irish Sea and the destroyer HMS Urchin in the Pacific. After the War, Denis enrolled at the Otago Medical School and managed to continue his pre-war involvement with theatre (in Wellington he had worked with a professional stage crew at The New Zealand Opera House, then reputed to be the largest and best equipped stage in Australasia). Shows that toured New Zealand traditionally opened in Dunedin: the first touring company after the war was The Italian Opera Company, and he also worked alongside the stage crew for other travelling shows including those of Laurence Olivier and JC Williamson.

Having qualified, Denis worked as a house surgeon in Wellington Hospital where he continued his involvement with the stage, re-met and married Cecil. In late 1953 the family moved to Mount Maunganui, just missing the train that eventually ended up in the river at Tangiwai, where Dr Fraser McDonald, soon to become Denis' brother-in-law, was one of the rescuers at that terrible accident.

At the Mount, Denis went into practice with Stan Debonair, and was very active in the local RSA. He was one of the generation of GPs who did round the clock house calls and claimed to be one of the few people to have crossed Tauranga Harbour in a horse and cart—a terrible storm prevented boats and planes from getting a seriously injured logger off Matakana Island, so they waited until low tide and met an ambulance at the Athenree side of the Harbour.

In the late 1960s the family moved to Auckland where Denis joined a practice in Blockhouse Bay. He was very active in medical circles and won a Nuffield Fellowship to investigate best practice at medical schools across the USA, Europe and Scandinavia. Over this period, Denis maintained an active lifestyle of hunting and spearfishing, taking the whole family on marvellous outdoors holidays.

After retiring from general practice at 60, Denis worked for the Health Department in Wellington. The Department's official retirement age was 60, so when he turned 65

they reluctantly said he had to go. Denis and Cecil moved back to Auckland and began a peripatetic life of rural locums, Denis throwing himself into good old-fashioned rural doctoring in places like Whataroa on the West Coast, where he was helicoptered out to sea to help injured fishermen or into the mountains to help injured hunters.

Denis was a keen camper and tramper. In rough country near Mt Cook one day, he was "hooping" (a gait between a walk and a run that very good trampers can keep up for hours) and was startled to hear someone rapidly approaching from behind; he threw himself out of the path, and was overtaken by a Ed Hillary coming along at a full run. In training for the Everest expedition, Hillary was carrying a full pack, with a full keg of beer perched on top!

Finally Denis and Cecil "really" retired to Henderson, but even there Denis would give lectures to the local medical group on esoteric things like osteo chondritis and other complaints that are commonly mis-or un-diagnosed.

In Denis' distinguished 50-year career, his intellect and deep insight into people gave him a rare and applauded medical diagnostic ability. This skill has touched the lives of hundreds of families. The great themes of Denis' life were the outdoors, the dramatic arts, medicine, teaching and family.

Denis will be much missed by Cecil, his wife of 59 years; by his 7 children: Clare, Denis, Michael, Susan, James, Margaret and Peter; and by his 17 grandchildren—Matthew, Peter, Anna and Michael; Daniel, Alex and Melanie; Clare and Madeleine; Matt, Tessa, Dillon and Blake; Timo and Jimmy; and Arlo and Francesca.

Clare Feeney, a professional speaker and environmental consultant from Auckland, wrote this obituary.