

The
**New Zealand
Medical Journal**

Journal of the New Zealand Medical Association

Vol 130 | No 1466 | 1 December 2017



**Asthma and Respiratory
Foundation NZ child and
adolescent asthma guidelines:
a quick reference guide**

**Physician advocacy in western medicine:
a 21st century challenge**

**Asbestos—worker
exposure, family disease**

**Teaching quality improvement to medical
students: over a decade of experience**

**New Zealand asthma
guidelines updated**

The
**New Zealand
Medical Journal**
Publication Information

published by the New Zealand Medical Association

NZMA Chair

Dr Kate Baddock

To contribute to the *NZMJ*, first read:
www.nzma.org.nz/journal/contribute

NZMJ Editor

Professor Frank Frizelle

Other enquiries to:

NZMA
PO Box 156
The Terrace
Wellington 6140
Phone: (04) 472 4741

NZMA Communications Manager

Sharon Cuzens

NZMJ Production Editor

Rory Stewart

© NZMA 2017

To subscribe to the *NZMJ*, email
julie@nzma.org.nz

Subscription to the *New Zealand Medical Journal* is free and automatic to NZMA members. Private subscription is available to institutions, to people who are not medical practitioners, and to medical practitioners who live outside New Zealand. Subscription rates are below. All access to the *NZMJ* is by login and password, but IP access is available to some subscribers.

Read our Conditions of access for subscribers for further information
www.nzma.org.nz/journal/subscribe/conditions-of-access

If you are a member or a subscriber and have not yet received your login and password, or wish to receive email alerts, please email: julie@nzma.org.nz

The NZMA also publishes the *NZMJ Digest*. This online magazine is sent out to members and subscribers 10 times a year and contains selected material from the *NZMJ*, along with all obituaries, summaries of all articles, and other NZMA and health sector news and information.

Subscription rates for 2017

New Zealand subscription rates

Individuals*	\$306
Institutions	\$530
Individual article	\$25

Overseas subscription rates

Individual	\$426
Institutions	\$571
Individual article	\$25

*NZ individual subscribers must not be doctors (access is via NZMA Membership)

New Zealand rates include GST. No GST is included in international rates.

Note, subscription for part of a year is available at pro rata rates.

Please email julie@nzma.org.nz for more information.

Individual articles are available for purchase by emailing nzmj@nzma.org.nz

NZMJDigest

published by the New Zealand Medical Association



NZMJDigest

<http://www.nzma.org.nz/publications/nzmjdigest>

The NZMA publishes the e-magazine NZMJDigest 10 times a year. It contains news and views from the profession and the NZMA, including the NZMA Chair's editorial, along with highlights from and links to the New Zealand Medical Journal.

Click on the image above to view the latest issue.

We welcome contributions from members and readers. To contribute to the NZMJDigest, please email digest@nzma.org.nz

EDITORIAL

7

New Zealand asthma guidelines updated
Richard Beasley, Robert J Hancox

GUIDELINES

10

Asthma and Respiratory Foundation NZ
child and adolescent asthma guidelines: a
quick reference guide

Innes Asher, David McNamara, Cheryl Davies,
Teresa Demetriou, Theresa Fleming,
Matire Harwood, Lorraine Hetaraka-Stevens,
Tristram Ingham, John Kristiansen, Jim Reid,
Debbie Rickard, Debbie Ryan

ARTICLES

34

Breastfeeding indicators among a nationally
representative multi-ethnic sample of New
Zealand children

Teresa Castro, Cameron Grant, Clare Wall,
Michaela Welch, Emma Marks, Courtney Fleming,
Juliana Teixeira, Dinusha Bandara, Sarah Berry,
Susan Morton

45

Teaching quality improvement to medical
students: over a decade of experience

Michelle R Wise, Bridget Kool, Lynn Sadler,
Roshini Peiris-John, Gillian Robb, Susan Wells

53

Deaf New Zealand sign language users'
access to healthcare

Joanne Witko, Pauline Boyles, Kirsten Smiler,
Rachel McKee

62

A New Zealand platform to enable genetic
investigation of adverse drug reactions

Simran DS Maggo, Eng Wee Chua, Paul Chin,
Simone Cree, John Pearson, Matt Doogue,
Martin A Kennedy

70

Acupuncture, ACC and the Medicines Act

Daniel J Ryan

78

Exposure to respirable crystalline silica in
the construction industry—do we have a
problem?

David McLean, Bill Glass, Andrea 't Mannetje,
Jeroen Douwes

VIEWPOINTS

83

Physician advocacy in western medicine: a
21st century challenge

Philip Bagshaw, Pauline Barnett

90

Asbestos—worker exposure, family disease

William Ivan Glass, Helen Clayson

CLINICAL CORRESPONDENCE

92

Fanconi anaemia and oral squamous cell
carcinoma: management considerations

Thasvir Singh, Kavin Andi

LETTER

96

Aggressive prostate cancer incidence in New
Zealand—"united we fall, divided we stand"

Venkatesh Vaidyanathan, Nishi Karunasinghe,
Vetrivhel Krishnamurthy, Chi Hsiu-Juei Kao,
Vijay Naidu, Radha Pallati, Alice Wang, Khanh Tran,
Prasanna Kallingappa, Anower Javed,
Syed M Shahid, Jonathan Masters, Clare Wall,
Ajit Narayanan, Lynnette R Ferguson

METHUSELAH

100

Tiotropium in early-stage chronic
obstructive pulmonary disease

100 YEARS AGO

101

Examination of the Heart, and Life
Insurance

PROCEEDINGS

102

Proceedings of the Waikato Clinical Campus
Biannual Research Seminar

Asthma and Respiratory Foundation NZ child and adolescent asthma guidelines: a quick reference guide

Innes Asher, David McNamara, Cheryl Davies, Teresa Demetriou, Theresa Fleming, Matire Harwood, Lorraine Hetaraka-Stevens, Tristram Ingham, John Kristiansen, Jim Reid, Debbie Rickard, Debbie Ryan

The New Zealand child and adolescent asthma guidelines provide health professionals who deliver asthma care with simple, practical and evidence-based guidance for the diagnosis and treatment of asthma in children of 15 years of age and below. Prior to this project, New Zealand's child and adolescent asthma guidelines had not been updated since 2005. While medications essentially have remained the same, the way they should be utilised has subtly changed in the last 12 years. The new guidelines align the latest research with specific information for the New Zealand context, including available medications and relevant content for treating Māori and Pacific adults with asthma. The aim of the guidelines is to improve outcomes and reduce inequalities, and discusses factors such as unhealthy indoor environments and inadequate income for the basics needed for wellbeing.

Breastfeeding indicators among a nationally representative multi-ethnic sample of New Zealand children

Teresa Castro, Cameron Grant, Clare Wall, Michaela Welch, Emma Marks, Courtney Fleming, Juliana Teixeira, Dinusha Bandara, Sarah Berry, Susan Morton

Although 97 percent of New Zealand children are breastfed initially, a large number are not being breastfed for as long as international guidelines recommend. New evidence from the *Growing Up in New Zealand* longitudinal study of more than 6,000 children and families (generalisable to the New Zealand national birth cohort) showed only one in six children achieved the World Health Organization-recommended six months of exclusive breastfeeding. One in eight achieved the recommendation of receiving some breast milk for two or more years. Duration of breastfeeding was also shown in the study to be associated with mothers' age, ethnicity, education, number of children and whether the pregnancy was planned.

Teaching quality improvement to medical students: over a decade of experience

Michelle R Wise, Bridget Kool, Lynn Sadler, Roshini Peiris-John, Gillian Robb, Susan Wells

Final year medical students at University of Auckland perform a clinical audit project within the constraints and context of a busy women's health service. Students work with a clinical supervisor to identify an area for potential improvement, set a standard of care, measure current practice, investigate reasons for not achieving the standard and make real-world suggestions to close the gap between research and observed practice. Since 2004, over 1,250 student projects have been completed, many of which have resulted in actual improvements to clinical care. Such experiential learning during medical school is important in preparing future doctors to incorporate quality improvement knowledge and skills into their daily practice.

Deaf New Zealand sign language users' access to healthcare

Joanne Witko, Pauline Boyles, Kirsten Smiler, Rachel McKee

Part of the District Health Boards Sub-Regional Disability Strategy is to develop a comprehensive New Zealand Sign Language (NZSL) policy for the Wairarapa, Hutt Valley and Capital and Coast District Health Boards. To support this, research was carried out to investigate the quality of access to health services for deaf NZSL users. A co-design approach was used to collect qualitative data. Results suggest deaf NZSL users face multiple barriers within the health system mainly stemming from language barriers and a lack of information accessible in NZSL.

A New Zealand platform to enable genetic investigation of adverse drug reactions

Simran DS Maggo, Eng Wee Chua, Paul Chin, Simone Cree, John Pearson, Matt Doogue, Martin A Kennedy

Pharmacogenomics is the study of how genes influence a patient's responses to drugs. This research area is helping to clarify how genetic differences contribute to the risk of side effects (ADRs) or failure of drug treatment, and how we might better tailor treatment to each patient, leading to improved safety and effectiveness. This study describes a Christchurch initiative to collect and study the genetic basis of ADRs.

Acupuncture, ACC and the Medicines Act

Daniel J Ryan

This study looked at acupuncture websites to see if they were breaching Section 58 of the Medicines Act, which prohibits claiming the ability to prevent, mitigate or cure a range of serious diseases. Seventy-three percent of the websites claimed they could treat/prevent mental illness, infertility and arthritis, 11% said they could treat/prevent cancer, 23% for diabetes, 19% for thrombosis and 14% for heart disease. This is the case despite a clear lack of evidence for the efficacy of acupuncture.

Exposure to respirable crystalline silica in the construction industry—do we have a problem?

David McLean, Bill Glass, Andrea 't Mannetje, Jeroen Douwes

Heavy exposure to dust containing very fine particles of silica has long been known to cause silicosis or a scarring of the lung. From many studies conducted overseas, it has been found that even low levels of exposure also increase the risk of lung cancer and kidney disease. We measured the levels of exposure of construction workers, mainly when cutting or grinding concrete. We found that a sizeable proportion of construction workers did have exposure that was hazardous, and recommend that dust levels need to be reduced to prevent disease.

Physician advocacy in western medicine: a 21st century challenge

Philip Bagshaw, Pauline Barnett

Some doctors believe it is a professional responsibility to speak out publicly in defence of the health of patients and communities. Others think they should only give their views privately when asked by health authorities. With many healthcare systems in Western countries under increasing stress, with underfunding and growing unmet need for care, it is important that these divergent views on such professional responsibilities are quickly resolved. In the UK, where the National Health Service is in serious trouble, medical representative organisations are increasingly speaking openly about the problems. Will such organisations in other Western countries do the same, as-and-when their healthcare systems are similarly threatened?

Asbestos—worker exposure, family disease

William Ivan Glass, Helen Clayson

Mesothelioma is a fatal disease resulting from asbestos exposure; such exposure usually occurs at work, however if the asbestos fibres are carried to the home from the workplace on the hair, clothes or boots of the worker, then family members in close contact with the worker can be exposed and may develop the disease. Because the link between work and home is not clear, the family member—usually a female—develops mesothelioma unexpectedly. As the family member did not develop the disease at work, ACC compensation is not available.

New Zealand asthma guidelines updated

Richard Beasley, Robert J Hancox

The publication of the *Asthma & Respiratory Foundation New Zealand child and adolescent asthma guidelines: a quick reference guide* in the New Zealand Medical Journal today¹ provides a much needed update of the New Zealand Paediatric Society's guidelines published in 2005.² The guidelines also complement the New Zealand adult asthma guidelines published in the New Zealand Medical Journal 12 months ago,³ which replaced those published by the New Zealand Guidelines Group in 2002.⁴ Together, these new guidelines provide simple and practical evidence-based recommendations for the diagnosis, assessment and management of asthma. It is worthwhile highlighting the similarities and differences between the adult and the child and adolescent guidelines, and what major changes have been made since the previous versions over a decade ago.

Diagnosis

Both recent guidelines emphasise that there is no single reliable 'gold standard' diagnostic test for asthma, and that the diagnosis of asthma is based on the recognition of characteristic patterns of symptoms and signs, which increase or decrease the probability of asthma. Key components of this probability-based approach are consideration of the response to treatment, and of alternative diagnoses, which may present in a similar way.

Asthma severity, control and future risk

Both guidelines have added the assessment of risk of future severe exacerbations as an important component of the clinical assessment, in addition to the evaluation of asthma severity and level of control. This is a practical way to identify patients within primary or secondary care, in whom more intensive management is warranted. Simple markers of this risk

include measures of healthcare use such as a recent ED visit or hospital admission, repeat courses of oral steroids, frequent prescriptions for beta agonist inhalers, psychosocial problems and socioeconomic disadvantage.

Inhaled corticosteroid (ICS) regimens

Updated recommendations include when to initiate ICS treatment and preferred dosing regimens. In children, it is recommended to introduce ICS for those who have symptoms more than twice per week as previously, whereas for adults the previous threshold of symptoms or beta agonist use daily has been changed to symptoms at least twice per week, thereby aligning more closely to the childhood recommendations. In both age groups, a severe exacerbation in the previous 12 months is also an indication for ICS therapy. These evidence-based recommendations are tempered by the realisation that compliance is likely to be poor among those with infrequent symptoms. Importantly, the starting dose of ICS should be 100µg per day of fluticasone propionate (or equivalent) for children and 200µg per day (or equivalent) for adults. These recommendations reflect the evidence that there is no additional benefit from starting ICS therapy at higher doses.⁵ The lower doses for children are based on the different dose-response relationship of ICS compared with adults.^{6,7}

Stepwise approach to management

Central to the guidelines is the stepwise approach to pharmacological treatment in which patients step up treatment to achieve control and reduce the risk of exacerbation, and then step down after a period of prolonged control to find and maintain the lowest required step. This feature has been maintained from previous versions of the asthma guidelines and is a key feature of most national and international guidelines.

ICS/LABA therapy

Both guidelines now recommend changing to ICS/LABA treatment in patients not controlled on initial doses of ICS, rather than increasing the ICS dose as previously recommended. Two regimens are suggested: a fixed dose ICS/LABA with SABA for relief, or the SMART regimen in which a combination ICS/fast-onset LABA inhaler is used for both maintenance and reliever therapy. The adult guidelines recommend that the SMART regimen is preferred for patients at risk of severe exacerbations because it is more effective at reducing severe exacerbations than fixed dose ICS/LABA with SABA reliever therapy.^{8,9} The SMART regimen is not currently approved for use in children under 12 years in New Zealand so the children's guidelines primarily recommend fixed dose ICS/LABA treatment, with the SMART regimen as an option for older children. Since the SMART regimen is known to be more effective than fixed dose ICS/LABA regimens in children aged 4–17 years,^{10,11} this may be an area where adolescents could follow the adult guidelines. A major change from the previous guidelines is that separate ICS and LABAs inhalers are no longer recommended, due to the risks of LABA monotherapy in patients who are non-compliant with ICS.¹²

Action plans

As before, both guidelines recommend that children and adults with asthma should be provided with asthma self-management plans. Prescribers (and their patients) will be relieved that they no longer have to use one version for all patients, and there are now four prototype plans available from the Asthma & Respiratory Foundation website (www.asthmafoundation.org.nz). This allows prescribers to select a plan which best suits their patient's needs and medication regimen.

Acute severe asthma

Similar algorithms are provided for the management of acute severe asthma in childhood/ adolescence and in adults, allowing for standardisation of the assessment and treatment of asthma across different ages. Key features include the assessment of severity on which the initial treatment is based, and repeat assessments of the response to treatment made over the

following 60 minutes, to determine likely requirement for referral or admission to hospital.

Non-pharmacological measures

The child and adolescent guidelines place major emphasis on addressing other ways to help children with asthma. A top 10 checklist is provided, including issues such as tobacco smoking and other environmental exposures, poor housing, socioeconomic disadvantage, health literacy, access and continuity of care. These are undoubtedly important issues which also apply to adult asthma and need to be addressed if better outcomes are to be achieved and inequities reduced.

Treatable traits

A novel feature of the adult guidelines is the concept of treatable traits, which recognises that poor respiratory health in a person with asthma may not necessarily be due to their asthma, particularly if they are receiving 'optimal treatment', but may be due to some other reason, which requires investigation and treatment in its own right.^{13,14} Treatable traits can be broadly characterised into overlapping disorders such as COPD, bronchiectasis and vocal cord dysfunction, comorbidities such as chronic rhinosinusitis, depression and anxiety, environmental factors such as tobacco smoke and occupational exposures, and treatment factors such as poor adherence or poor inhaler technique. It would be reasonable to suggest that treatable traits are also an important consideration in the management of children and adolescents with asthma who have persistent symptoms despite optimal pharmacological treatment.

The authors of the child and adolescent asthma guidelines should be congratulated on their practical and evidence-based guidelines. The consistency and standardisation of the recommendations with the adult guidelines should allow a smooth transition in the management of patients as they progress from childhood to adolescence and then adult life. After a long wait, we now have guidelines for New Zealand children, adolescents and adults that are both up-to-date and fit-for-purpose. The challenge is now is to get their recommendations translated into clinical practice.

Competing interests:

Nil.

Author information:

Richard Beasley, Medical Research Institute of New Zealand, Wellington, Capital & Coast District Health Board, Wellington; Robert J Hancox, Department of Preventive & Social Medicine, University of Otago, Dunedin.

Corresponding author:

Professor Richard Beasley, Medical Research Institute of New Zealand, Private Bag 7902, Wellington 6242.

richard.beasley@mrinz.ac.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7424>

REFERENCES:

1. Asher I, McNamara D, Davies C, et al. Asthma & Respiratory Foundation New Zealand child and adolescent asthma guidelines: a quick reference guide. *N Z Med J.* 2017; 130(1466):10–33.
2. Paediatric Society of New Zealand. Best Practice Evidence-Based Guideline: Management of asthma in children aged 1–15 years. 2005. www.paediatrics.org.nz
3. Beasley R, Hancox R, Harwood M, et al. Asthma and Respiratory Foundation NZ adult asthma guidelines: a quick reference guide. *N Z Med J.* 2016; 129(1445):83–102.
4. New Zealand Guidelines Group. Best Practice Evidence-Based Guideline: The diagnosis and treatment of adult asthma. September 2002.
5. Powell H, Gibson P. High dose versus low dose inhaled corticosteroids as initial starting dose for asthma in adults and children. *The Cochrane Collaboration. The Cochrane Library* 2008; Issue 4.
6. Holt S, Suder A, Weatherall M, et al. Dose-response relation of inhaled fluticasone propionate in adolescents and adults with asthma: meta-analysis. *BMJ.* 2001; 323:253–6.
7. Masoli M, Weatherall M, Holt S, Beasley R. Systematic review of the dose-response relation of inhaled fluticasone propionate. *Arch Dis Child.* 2004; 89:902–7.
8. Kew KM, Karner C, Mindus SM, Ferrara G. Combination formoterol and budesonide as maintenance and reliever therapy versus combination inhaler maintenance for chronic asthma in adults and children. *Cochrane Database Syst Rev* 2013;CD009019.
9. Patel M, Pilcher J, Pritchard A, et al. Efficacy and safety of maintenance and reliever combination budesonide–formoterol inhaler in patients with asthma at risk of severe exacerbations: a randomised controlled trial. *Lancet Respir Med.* 2013; 1:32–42.
10. Bisgaard H, Le Roux P, Bjamer D, et al. Budesonide/formoterol maintenance plus reliever therapy: a new strategy in pediatric asthma. *Chest.* 2006; 130:1733–43.
11. Edwards SJ, von Maltzahn R, Naya IP, Harrison T. Budesonide/formoterol for maintenance and reliever therapy of asthma: a meta-analysis of randomised controlled trials. *Int J Clin Pract.* 2010; 64:619–27.
12. Beasley R, Perrin K, Weatherall M, Wijesinghe M. Call for withdrawal of LABA single-therapy inhaler in asthma. *Lancet.* 2010; 376:750–1.
13. Agusti A, Bel E, Thomas M, et al. Treatable traits: toward precision medicine of chronic airway diseases. *Eur Respir J.* 2016; 47:410–9.
14. Agusti A, Bafadhel M, Beasley R, et al. Precision medicine in airway diseases: moving to clinical practice. *Eur Respir J.* 2017; 50:doi.org/10.1183/13993003.01655-2017.

Asthma and Respiratory Foundation NZ child and adolescent asthma guidelines: a quick reference guide

Innes Asher, David McNamara, Cheryl Davies, Teresa Demetriou,
Theresa Fleming, Matire Harwood, Lorraine Hetaraka-Stevens,
Tristram Ingham, John Kristiansen, Jim Reid, Debbie Rickard, Debbie Ryan

ABSTRACT

The purpose of the *New Zealand Child and adolescent asthma guidelines: a quick reference guide* is to provide simple, practical, evidence-based recommendations for the diagnosis, assessment and management of asthma in children and adolescents in New Zealand, with the aim of improving outcomes and reducing inequities. The intended users are health professionals responsible for delivering asthma care in the community and hospital emergency department settings, and those responsible for the training of such health professionals.

Abbreviations:

FEV ₁	Forced expiratory volume in one second
ICS	Inhaled corticosteroid
LABA	Long-acting beta-agonist
MDI	Metered dose inhaler
PEF	Peak expiratory flow
SABA	Short-acting beta-agonist
SpO ₂	Oxygen saturation

Inequities in New Zealand

Despite advances in knowledge about asthma and its management, we could be doing better. We believe that all children in New Zealand have the right to achieve the highest standard of asthma outcomes equally. In New Zealand, a large number of children are not faring well with their asthma, especially due to disadvantages that arise from inadequate income for the basics needed for wellbeing, and unhealthy indoor environments (homes which are crowded, cold, damp, mouldy,¹ smoke-exposed or with unflued gas heating²). Children aged 13–17

years usually do not have free primary healthcare visits or prescriptions. Māori and Pacific children with asthma are more likely to have severe asthma symptoms and be hospitalised, but are less likely to be prescribed inhaled corticosteroid (ICS), have an action plan or receive adequate education (see “*Māori—getting it right for Māori children with asthma*” and “*Pacific peoples—getting it right for Pacific children with asthma*”). Other groups who experience inequities include refugees, people living in remote rural areas and people with low English language proficiency. All health

professionals have a role in improving outcomes and reducing inequities, and these guidelines specify the actions required regarding asthma.

New reports to inform us

Three important reports were released by the Asthma and Respiratory Foundation of New Zealand in 2015: *The Impact of Respiratory Disease in New Zealand: 2014 Update*,³ *He Māramatanga Huangō: Asthma Health Literacy for Māori Children in New Zealand*⁴ and *Te Hā Ora: The National Respiratory Strategy*.⁵ In 2017 *The Impact of Respiratory Disease in New Zealand* report was updated. These reports describe the context of the growing impact of asthma in New Zealand, especially among children, the inequities suffered by Māori, Pacific peoples and low-income families, and the intersectorial and holistic approaches needed to tackle the issues.

Other guidelines consulted

This guide is a complete update of the outdated Paediatric Society of New Zealand 2005 *Management of Asthma in Children aged 1–15 years*. The following guidelines were reviewed in the preparation of this document: the National Asthma Council of Australia 2015 *Australian Asthma Handbook* version 1.1, including the companion *Quick Reference Guide*,⁶ the Global Initiative for Asthma 2016 *Asthma Management and Prevention*, including the companion *Pocket Guide*,⁷ the SIGN 2016 *British Guidelines on the Management of Asthma*, including the *Quick Reference Guide*,⁸ and the Asthma and Respiratory Foundation NZ *Adult Asthma Guidelines: A Quick Reference Guide*.⁹

A systematic review was not performed, although relevant references were reviewed as required to formulate this guideline, and to clarify differences in recommendations made between guidelines. Readers are referred to the above published guidelines and handbooks for the more comprehensive detail and references that they provide. Additional analyses and reviews on the assessment and management of preschool wheezing were consulted.^{10–12}

Grading

No levels of evidence grades are provided here due to the format of this quick reference guide. Readers are referred to the above published guidelines and handbooks for the level of evidence for the recommendations on which the *Child and Adolescent Asthma Guidelines: A Quick Reference Guide* are based.

Age

Adolescents

These guidelines apply to children 15 years and below. However, adolescents mature at different rates, and for many who are still maturing and require adult support with their asthma care, these guidelines will usually apply. Once adolescents are largely responsible for their own management, application of the recently published *Adult Asthma Guidelines: A Quick Reference Guide*, intended for those 16 years and over, becomes more appropriate. Special care is needed to ensure that the adolescent transitions in a developmentally appropriate way as they become more independent, make their own decisions and emerge as adults. Adolescents transitioning from family to self-management may have differing priorities.

Children aged under five years

There are special considerations in young children (1–4 years) who wheeze, as many of them do not go on to develop asthma (see *Diagnosis*).

Guideline development

The Guideline Development Group included representatives from a range of professions, disciplines and backgrounds relevant to the scope of the guidelines. A *Draft for Consultation* of this report was peer-reviewed by a wide range of respiratory health experts and key professional organisations (see Appendix C).

The guidelines are primarily presented through lists, tables and figures in an electronic format, which can be used in clinical practice. Key references are provided where necessary to support recommendations that may differ from previous or other guidelines, or standard clinical practice.

Dissemination plan

The guidelines will be translated into tools for practical use by health professionals, and used to update existing consumer resources. They will be published on the Asthma and Respiratory Foundation NZ website, and disseminated widely via a range of publications, training opportunities and other communication channels to health professionals, nursing and medical schools, primary health organisations and district health boards.

Implementation

The implementation of the *Child and Adolescent Guidelines: A Quick Reference Guide* by organisations will require communication, education and training strategies.

Expiry date

The expiry date of the guide is 2022.

Health professional to 10 actions These are the top 10 ways health professionals can help (apart from prescribing medicines)

1. Relationships

Encourage the continuity of care with doctors and nurses in your practice and secondary care, and make follow-up appointments—relationships help. Easy access to a trusted nurse and telephone follow-up is recommended.

2. Wellness

Work with families to attain and maintain wellness, and not accept sickness as the norm.

3. Smoke exposure

Ask about smoke exposure, encourage reducing tobacco smoke exposure in the child's environment (home and car) and recommend smoking cessation. If appropriate, give advice and refer to a local smoking cessation service or Quitline (0800 778 778). Provide Health Sponsorship Council's pamphlet *A Guide to Making Your Home and Car Smokefree* (www.healthed.govt.nz/).

4. Housing

A lot of New Zealanders live in unhealthy housing, and conditions are worse in private rental housing. Some families are homeless. Therefore ask about housing and unhealthy features (crowding, cold, damp, mouldy, unflued gas heater). (<http://www.asthmafoundation.org.nz/about-us/advocacy/national-respiratory-strategy>; <http://www.energywise.govt.nz/>). Provide the family with information about having a healthy home ("Tips for healthy living" <http://www.asthmafoundation.org.nz/your-health/healthy-living>) and if relevant, refer for healthy housing assessment if available in your region.

5. Income

Assume that most families struggle with income and ask about it. Inquire about the ability to access the doctor, the pharmacy and paying for prescriptions. Does the

child have partly or uncontrolled persistent asthma and meet criteria for Child Disability Allowance?¹³ (<http://www.workandincome.govt.nz/>). It is important for all family members to use the same pharmacy because once patients and their families have collected 20 new prescription items in a year, they won't have to pay any more prescription charges until 1 February the following year (<http://www.health.govt.nz/your-health/conditions-and-treatments/treatments-and-surgery/medications/prescription-charges>).

6. Health literacy

Assume little health literacy, and use steps described in *He Māramatanga Huangō: Asthma Health Literacy for Māori Children in New Zealand*. Specifically ask the child and whānau what they understand, what they want to know, and use simple language to explain about asthma. For example, use the term 'asthma flare-up' rather than 'asthma exacerbation'.

7. Adherence

Firstly, assume inhaler device technique is poor and check it. Secondly, assume adherence is imperfect and don't judge. Ask questions in an open way, such as "Many people take less preventer than the doctor prescribes—about how many times a week do you take your asthma preventer?"¹⁴⁻¹⁶

8. Asthma action plan

Develop an appropriate asthma action plan with the child and family and check on each visit. Plans should be made available to schools and child care facilities where appropriate. (<http://www.asthmafoundation.org.nz/resources>).

9. Access

Help the family to understand how to access care appropriate to asthma severity, and identify any barriers they have. Consider referral to asthma educator, Māori providers or paediatrician where available and appropriate.

10. Ambulance

Ensure the family know when and how to call an ambulance. In some regions this service may incur a charge.

Diagnosis

Goal: All children who have asthma are promptly and correctly diagnosed

Approach to diagnosis

- The diagnosis of asthma is based on the recognition of a characteristic pattern of symptoms and signs (Table 1) and response to treatment, in the absence of an alternative explanation.
- Initial diagnosis is probability-based and should always be reconsidered if the patient fails to respond to therapy, or has atypical symptoms or signs.
- The key to making the diagnosis of asthma is to take a careful clinical history and assess clinical +/- spirometry response to inhaled bronchodilator and/or ICS treatment. There is no reliable single 'gold standard' diagnostic test.
- The diagnosis and monitoring of asthma requires frequent and repeated review. This may require the use of recall or follow-up systems (Figure 2).
- Algorithms to guide the diagnosis in 1–4 year olds (Figure 1A) and 5–15 year olds (Figure 1B).

Practice points

- During a trial of therapy, give the patient a label of 'suspected asthma' as a means of communicating with other health professionals.
- In most children, observing a symptomatic response to treatment may help to confirm the diagnosis, but a limited response to bronchodilator or ICS does not rule out asthma.
- In children with a high probability of asthma, start a trial of treatment (see Figures 4 and 5) and assess the response to therapy.
- In children with a low probability of asthma, perform further investigations, such as chest x-ray and/or specialist referral prior to initiating preventer therapy.
- Spirometry may be helpful in older children (≥ 12 years or six years and above if paediatric-trained technician).
- In New Zealand, bronchiectasis should be considered for all children with asthma symptoms at any age. Sometimes bronchiectasis co-exists

with asthma, and can be missed on a chest x-ray. Chronic wet cough is a key marker.

Children 1–4 years of age

- Young children 1–4 years are a special group, as about half of those who wheeze do not have asthma at school age and later.
- They are managed according to three patterns of symptoms and labelled accordingly to assist making decisions about prescribing ICS.
- In each of these groups, a bronchodilator should be prescribed as for asthma, according to clinical severity (see Figure 4).

Infrequent preschool wheeze

For those with infrequent symptoms, or who wheeze only with viral illnesses, ICS are not indicated. An alternative term sometimes used is 'infrequent episodic (viral) wheeze'.

Frequent preschool wheeze

For those with frequent episodes of wheeze (more than every 6–8 weeks), only with viral illnesses, but no symptoms in the interval between—give a trial of ICS for a minimum of eight weeks. If there is a positive response, these children should then be labelled as 'preschool asthma', if not, the treatment should be stopped and the child should remain labelled as 'frequent preschool wheeze'. An alternative term sometimes used is 'frequent episodic (viral) wheeze'.

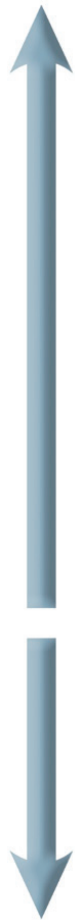
Preschool asthma

Those with frequent symptoms typical for asthma during and in the interval between viral illnesses. Treat as for asthma and give a trial of ICS (preferred) or montelukast, and the same treatment is indicated if there are severe attacks (see Figure 6). An alternative term sometimes used for this pattern is 'multi-trigger wheeze'. This label does NOT mean the child will go on to have asthma at school age or as an adult, which may be reassuring for many families.

Wheezing in children under one year

In children under one year, bronchiolitis is the most common cause of wheezing, and the PREDICT Australasian Bronchiolitis Clinical Practice Guideline¹⁷ should be followed. If the illness does not seem to be bronchiolitis, then refer to Table 1 and Figure 1A for guidance.

Table 1: Clinical features that increase or decrease the probability of asthma in children and adolescents.



<p>A. Asthma more likely</p> <ul style="list-style-type: none"> • More than one of the following: <ul style="list-style-type: none"> - Wheeze (most sensitive and specific symptom of asthma) - Breathlessness - Chest tightness - Cough • Particularly if: <ul style="list-style-type: none"> - Typically worse at night or in the early morning - Provoked by exercise, cold air, allergen exposure, irritants, viral infections, stress and aspirin - Recurrent or seasonal • Personal history of atopic disorder or family history of asthma • Widespread wheeze heard on chest auscultation • Otherwise unexplained expiratory airflow obstruction on spirometry • Otherwise unexplained blood eosinophilia or raised exhaled nitric oxide • Bronchial hyper-responsiveness on challenge testing at appropriate age • Positive response to bronchodilator (clinical or lung function) <p>B. Asthma less likely</p> <ul style="list-style-type: none"> • Isolated cough in absence of wheeze or difficulty breathing • History of wet, moist or productive cough—consider alternative diagnosis • No wheeze or repeatedly normal physical examination when symptomatic • Normal spirometry or peak flow (PEF) when symptomatic • No response to trial of asthma treatment • Features that point to an alternative diagnosis (see C below) <p>C. Red flags suggesting alternate diagnoses*</p> <ul style="list-style-type: none"> • Daily symptoms from birth • Frequent or daily wet, moist-sounding or productive cough • Digital clubbing • Chest wall deformity • Failure to thrive • Heart murmur • Spilling, vomiting or choking • Asymmetrical chest findings • Stridor as well as wheeze • Persistent ear, nose or sinus infection • Family history of unusual chest disease • Symptoms much worse than objective signs or spirometry
--

*Consider aspiration, bronchiectasis, ciliary dyskinesia, cystic fibrosis, developmental airway anomaly, foreign body aspiration, heart disease, hyperventilation, immunodeficiency, tuberculosis, vocal cord dysfunction.

Figure 1A: Diagnostic pathway for asthma and wheeze in children 1–4 years.^{6,8}

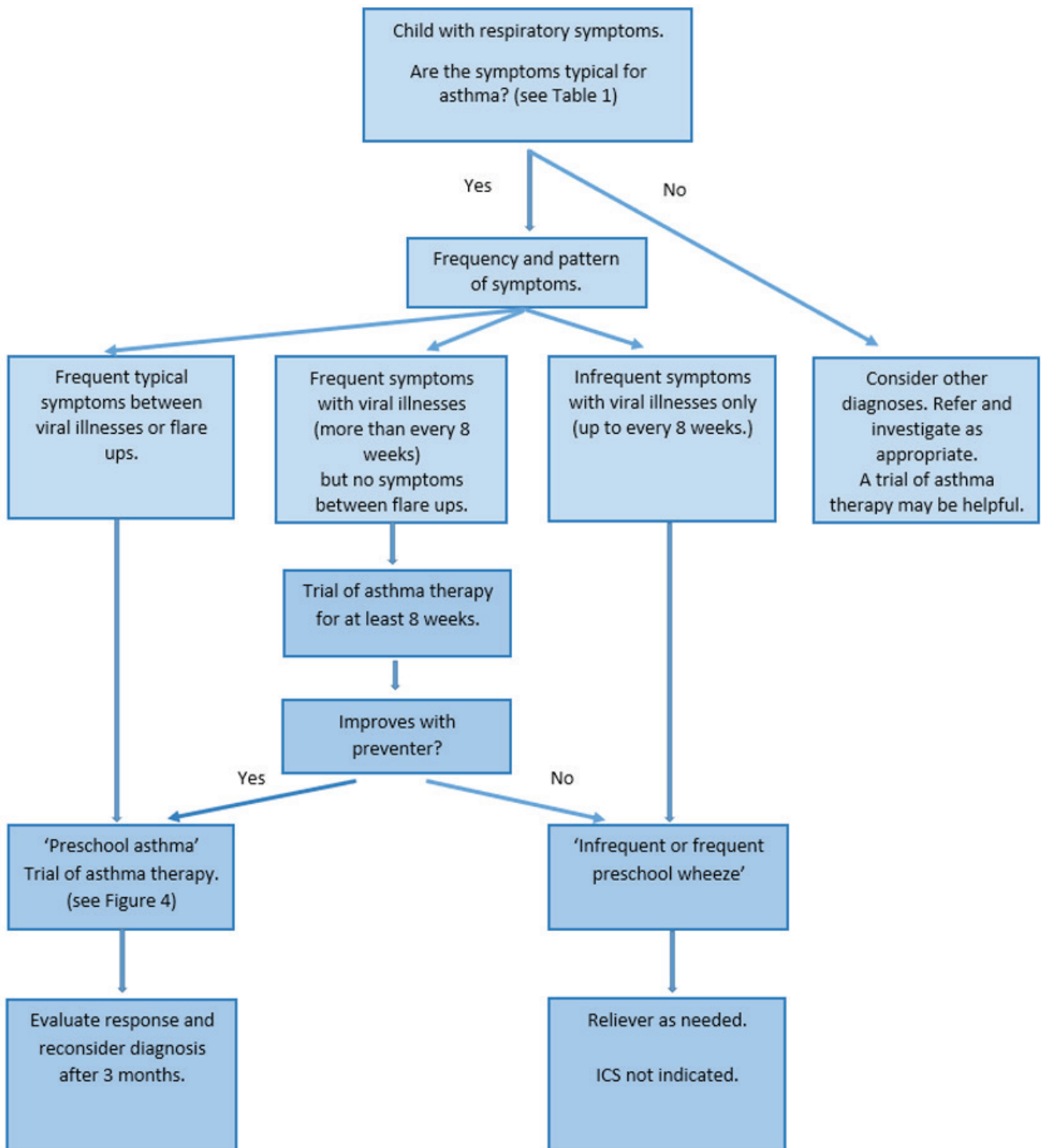
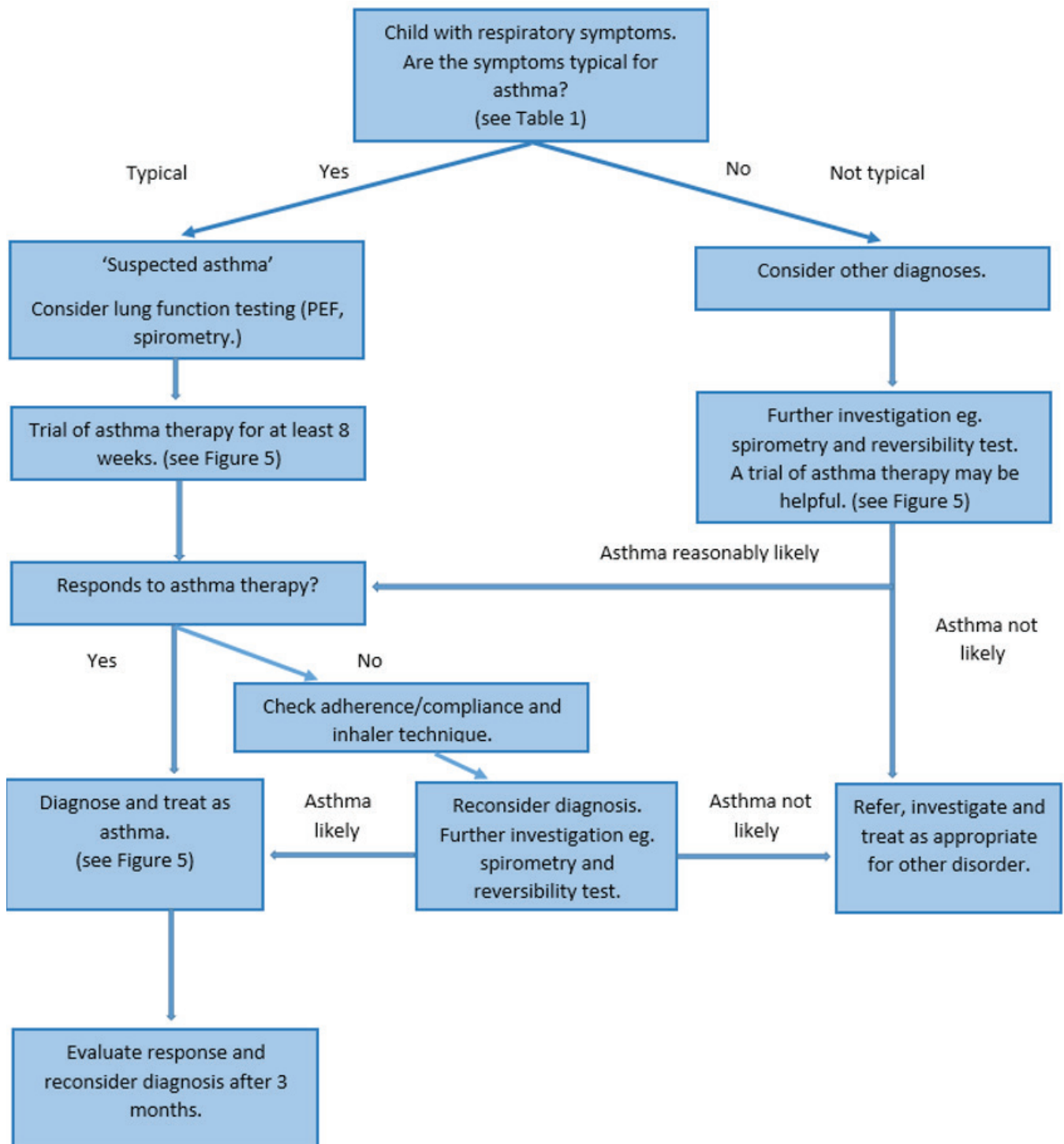


Figure 1B: Diagnostic pathway for asthma and wheeze in children 5–15 years.^{18,19}



Assessing asthma severity, control and future risk


Goal: All children with asthma are assessed for their severity, control and future risk

Evaluation of asthma control and severity

- Evaluation of asthma severity, the level of control and the risk of future events are all-important components of the assessment of children with asthma.
- Asthma control is defined by the frequency of symptoms, the degree to which symptoms affect sleep and activity, and the need for reliever medication.

- Poor asthma control is defined as regular symptoms occurring in a usual week that affect the patient’s quality of life, or according to the asthma symptom control measures below.
 - Poor control should trigger a review of adherence, inhaler technique and preventer therapy.
 - If poor control persists, then reconsider the diagnosis.
 - If poor control persists despite above, then consider increasing the asthma treatment step.
 - The level of asthma control should be assessed regularly. Two methods for assessing asthma symptom control are the Asthma Control Test and the GINA Assessment.
1. Asthma Control Test for children 4–11 years (below; Adult guideline for ≥12 years⁹).

Is your child’s (4-11yrs) asthma under control?



Asthma Control Test™

The first step to achieving control over your child’s asthma is to know where they’re at right now. This test is a way of assessing your child’s present level of asthma control.^{1,2} It will provide a score that may help your health care professional determine if your child’s asthma treatment plan is working or if it might be time for a change. Take five minutes now and do this simple 3 step test with your child.

STEP 1 Let your child answer these questions. You may help, but let your child select the response.

Q1 How is your asthma today? SCORE

0 Vary Bad 1 Bad 2 Good 3 Vary Good

Q2 How much of a problem is your asthma when you run, exercise or play sports?

0 It’s a big problem, I can’t do what I want to 1 It’s a problem and I don’t like it 2 It’s a little problem but it’s ok 3 It’s not a problem

Q3 Do you cough because of your asthma?

0 Yes, all of the time 1 Yes, most of the time 2 Yes, some of the time 3 No, none of the time

Q4 Do you wake up at night because of your asthma?

0 Yes, all of the time 1 Yes, most of the time 2 Yes, some of the time 3 No, none of the time

STEP 1 SUBTOTAL

Continue the test over

STEP 2 Complete these questions on your own

Q5 During the last 4 weeks, how many days did your child have any daytime asthma symptoms? SCORE

Not at all 1-3 days 4-10 days 11-18 days 19-24 days Everyday

5 4 3 2 1 0

Q6 During the last 4 weeks, how many days did your child wheeze during the day because of asthma?

Not at all 1-3 days 4-10 days 11-18 days 19-24 days Everyday

5 4 3 2 1 0

Q7 During the last 4 weeks, how many days did your child wake up during the night because of asthma?

Not at all 1-3 days 4-10 days 11-18 days 19-24 days Everyday

5 4 3 2 1 0

STEP 2 SUBTOTAL

STEP 3 Add step 2 subtotal to step 1 subtotal (from the front) to get the final score

STEP 1 SUBTOTAL + **STEP 2 SUBTOTAL** = **TOTAL**

What does your child’s Asthma Control Test™ result mean?
Your child’s test result is an assessment of their level of asthma control.¹

SCORE: 20 or more Your child’s asthma appears to be controlled?²
Even so, it can change over time so it’s important to retest your child regularly. Continue to talk to your health care professional about their asthma control.

SCORE: 19 or less Your child’s asthma may be uncontrolled or only partly controlled.²
Make an appointment to discuss your child’s asthma score with their health care professional.*

Modified US version for use in New Zealand. This does not replace a full assessment from your Doctor. Asthma Control Test™ copyright, QualityMetric Incorporated 2002, 2004. All Rights Reserved. Asthma Control Test™ is a trade mark of QualityMetric Incorporated. Asthma Control Test is distributed by GlaxoSmithKline NZ Limited, Auckland.

References: 1. Liu A et al. *J Allergy Clin Immunol*. 2007;119:817-825
2. Koolen BB et al. *Eur Respir J*. 2011;38:561-566. TAPS NAB093/16FE/AST/0006

*Please note that normal doctor fees will apply.

¹The C-ACT contact information and permission to use: Mapi Research Trust, Lyon, France. Internet: <https://eprovide.mapi-trust.org/>

Table 2: GINA assessment of asthma symptom control in people six years and over (1) (See Table 3).

A. Asthma symptom control		Level of asthma symptom control		
In the past four weeks, has the patient had:		Well controlled	Partly controlled	Uncontrolled
• Daytime asthma symptoms more than twice/week?	Yes <input type="checkbox"/> No <input type="checkbox"/>	None of these	1–2 of these	3–4 of these
• Any night waking due to asthma?	Yes <input type="checkbox"/> No <input type="checkbox"/>			
• Reliever needed for symptoms* more than twice/week?	Yes <input type="checkbox"/> No <input type="checkbox"/>			
• Any activity limitation due to asthma?	Yes <input type="checkbox"/> No <input type="checkbox"/>			

*Excludes reliever taken before exercise.
 (GINA recommends assessment of risk factors as an essential part of the assessment of asthma control).
 (Modified with permission of GINA).

- The GINA yes/no questions about the four criteria in Table 2 above, regardless of current treatment regimen.⁷

Practice points—severity and future risk

- Assessment of asthma also involves risk of adverse outcomes, including severe exacerbations, deaths and treatment-related adverse effects (Table 3).
- Severity of asthma is defined by the treatment step (Figures 4 and 5) needed to maintain good control. Work with patient/parent to determine what good control looks like.⁶

- For symptomatic children, asthma severity can be determined only after a therapeutic trial of ICS for at least eight weeks (Figures 4 and 5). Start the therapeutic trial and book the follow-up appointment for eight weeks later.
- The best predictor of future asthma attacks is the number of exacerbations in the last 12 months.
- Growth (height and weight) should be measured at least annually in children with asthma, and plotted on a percentile chart. Fall-off on percentiles suggests poor asthma control; other causes include malnutrition, frequent oral corticosteroids or after initiation of higher dose ICS.

Table 3: Features associated with increased risk of severe asthma exacerbations and/or death from asthma.²⁰

<p>A. Asthma</p> <ul style="list-style-type: none"> Poor asthma control Hospitalisation or emergency department visit for asthma in the last year Extreme inhaled bronchodilator use (>1 canister per month) History of sudden asthma attacks Intensive care admission or intubation (ever) Requirement for long-term oral steroids <p>B. Comorbidity</p> <ul style="list-style-type: none"> Major psychosocial problems Alcohol and drug abuse in family Severe food allergy and anaphylaxis <p>C. Other factors</p> <ul style="list-style-type: none"> Poor inhaler technique Underuse or poor adherence to ICS treatment Tobacco smoke exposure Discontinuous medical care Socioeconomic disadvantage Financial hardship Unhealthy housing Māori and Pacific ethnicity Child protection issues (consider Vulnerable Children Act 2014). www.legislation.govt.nz/act/public/2014/0040/latest/DLM5501618.html
--

- Increase in weight may reflect inappropriate diet and steroid dose.
- Monitor healthcare use. Children and adolescents with high healthcare use (such as hospital admissions, emergency department visits, emergency doctor visits and unplanned doctor visits) are at high risk for severe or life-threatening asthma.
- Monitor medicine use. Children and adolescents with high medication requirements or usage (such as courses of oral steroids, frequency of beta-agonist prescriptions and more prescriptions for beta-agonists than ICS) are at high risk for severe or life-threatening asthma.
- Psychosocial stressors are potent triggers of asthma symptoms. Identification of these triggers and the introduction of very simple strategies, such as slow, relaxed breathing when stressed, may help the patient and whānau in managing symptoms.
- If anxiety or panic play a part, involve the family to support the patient and consider referral for psychological counselling.
- Dysfunctional breathing or a breathing pattern disorder can be a contributing factor in the severity of asthma. A physiotherapist can advise on breathing awareness and exercises to help relaxation and improve effectiveness of breathing.
- Keeping the nose clear will help asthma control, as it filters, warms and humidifies the air to the lungs. Saline drops and frequent blowing are usually adequate.
- Asthma control may be improved by better insulation and avoiding cold, damp, mouldy or crowded housing.
- Unflued gas heaters may make asthma symptoms worse (<http://www.health.govt.nz/your-health/healthy-living/environmental-health/household-items-and-electronics/unflued-gas-heaters>).
- House dust mite avoidance measures are not effective³⁸ unless the child has symptoms that are clearly triggered on exposure to dust mite allergens.
- A healthy diet is wise, but other modifications to diet are unlikely to improve asthma control unless food allergy is confirmed.

Management approaches

Identifying management goals with the child & whānau

Goal: The child and family participate in goal-setting

- Managing asthma requires a partnership between the child, their parents, their whānau and their healthcare team. This will change and develop as children age and involves patient willingness and understanding, agreeing on management goals.
- Management and partnership are based on a cycle of repeated assessment, adjustment of treatment and review of responses, as outlined in Figure 2.

Non-pharmacological measures

Goal: Personal, whānau or environmental factors which may be unsettling asthma are identified and addressed (see *Health professional to 10 actions* section)

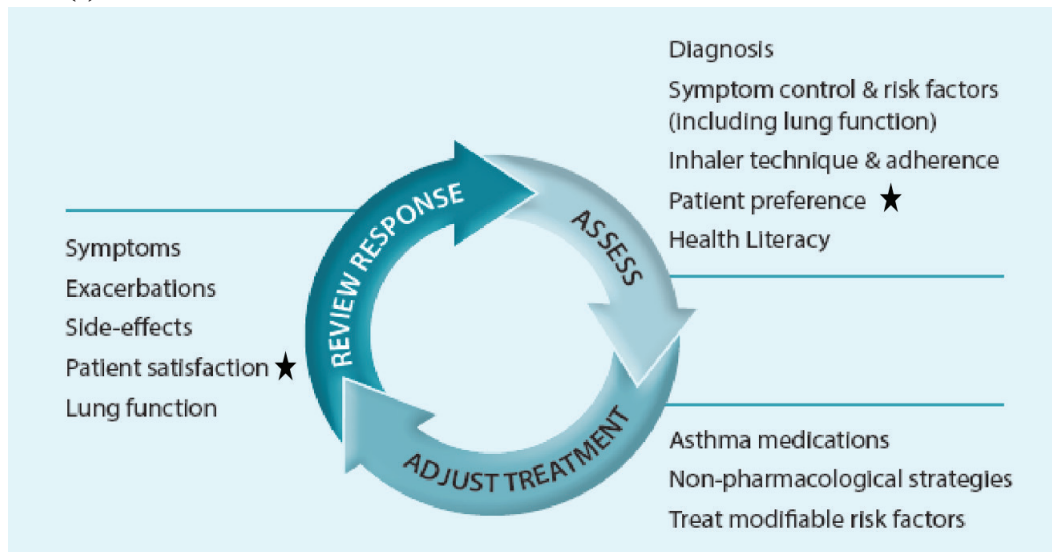
- To improve asthma outcomes, avoid smoke exposure and known triggers.
- Avoid triggers which are known to provoke or precipitate asthma attacks (except exercise) or anaphylaxis.
- Exercise and physical activity should be encouraged, as exercise-induced asthma can be managed. Chlorinated swimming pools may be a trigger for some children.

Self-management

Goal: Effective self/family education and management is achieved

- Asthma education and improving health literacy and self-efficacy are fundamental in asthma management, and are the responsibility of all health professionals.
- All patients with asthma and their caregivers should be offered management education, which should include a written personalised asthma

Figure 2: Asthma management as a continuous cycle of monitoring and reassessment, adapted from GINA (1).



*(patient or parent).

- action plan. Ask the patient/parent how best to achieve this.^{6,8}
- Adherence to treatment should be routinely assessed and encouragement provided as part of the self-management education. The health professional should gain an understanding of why the patient/parent is doing anything different^{7,8}
- Asthma management in all contexts needs attention, including child care and school environments, and support of teachers.

Practice points—enhancing self-management

- Asthma education should increase health knowledge about asthma, general health literacy and self-efficacy, and should be reinforced every visit.
- Teach families to recognise when asthma is poorly controlled, know when and how to call emergency services.
- Asthma education should utilise a variety of media, including printed materials as well as verbal explanations, and printed materials in the first language if possible, eg, www.pamp.co.nz, which produces simple, individualised pictorial asthma medication plans in Te Reo Māori, Samoan, Tongan, Tuvaluan and Chinese.²²

- Education should be delivered in chunks and delivered across multiple visits instead of all at once.
- Education should be developmentally appropriate. As children mature, offer further information and coach to take increasing responsibility for their care.
- Inhaler technique should be routinely assessed wherever possible and training provided as part of self-management education.

Asthma action plans

Goal: All children with asthma are provided with an asthma action plan

- To assist in self-management of childhood asthma, consider all of the child’s regular caregivers and environments in preparing and distributing the action plan.
- Asthma action plans that are symptom-based, rather than PEF-based, are preferred in children, although some older children may want a PEF-based plan.
- Child asthma action plans from the Asthma and Respiratory Foundation NZ can be downloaded from <http://www.asthmafoundation.org.nz/> resources
- The *Child Asthma Action Plan* should be written and reviewed with the

family/caregivers. It must be individualised for the child and culturally appropriate (see Appendix A).

- A *Child Asthma Symptom Diary* may be used to clarify the pattern of symptoms and response to treatment, to guide the Action Plan (see Appendix B).

Practice points—asthma action plans

- Always involve the child by using developmentally-appropriate language.
- Ensure the child (in an age appropriate manner) and the family/caregivers understand the plan.
- Keep a record of the plan and provide copies of the *Child Asthma Action Plan* for all caregivers—including child care or school.
- Arrange a formal review, at least annually, of the *Child Asthma Action Plan* with the family/caregivers. Frequency of reviews will be dependent on family/carer's confidence and competence with asthma management.
- Ensure family/caregivers understand the importance of not running out of inhalers and prescribed medication. Check that they know the process for obtaining repeat prescriptions.
- Ensure enough medications are prescribed and reinforce the need for appropriate regular clinical assessment.

Adolescents: getting it right for adolescents with asthma

Goal: Adolescents with asthma transition smoothly towards emerging adulthood, with good asthma control

- Children are normally seen accompanied by a caregiver, with caregivers taking responsibility for management, while most adults are seen on their own and assumed to be self-managing. Adolescents require an approach which is inbetween and enables them to take increasing responsibility. Ensure adolescents have a developmentally appropriate understanding of their asthma and treatment. If they have had asthma for a long time, they may not have had updated information since childhood.

- Assume adolescents are interested in WHY they should control their asthma and HOW it might benefit their own goals, such as by saying, “*good asthma control will help you get on with life*”, which may be more motivating than saying “*Aim for good control*”. Assess compliance with open and nuanced questions, such as “*When you get wheezy, what do you normally do?*” or “*Which doses do you find easiest to remember?*”
- Develop a treatment regimen; consider simple regimes. Ensure the young person is aware of what to do if symptoms escalate, and has someone to contact if they have concerns.
- Offer the chance to ask questions.
- Arrange follow-up appointments and ensure the adolescent knows how and when to instigate appointments.^{7,8}

Practice points—adolescents

- Prioritise the relationship, introduce yourself and offer continuity of care.
- Give adolescents the full consultation time. Consider if a practice nurse could play a coaching role.
- See adolescents individually first, and then with parents/caregivers as appropriate. Ensure the adolescent knows that as they age they will take more responsibility for their own healthcare and that they can make appointments for themselves.
- Explain confidentiality, which can be as simple as having health privacy information on the wall and providing a brief verbal outline, such as “*Your health information is confidential. We are not allowed to tell other people, like school or family members, unless you agree or unless there are serious safety issues.*”
- Explain risks of sharing inhalers with others (infection, inhaler runs out more quickly).
- Ask about smoking and e-cigarettes (vaping) and advise.
- Assume that the young person is likely to have other health issues and questions. Complete a brief HEADSS (Home & Environment, Education & Employment, Activities, Drugs, Sexuality, Suicide/Depression) or holistic

psychosocial assessment if practicable. Ask if they have questions about asthma, or about how they are feeling²³⁻²⁸ (http://www.health.govt.nz/system/files/documents/publications/depression_summary.pdf).

- Consider these eight key points from this recent systematic review:²⁹
 - a) Many adolescents have poor knowledge about asthma and treatments.
 - b) Non-adherence is frequently caused by forgetting to take medication.
 - c) Adolescents with established routines are better able to self-manage.
 - d) Some adolescents do not use treatments or use them incorrectly due to erroneous beliefs about their asthma and medication.
 - e) Asthma self-management is difficult for those with a lack of support at school.
 - f) Parents play a key role in reminding adolescents to take medication.
 - g) Many adolescents are embarrassed about having asthma and using medication, particularly around their friends and peers.
 - h) Many adolescents report difficulties in communicating with their healthcare professional.

Māori—getting it right for Māori children with asthma

Goal: Māori children have asthma outcomes equal to non-Māori and non-Pasifika children

Māori rights in regards to health, recognised in Te Tiriti O Waitangi and other national and international declarations, promote both Māori participation in health-related decision making, as well as equity of health outcomes for all New Zealanders. Currently, Māori with asthma are more likely to be hospitalised or die due to asthma. Despite this, Māori with asthma are less likely to be prescribed ICS, have an asthma action plan or receive adequate education. Major barriers to good asthma management for Māori may include access to care, discontinuity and poor quality care, and poor health literacy. Māori whānau have greater exposure to environmental triggers for asthma, such as smoking and poor housing.^{9,30}

It is recommended that for Māori with asthma:

- Asthma providers should undertake clinical audit or other similar quality-improvement activities to monitor and improve asthma care and outcomes for Māori.³¹
- A systematic approach to health literacy and asthma education for Māori whānau is required. The evidence of the health literacy demands, the barriers and facilitators, and steps to delivering excellent asthma management with Māori, which are described in He Māramatanga Huangō: Asthma Health Literacy for Maori Children in New Zealand (www.health.govt.nz), also apply to adults. Asthma healthcare providers should support staff to develop cultural competency skills for engaging Māori with asthma and their whānau, in line with professional requirements (<http://www.health.govt.nz/publication/equity-health-care-maori-framework>).
- Māori leadership is required in the development of asthma management programmes that improve access to asthma care and facilitate ‘wrap-around’ services to address the wider determinants (such as housing or financial factors) for Māori with asthma (<http://practice.mvcot.govt.nz/policy/assessment-and-decision-making/resources/working-with-maori.html>; <http://what-works.org.nz/kaupapa-maori/>).

Pacific peoples—getting it right for Pacific children with asthma

Goal: Pacific children have asthma outcomes equal to non-Pacific & non-Māori children

The Pacific population is diverse and growing fast, with Pacific children numbering one in four babies born in Auckland. Pacific children have great disparities and unequal access to healthcare compared with other New Zealand children, which is well documented.³²⁻³⁷ Changes will come from health workers understanding the drivers for poor health in minority groups, and action at multiple levels of the health and social systems. Central action to improve the health of Pacific children will

be a commitment to work with the strengths of the Pacific communities.

The following recommendations for health services and practitioners are based on theory and lessons from good practice:

- Understand the Pacific population profile, with the majority living in urban areas. Perform an audit on the clinical activities and understand who is registered with the service, and who is registered but does not attend.
- With over 60% of Pacific children living in families with hardship and 30% in severe hardship, material insecurities will affect the family's engagement with health providers. Practitioners should explore these insecurities and set up effective pathways to address them.
- Research shows communication difficulties are a barrier for healthcare. Assess the level of English language proficiency, and use interpreters if necessary. The Health and Disability Commissioner's Code of Rights (<http://www.hdc.org.nz/the-act--code/the-code-of-rights>) outline the right to a competent interpreter.

Health systems approaches

Goal: All aspects of the health system will support better asthma care, aiming to decrease inequities and improve outcomes

Good asthma management requires a system approach incorporating information systems to improve quality and service delivery. The following are recommended:

- Educate clinicians by providing educational outreach visits, such as training visits to GP practices.
- Computerised decision-support systems, such as web-based systems for self-management. These should

incorporate simple tools for the assessment and monitoring of asthma control.

- School-based asthma interventions, such as education programmes and *Asthma Friendly Schools*.
- Pharmacy-based interventions, such as inhaler technique education and the identification of asthma medicine uptake from dispensing history, eg, infrequent preventer dispensing history.
- Continuity of care between doctors, nurses, pharmacists and patients.

Medicines

Inhaler devices at different ages

Goal: The correct inhaler device is considered and age appropriate

- Prescribe an inhaler device that is appropriate for the development of the child and that the child and/or caregiver is able to demonstrate they can use well (Table 4).
- Health professionals who teach patients should ensure they have correct inhaler technique themselves.
- When teaching inhaler technique, have the child or caregiver demonstrate how they use the device. Use checklists and reminder lists to identify and correct errors.
- Inhaler technique needs to be taught repeatedly. Check inhaler technique and adherence every visit by asking child or caregiver to demonstrate how they use the device.
- Advise not to share inhalers.
- Consider alternative inhaler devices if the patient has persistent difficulty with technique.

Table 4: Inhaler devices recommended by age group.

Inhaler device	<2 years	2–4 years	5–7 years	8–15 years
MDI, small volume spacer & mask	Yes	May transition to no mask		
MDI & spacer No mask		Possible	Yes	Yes
MDI (alone)*				Possible, but use with a spacer is preferable
Dry powder device			Possible	Yes
Breath-activated device			Possible	Yes

*A spacer should be used with the metered dose inhaler (MDI) for the regular administration of inhaled corticosteroids, and for the administration of short-acting beta agonist (SABA) in the setting of an acute attack. The use of a spacer is always recommended.

Figure 3: Stepwise approach to treatment of children with wheeze 1–4 years.

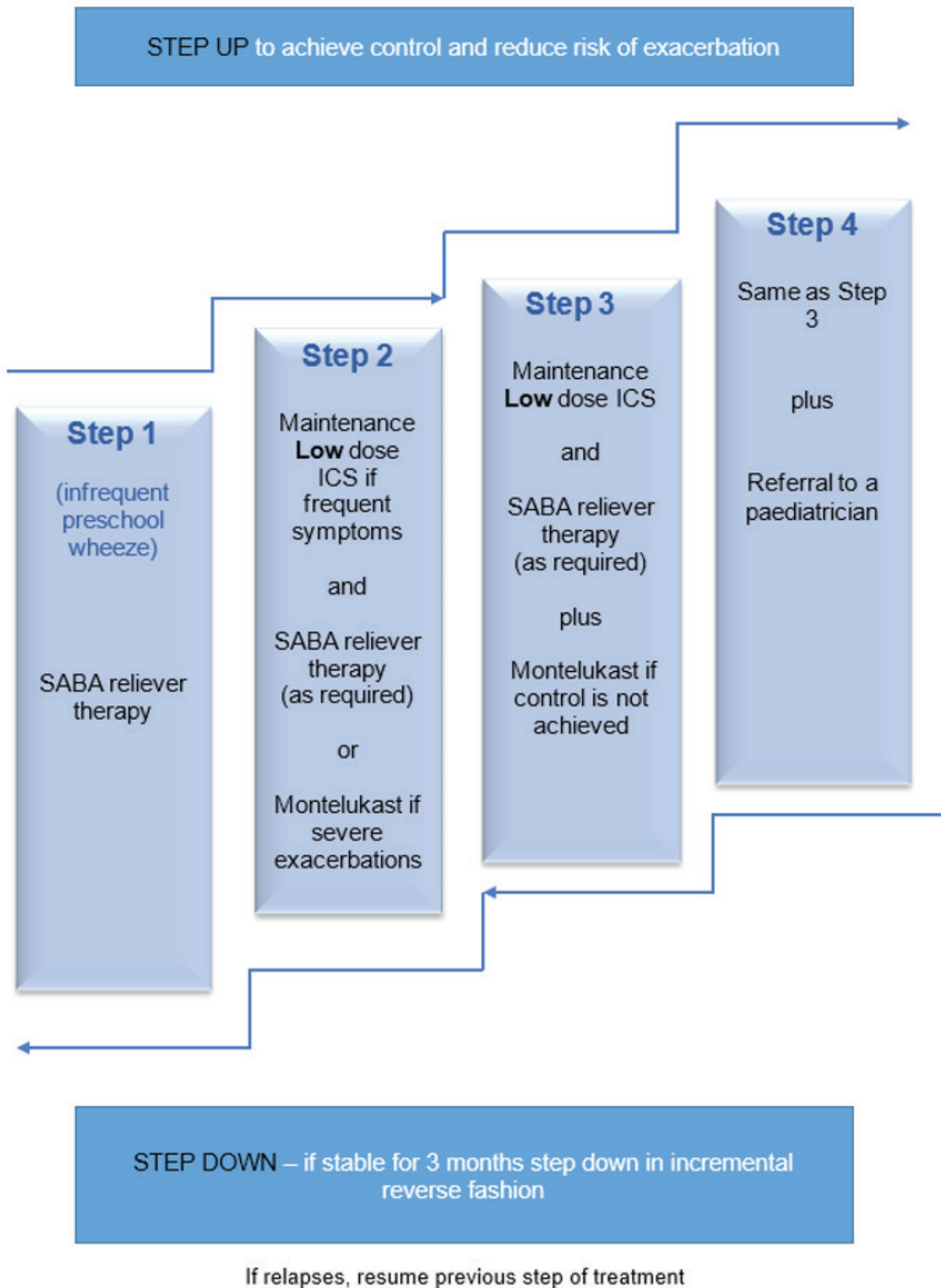
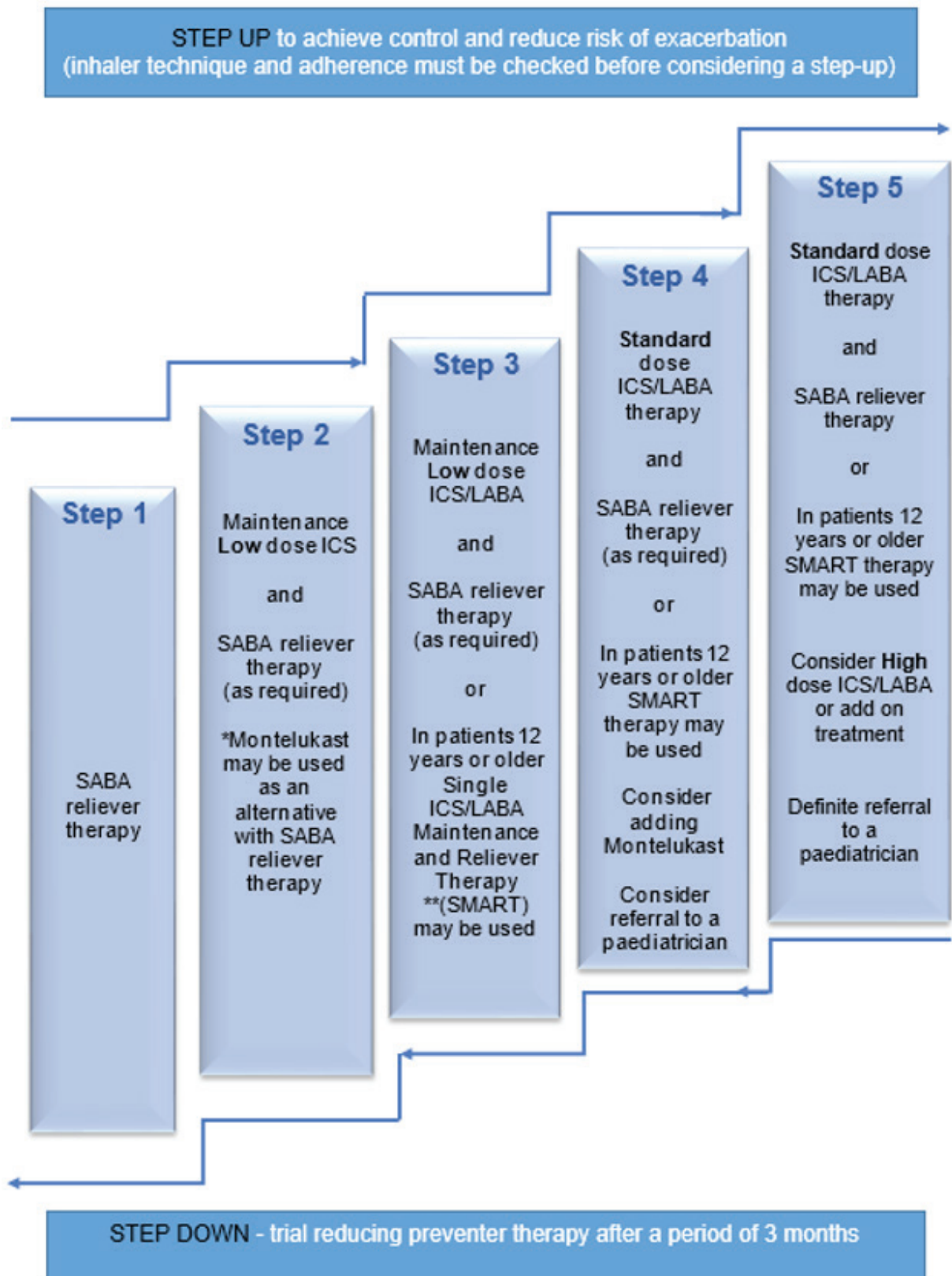


Figure 4: Stepwise approach to treatment of children with asthma 5–15 years.



*Montelukast not funded in this instance
**Budesonide 100mcg and Formoterol 6 mcg

Stepwise approach to long-term asthma treatment

Goal: The right step of medicine in the right device is used for the age and symptoms of the child

- In the stepwise approach to management, children step-up and down therapy as required to achieve and maintain control of their symptoms and reduce the risk of exacerbations.^{6,8,39}
- Achieving good control requires frequent and repeated assessments. This may require the use of recall or follow-up systems.
- At each step, check inhaler technique, adherence to treatment, understanding of a self-management plan, and barriers to self-care.^{6,8,42-46}

Practice points—stepwise management

- Step-up and step-down are determined by asthma control (see section *Evaluation of asthma control and severity*). Step-up may be required when asthma is partially controlled or uncontrolled. Once asthma has been well-controlled for at least eight weeks, consider step-down and reassess control after at least eight weeks.
- Many children have intermittent asthma (Step 1) and do not need an asthma preventer.
- Recommended doses of ICS are lower in children than adults (see Table 5). The usual maximum daily dose in children is also lower than adults, and equivalent to beclomethasone 800 micrograms or fluticasone propionate

500 micrograms. Both these doses are at the top of the dose response curve. If this dose is exceeded there is no therapeutic benefit and there is an increase in adverse medication effects. At Step 5, oral steroids, oral theophylline and even subcutaneous monoclonal antibody therapy (IgE) may be considered as an add-on treatment, if directed by a paediatrician.

- Alternative therapies, such as sodium cromoglycate, may be considered in some children on the lower steps. Long-acting muscarinic receptor antagonists may be future add-on therapy, such as tiotropium, which is currently licensed for use but not funded for asthma indications in New Zealand.²¹
- Remember non-pharmacological approaches to management as well as medicines (see *Health Professional Top 10 Actions* and *Non-pharmacological measures* sections).

Initial treatment choices (when to add ICS)

Goal: For all children with asthma it should be clear if ICS should be prescribed, and if so, a prescription given and the medicine taken

- At initial diagnosis, all children with asthma should be provided with a short-acting beta-agonist (SABA) to take as required for relief of symptoms.
- The key issue is when to start ICS therapy. It is recommended that ICS therapy is introduced if children have symptoms >2 times per week.

Table 5: The recommended low and standard daily dose of ICS in children with asthma. “High” doses are double the standard doses (see Tables 4 and 5).

Low dose		Standard dose	
Beclomethasone dipropionate	200mcg/day	Beclomethasone dipropionate	400-500mcg/day
Beclomethasone dipropionate ultrafine	100mcg/day	Beclomethasone dipropionate ultrafine	200mcg/day
Budesonide	200mcg/day	Budesonide	400mcg/day
Fluticasone propionate	100mcg/day	Fluticasone propionate	200-250mcg/day

- An exacerbation requiring oral steroids in the previous year is widely regarded as a requirement for regular ICS therapy.

Practice points on ICS

- The daily doses of ICS in children, which achieve 80–90% of maximum efficacy, are the low doses shown in Table 5. The doses labelled ‘standard’ doses are the same microgram/day ‘standard’ doses in the adult asthma guidelines.
- ICS should be administered from a MDI with spacer, or from a dry-powder inhaler. The child’s ability to use the inhaler should be checked.^{6,7,47,48}

When to add long-acting beta-agonist (LABA) therapy

Goal: LABAs should never be prescribed without ICS

- Combination ICS/LABA combined single inhaler treatment should be prescribed at a fixed maintenance dose and patients also prescribed a SABA as a reliever therapy. LABA monotherapy is unsafe.
- LABAs should not be used in children ≤4 years of age.
- LABAs (with ICS) should not be initiated when the child is clinically unstable. They should be stopped if they are ineffective or worsen asthma stability.
- The SMART (Single Maintenance And Reliever Therapy) regimen is an alternative for children 12 years or older. It

involves using a low-dose budesonide–formoterol combination powder inhaler. The same inhaler is used for both regular twice-daily maintenance use, and for relief of symptoms, instead of salbutamol.^{7,45,49,50} A self-management plan prototype is in the Adult Asthma Guidelines.⁹

- The LABA should be stopped if the child deteriorates after starting it.
- The LABA should be stopped after three months if ineffective.

Treatment of acute severe asthma (primary care, after-hours care or ED)

Goal: All children should be managed to avoid life-threatening asthma or death

- Acute asthma management is based on:
 - Objective measurement of severity.
 - Assessment of the need for referral to hospital and/or hospital admission (Table 5).
 - Administering treatment appropriate for the degree of severity.
 - Repeatedly reassessing the response to treatment.
- Monitor pulse rate, respiratory rate, accessory muscle use and ability to speak (words/breath).
- Key priorities include identification of a life-threatening attack requiring urgent admission to intensive care, and a severe asthma attack requiring hospital admission (Table 6 and Figure 6).

Table 6: Criteria for acute referral to hospital and/or hospital admission in children and adolescents.

<ul style="list-style-type: none"> • Child with any feature of life-threatening asthma • Child with any feature of an acute severe attack persisting after initial treatment • Child in whom other considerations suggest that admission may be appropriate: <ul style="list-style-type: none"> - Still have significant symptoms - Psychosocial problems in child or parent/caregiver - Physical disability or learning difficulties - Previous near fatal or brittle asthma - Exacerbation despite adequate dose of oral steroids pre-presentation - Presentation at night - Remote location or without transportation/communication

Practice points—acute severe asthma

- A lack of response to initial bronchodilator treatment and/or a requirement for repeat doses two-hourly or more often indicates the need for referral to hospital and/or admission.
- For most children, initial treatment with beta-agonist via a spacer and oral steroids is likely to be sufficient. Reserve nebulised beta-agonists for those with severe asthma who require continuous oxygen.
- The standard regimen for a course of prednisone in the situation of severe asthma is 1–2 milligrams/kg (to a maximum of 40 milligrams) daily for 3–5 days.
- Steroids, such as oral prednisone are not likely to be effective in children <5 years. In this age group, they should be reserved for children admitted to

hospital (or who are en route) and who are on oxygen.

- In hospital consider IV magnesium sulphate, aminophylline or salbutamol according to local protocol.
- Non-invasive ventilation in life-threatening asthma is not recommended outside of an intensive care setting.
- For children with acute severe asthma who are treated in primary care or discharged from the after-hours clinic or ED, long-term management should be reviewed and follow-up appointment within the week with their primary healthcare team should be arranged.
- All children ≥5 years who have presented with acute severe asthma and who are not taking ICS should be prescribed ICS before going home.^{8,51–57}

Figure 5: Algorithm for community management of moderate, severe and life-threatening acute asthma in children and adolescents. (Mild asthma is asthma symptoms not usually requiring medical attention and should be managed according to the asthma action plan.)

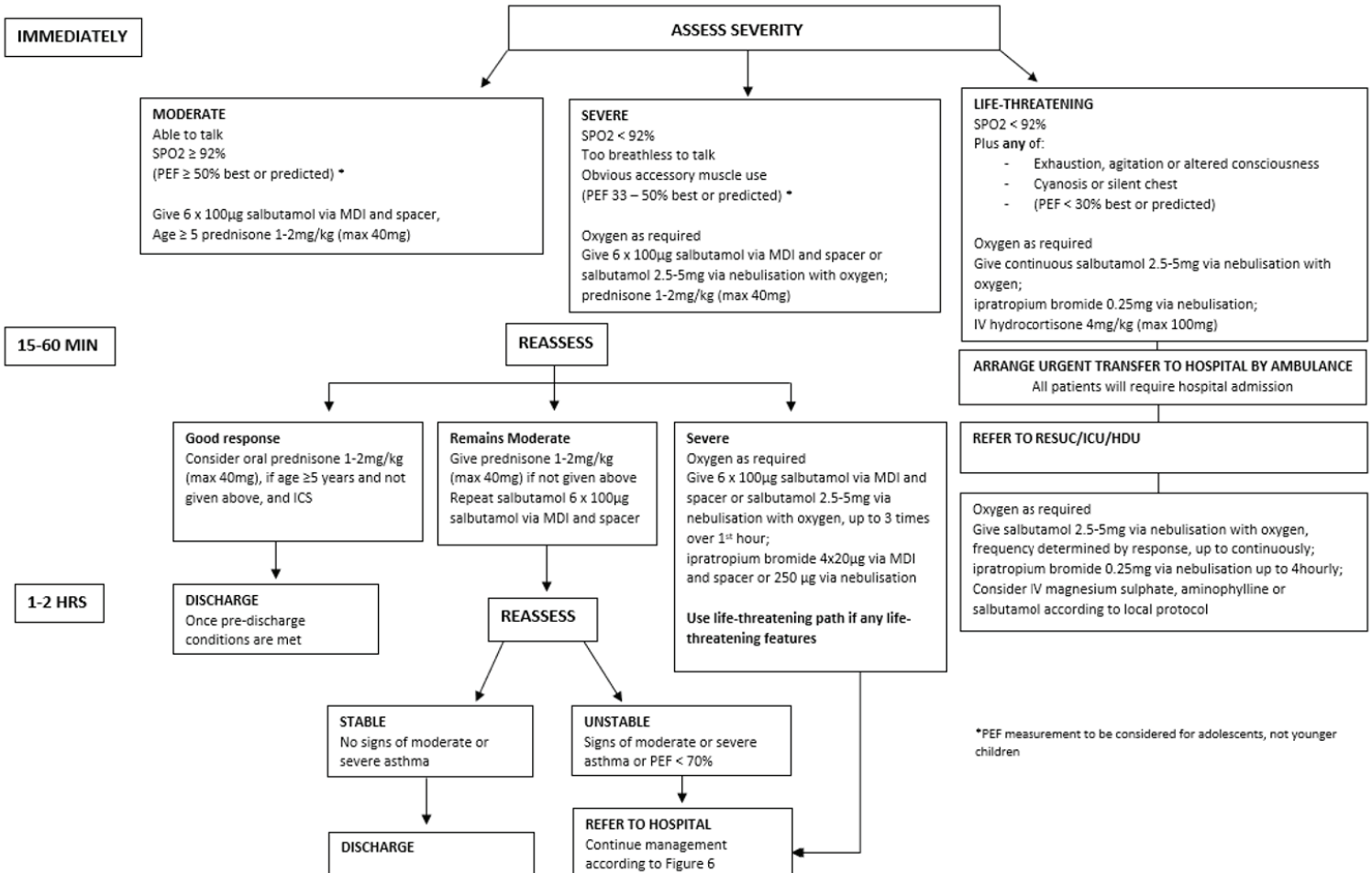


Table 7: Pre-discharge considerations in children and adolescents.

1. Most children presenting with acute exacerbations of asthma should have a course of oral prednisone, 1–2 milligrams/kg (to a maximum of 40 milligrams) daily for 3–5 days.
2. All children admitted to hospital for asthma should have a structured review assessing control, inhaler technique, asthma education, an action plan and follow-up.
3. It is recommended that children have prednisone and ICS dispensed prior to discharge to ensure there are no barriers to taking medication.
4. Before sending a child home, ensure that the child with caregiver:
 - Understands treatment prescribed and the signs of worsening asthma.
 - Can demonstrate inhaler use correctly and has a supply of the medication.
 - Understands how to contact emergency services/seek further advice if symptoms deteriorate (ie, has an action plan).
 - Arranges an early follow-up appointment with their primary healthcare team for review (within a week).
 - Consider referral to asthma educator.
 - Consider housing and social implications, eg, social worker involvement.
 - Encourage notification of hospital admission to school or child care centre.

Checks at follow-up visit after admission

1. Clinical assessment—resolution of symptoms and signs would be expected.
2. Consider spirometry in older children.
3. Understands treatment prescribed and the signs of worsening asthma.
4. Can demonstrate inhaler use correctly and has a supply of the medication.
5. Understands how to contact emergency services/seek further advice if symptoms deteriorate.
6. Check written action plan.
7. Check housing and social implications.

Appendix A



Well

When I'm well:

- I have no cough
- I play just like other children
- I use my reliever puffer less than 2 times a week

My puffers are:

Preventer: I take this every day even when I'm well.
 The name of my preventer is _____ The colour is _____
 I take _____ puffs in the morning and _____ puffs at night through a spacer.

Reliever: I take this only when I need it
 The name of my reliever is _____ The colour is _____
 I take _____ puffs through a spacer when I wheeze, cough or when it's hard to breathe.

If I find it hard to breathe when I exercise I should: Take _____ puffs of my reliever



Worse

When my asthma is getting worse:

- I cough or wheeze and it's hard to breathe, or
- I'm waking at night because of my asthma, or
- I cough or wheeze when I play, or
- I need my reliever inhaler to control my asthma more than 2 times per week

If my asthma gets worse I should:

Keep taking my preventer every day as normal and take _____ puffs of my reliever every 4 hours
 If I'm not getting better doing this I should see my doctor today

Contact:



Worried

My asthma is a worry when:

- My reliever isn't helping, or
- I'm finding it hard to breathe, or
- I'm breathing hard and fast, or
- I'm sucking in around my ribs/throat, try looking under my shirt
- I'm looking pale or blue

- Sit me down and try to stay calm
- Give me 6 puffs of reliever through a spacer, taking 6 breaths for each puff
- **If I don't start to improve I need help now**

Emergency

DIAL 111 and ask for an ambulance

WHILE YOU'RE WAITING:

- Try to stay calm and keep me sitting upright
- Give 6 puffs of reliever through a spacer every 6 minutes with 6 breaths for each puff until help arrives

Date Prepared: _____ Doctors Signature: _____ Plan to be reviewed when treatment changed

Philippa Howden-Chapman	St Johns Ambulance
POI Team Public Health South	Starship Children's Health
ProCare Clinical Advisory Committee	Te Ora: Māori Medical Practitioners
Respiratory and Sleep Medicine Auckland University	Te Rūngangao Aotearoa (NZNO Māori)
Royal Australasian College of Physicians	Thoracic Society of Australia and NZ—NZ Branch
Royal NZ College of GPs	TSANZ Nurses special interest group
Special Education	Wellington Free Ambulance

Competing interests:

Dr Harwood reports personal fees from Astra Zeneca Limited outside the submitted work; Dr Reid reports affiliation with Contract Research outside the submitted work; Dr Asher reports one grant from Boehringer Ingelheim New Zealand outside the submitted work; Dr Ingham reports personal fees from Te Hā Ora: The Asthma & Respiratory Foundation of New Zealand, grants from Janssen Research & Development, non-financial support from Astra Zeneca, outside the submitted work; Teresa Demetriou reports grants from REX Medical outside the submitted work.

Author information:

Innes Asher, Paediatrics: Child and Youth Health, University of Auckland, Auckland; David McNamara, Respiratory Service, Starship Children's Health, Auckland District Health Board, Auckland; Cheryl Davies, Tu Kotahi Māori Trust, Lower Hutt, Wellington; Teresa Demetriou, Asthma and Respiratory Foundation NZ, Wellington; Theresa Fleming, Paediatrics: Child and Youth Health & Psychological Medicine, University of Auckland, Auckland; Matire Harwood, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, Auckland; Lorraine Hetaraka-Stevens, National Hauora Coalition, Auckland; Tristram Ingham, Medicine, University of Otago, Wellington; John Kristiansen, Health Quality and Safety Commission, Auckland; Jim Reid, Dept of the Dean, Dunedin School of Medicine, Dunedin; Debbie Rickard, Capital and Coast District Health Board, Wellington; Debbie Ryan, Pacific Perspectives Limited, Wellington.

Corresponding author:

Professor Innes Asher, Paediatrics: Child and Youth Health, University of Auckland, Private Bag 92019, Auckland 1142.
i.asher@auckland.ac.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7425>

REFERENCES:

- Howden-Chapman P, Baker MG, Bierre S. The Houses children live in. Policies to improve housing quality Policy Quarterly. 2013; 9(2):35–9.
- Ministry of Health. Un-flued gas heaters 2016. Available from: <http://www.health.govt.nz/your-health/healthy-living/environmental-health/household-items-and-electronics/unflued-gas-heaters>
- Telfar Barnard L, Baker M, Pierser N, Zhang J. The impact of respiratory disease in New Zealand: 2014 update. Wellington: The Asthma Foundation; 2015.
- Jones B, Ingham T. He Māramatanga Huangō: Asthma health literacy for Māori children in New Zealand: Report to the Ministry of Health. Wellington: Asthma Foundation; 2015.
- Asthma and Respiratory Foundation of New Zealand. Te Hā Ora (The Breath of Life): National Respiratory Strategy. Wellington: The Asthma Foundation 2015.
- National Asthma Council Australia. Australian Asthma Handbook Version 1.1 April 2015 2015 [cited 2016 7 June]. Available from: www.nationalasthma.org.au/handbook

7. Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention, 2016. Available from: www.ginasthma.org
8. SIGN 153. British guideline on the management of asthma 2016. Available from: <http://www.sign.ac.uk/>
9. Beasley R, Hancox B, Harwood M, Perrin K, Poot B, Pilcher J, et al. Asthma and Respiratory Foundation NZ Adult Asthma Guidelines: A Quick Reference Guide. *New Zealand Medical Journal*. 2016; (1445):83–102.
10. Brand PLP, Caudri D, Eber E, Gaillard EA, Garcia-Marcos L, Hedlin G, et al. Classification and pharmacological treatment of preschool wheezing: changes since 2008. *European Respiratory Journal*. 2014; 43:1172–7.
11. Kaiser SV, Huynh T, Bacharier LB, Rosenthal JL, Bakel LA, Parkin PC, et al. Preventing Exacerbations in Preschoolers With Recurrent Wheeze: A Meta-analysis. *Pediatrics*. 2016; e-pub June.
12. Castro-Rodriguez JA, Custovic A, Ducharme F. Treatment of asthma in young children: evidence-based recommendations. *Asthma Research and Practice*. 2016; 2:5–11.
13. Work and Income. Guidance on the severity of disability for child disability allowance. Available from: <http://www.workandincome.govt.nz/providers/health-and-disability-practitioners/guides/guidance-on-the-severity-of-disability-for-child-disability-allowance.html>
14. Gardiner and Dvorkin (2006) Promoting medication adherence in children. *American Family Physician* 74 (5) 793-798
15. Fleming T, Watson P. Enhancing compliance in adolescents: if only they'd do what they're told *Current Therapeutics*, Vol. 43, No. 3, 2002 Mar: 14 <<http://search.informit.com.au/documentSummary;dn=526872563101018;res=IELHEA>>
16. Duncan C MJ, Wu YP, Fredericks EM. Practice-based approach to assessing and treating nonadherence in pediatric regimens. *Clinical Practice in Pediatric Psychology*. 2014; 3(2):322–36.
17. PREDICT. Bronchiolitis Guideline 2017. Available from: <http://www.predict.org.au/2016/project-snapshot-development-of-the-australasian-bronchiolitis-clinical-practice-guideline/>
18. Fingleton J, et al. Bronchodilator responsiveness: interpret with caution. *Thorax* 2012; 67:667–8.
19. Jamison JP, McKinley RK. Validity of peak flow rate variability for the diagnosis of asthma. *Clin Sci* 1993; 85:367–71.
20. Crane, et al. Markers of risk of asthma death or readmission in the 12 months following a hospital admission for asthma. *Int J Epidemiol* 1992; 21:737–44.
21. Kerstjens, et al. Tiotropium in asthma poorly controlled with standard combination therapy. *N Engl J Med* 2012; 367:1198–1207.
22. Kristiansen J, Hetutu E, Manukia M, Jelleyman T. An evaluation of a pictorial asthma medication plan for Pacific children. *New Zealand Medical Journal* 2012; 125(1354):42–50. <http://journal.nzma.org.nz/journal/125-1354/5173/>
23. Neinstein LS, Katzman D, Callahan T, Gordon C, Joffe A, Rickert V. (2016) Adolescent health care: a practical guide Wolters Kluwer
24. Tylee, Haller, Graham, et al. Youth-friendly primary-care services: How are we doing and what more needs to be done? *Lancet*, 369 (2007), pp. 1565–1573.
25. WHO (2005) Adolescent friendly health services: Making it happen WHO (2005).
26. Department of Health. 'You're Welcome': Quality criteria for young people friendly health services. 2011: http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_126813
27. Farrant PD. Watson-Health care delivery: Perspectives of young people with chronic illness and their parents *J Paediatr Child Health*, 40 (2004), pp. 175–179.
28. Klein D, Goldenring J, Adelman W. HEADSSS 3.0: The psychosocial interview for adolescents updated for a new century fueled by media. *Contemporary Pediatrics*; 2014. Available from: <http://contemporarypediatrics.modernmedicine.com/contemporary-pediatrics/content/tags/adolescent-medicine/heedsss-30-psychosocial-interview-adolesce?page=full>
29. Holley S, Morris R, Knibb R, Latter S, Lioffi C, Mitchell F, et al. Barriers and Facilitators to Asthma Self-Management in Adolescents: A Systematic Review of Qualitative and Quantitative Studies. *Pediatric Pulmonology*. 2016; early e-pub.
30. Ministry of Health. *Tatau Kahukura: Māori Health Chart Book 2010*, 2nd Edition. Wellington: Ministry of Health; 2010.
31. Ministry of Health. 2014. *Equity of Health Care for Māori: A framework*. Wellington:

- Ministry of Health. <http://www.health.govt.nz/publication/equity-health-care-maori-framework>
32. Simpson J, Oben G, Craig E, Adams J, Wicken A, Duncanson M, et al. The Determinants of Health for Pacific Children and Young People in New Zealand (2014): New Zealand Child and Youth Epidemiology Service; 2016. Available from: <http://hdl.handle.net/10523/6747>
 33. Craig E, Reddington A, Adams J, Dell R, Jack S, Oben G, et al. The Health of Pacific Children and Young People with Chronic Conditions and Disabilities in New Zealand (2013): New Zealand Child and Youth Epidemiology Service; 2016. Available from: <http://hdl.handle.net/10523/6746>
 34. Craig E, Adams J, Oben G, Reddington A, Wicken A, Simpson J. The Health Status of Pacific Children and Young People in New Zealand: New Zealand Child and Youth Epidemiology Service; 2012. Available from: <http://hdl.handle.net/10523/6131>
 35. Davis P, Sualii-Sauni T, Lay-Yee R, Pearson J. Pacific Patterns in Primary Health Care: A comparison of Pacific and all patient visits to doctors: The National Primary Medical Care Survey (NatMedCa): 2001/02. Wellington: Ministry of Health; 2005.
 36. Health Mo. Tupu Ola Moui Pacific Health Chart Book 2012. Wellington: Ministry of Health; 2012.
 37. Gray B. The use of interpreters In: St George IM, editor. Cole's medical practice in New Zealand. 12th ed. Wellington: Medical Council of New Zealand; 2013.
 38. Gotzsche PC, Johansen HK. House dust mite control measures for asthma. The Cochrane Collaboration 2008.
 39. Holt S, et al. The use of the self-management plan system of care in adult asthma. *Prim Care Resp J* 2004; 13:19–27.
 40. Gibson PG, Powell H. Written action plans for asthma: an evidence-based review of the key components. *Thorax* 2004; 59:94–9.
 41. Robson B, Harris R. Hauora: Māori Standards of Health IV. A study of the years 2000–2005. 2007. Tatau Kahukura: Māori Health Chart Book 2015, 3rd edition. Accessed <http://www.health.govt.nz/publication/tatau-kahukura-maori-health-chart-book-2015-3rd-edition>
 42. D'Souza W, et al. Asthma morbidity 6 yrs after an effective asthma self-management programme in a Māori community. *Eur Respir J* 2000; 15:464–9.
 43. Pilcher J, et al. Combination budesonide/formoterol inhaler as maintenance and reliever therapy in Māori with asthma. *Respirology* 2014; 19:842–51.
 44. Asthma and Respiratory Foundation of New Zealand 2015. Te Hā Ora (The Breath of Life): National Respiratory Strategy. Wellington: The Asthma Foundation.
 45. Powell H, Gibson P. High dose versus low dose inhaled corticosteroids as initial starting dose for asthma in adults and children. The Cochrane Collaboration. The Cochrane Library 2008, Issue 4.
 46. Holt S, et al. Dose-response relation of inhaled fluticasone propionate in adolescents and adults with asthma: meta-analysis. *BMJ* 2001;323:253.
 47. Patel M, et al. Efficacy and safety of maintenance and reliever combination budesonide-formoterol inhaler in patients with asthma at risk of severe exacerbations: a randomised controlled trial. *Lancet Respir Med* 2013; 1:32–42.
 48. Beasley R, et al. Call for withdrawal of LABA single-therapy inhaler in asthma. *Lancet* 2010; 376:750–1.
 49. Aldington S, Beasley R. Asthma exacerbations. 5: Assessment and management of severe asthma in adult in hospital. *Thorax* 2007; 62:447–58.
 50. Llewellyn P, et al. The relationship between FEV1 and PEF in the assessment of the severity of airways obstruction. *Respirology* 2002; 7:333–7.
 51. Rowe BH, et al. Corticosteroids for preventing relapse following acute exacerbations of asthma (Review). The Cochrane Collaboration. The Cochrane Library 2007, Issue.
 52. Bowler SD, et al. Corticosteroids in acute severe asthma: effectiveness of low doses. *Thorax* 1992; 47:584–7.
 53. Camargo CA, et al. Continuous versus intermittent beta-agonists for acute asthma. The Cochrane Collaboration. The Cochrane Library 2003.
 54. Cates CJ, et al. Holding chambers (spacers) versus nebulisers for beta-agonist treatment of acute asthma (Review). The Cochrane Collaboration. The Cochrane Library 2013, Issue 9.
 55. Emerman CL, et al. Comparison of 2.5 vs 7.5 mg of inhaled Albuterol in the treatment of acute asthma. *Chest* 1999; 115:92–6.

Breastfeeding indicators among a nationally representative multi-ethnic sample of New Zealand children

Teresa Castro, Cameron Grant, Clare Wall, Michaela Welch, Emma Marks, Courtney Fleming, Juliana Teixeira, Dinusha Bandara, Sarah Berry, Susan Morton

ABSTRACT

AIMS: To describe breastfeeding initiation and duration, and demographic associations with breastfeeding duration within a representative sample of New Zealand infants.

METHODS: In 6,685 singletons enrolled in the *Growing Up in New Zealand* cohort we described breastfeeding initiation (96%), any (94%) and exclusive (93%) breastfeeding (EBF) duration. We used adjusted relative risk (RR) and 95% confidence intervals (CI) to describe associations with breastfeeding duration.

RESULTS: Breastfeeding initiation occurred for 97%. Sixteen percent were EBF to age six months and 13% were breastfed to age 24 months. Exclusive breastfeeding for ≥ 4 months was less likely for children of mothers of Māori (RR=0.80, 95% CI 0.73–0.87), Pacific (0.90, 95% CI 0.83–0.98) or Asian (0.80, 95% CI 0.74–0.86) ethnicity. Children of mothers aged 20–29 years (1.24, 95% CI 1.04–1.49); ≥ 30 years (1.36, 95% CI 1.14–1); with a tertiary education (1.14, 95% CI 1.08–1.21); or planned pregnancy (1.14, 95% CI 1.08–1.21); and children with older siblings (RR=1.31, 95% CI 1.17–1.47) were more likely to be exclusively breastfed for ≥ 4 months. Children were more likely to be breastfed ≥ 6 months if their mother was aged 20–29 (1.26, 95% CI 1.10–1.45) or ≥ 30 years (1.40, 95% CI 1.22–1.61), had a tertiary education (1.11, 95% CI 1.06–1.59) or planned pregnancy (1.11, 95% CI 1.06–1.15), or if they had older siblings (1.04, 95% CI 1.00–1.08).

CONCLUSION: In New Zealand, most children are initially breastfed, however a large proportion did not receive the recommended duration of any or exclusive breastfeeding. Maternal age, education, parity and pregnancy planning identify children at risk of shorter duration of breastfeeding and EBF, and maternal ethnicity identifies children at risk of shorter EBF duration.

Breastfeeding reduces the risk of child deaths and of infectious disease morbidity.¹ Breastfeeding is associated with fewer dental malocclusions, higher intelligence quotient scores and a reduced risk of overweight and diabetes.¹

The World Health Organization (WHO) recommends breastfeeding initiation in the first hour after birth, exclusive breastfeeding to age six months and continued breastfeeding to age two years and beyond, with nutritionally, adequate, safe and

age-appropriate complementary feeding starting at age six months.² The Global Strategy for Infant and Young Child Feeding (IYCF) developed by the WHO and the United Nations Children's Fund (UNICEF) establishes the worldwide core indicators for breastfeeding monitoring: early breastfeeding initiation, exclusive breastfeeding to age six months and continued breastfeeding to age one year. Where core indicators are unavailable, alternative indicators that can be used are: child ever breastfed, continued

breastfeeding at two years, age-appropriate breastfeeding, predominant breastfeeding under six months of age and median breastfeeding duration. With the exception of 'early initiation of breastfeeding' and 'children ever breastfed', all indicators should describe current status.³

Reviews conducted by the Organisation for Economic Co-operation and Development (OECD), which includes mostly high-income countries, highlight that, despite the high policy relevance of breastfeeding as an indicator of child's wellbeing, the poor quality of national-level data limits the capacity for between-country comparisons.⁴ For example, a recent WHO attempt to describe global trends in breastfeeding indicators showed that, among high-income countries, data was limited to 37 of 75 countries and, for most, information about early initiation or exclusive or continued breastfeeding duration at two years were not available.¹

In general, the proportion of children ever breastfed is smaller in high-income (averaging 82%), compared with low-and-middle-income countries (averaging 95%). While most infants in low-income countries are still breastfed at age one year, this duration is achieved on average for less than 20% of infants in high-income countries.¹ In addition, the available data show wide variability in exclusive breastfeeding rates between high-income countries. For example at age six months, the percentage of children exclusively breastfed were: Spain (25%), Canada (14%), the US (14%), Sweden (12%), Poland (4%) and Bulgaria (2%).⁵⁻⁶

In New Zealand, data on breastfeeding indicators are collected by Lead Maternity Carers (LMCs) and Well Child providers (*Plunket National Child Study*).⁷ In 2010, 85.7% of all women giving birth were registered with a LMC provider. LMCs report information on breastfeeding status (exclusively, fully, partial, artificial feeding) when infants are discharged from their care.⁷ The *Plunket system* collects information on breastfeeding at Well Child visits at age six weeks, three and six months. It also reports on food and drink items that infants received in the 48 hours prior to each visit.

One important limitation of the breastfeeding data available in New Zealand is that coverage for all births is incomplete. The *Plunket system*⁷ currently enrolls 88% of the newborn population, however Māori and Pacific mothers are under-reported in

the data collected.⁸ Currently, New Zealand does not have data on breastfeeding indicators that is generalisable to the national birth cohort.

Successful breastfeeding depends on several factors related to the child, mother and environment. The investigation of social determinants of initiation, duration and exclusivity of breastfeeding, at a population level, has important implications for public health interventions that aim to increase breastfeeding rates and to allow measurement of effectiveness of interventions over time.⁹ We aimed to describe, in a contemporary representative sample of New Zealand children, the indicators of breastfeeding initiation and duration, and to identify independent maternal and household characteristics associated with duration of any and exclusive breastfeeding.

Methods

Growing up in New Zealand cohort and study population

We completed this study within New Zealand's contemporary child cohort study, *Growing Up in New Zealand*, which enrolled 6,822 pregnant women. The child cohort consists of 6,853 children who survived to age six weeks.^{10,11} Eligibility of pregnant women was determined by an estimated delivery date between 25 April 2009 and 25 March 2010, and residence while pregnant in the New Zealand region defined by the three contiguous district health boards of Auckland, Counties Manukau and Waikato. There were no other inclusion or exclusion criteria. Alignment of the cohort with all births in New Zealand between 2007 and 2010 has been demonstrated.¹² Ethical approval was granted by the Ministry of Health Northern Y Regional Ethics Committee (NTY/08/06/055), and written informed consent was obtained from all mothers.¹²

Measurement of breastfeeding initiation and duration

WHO definitions of breastfeeding initiation and duration were used: a) not breastfed; b) any breastfeeding, defined as receiving some breast milk but also receiving other milk and/or solids and; c) exclusive breastfeeding, defined as receiving only breast milk and no other milk, solids, fluids or water.¹³ Information on these indicators was obtained through maternal report with data collected at face-to-face interviews when the children were nine

months old and telephone interviews when the children were six weeks, 31 and 45 months old.

The description of breastfeeding initiation used information collected when the children were nine, 31 and 45 months old. Breastfeeding initiation was defined by the question “*Did you ever breastfeed this baby?*” The description of breastfeeding duration used information collected when the children were nine and 31 months old and was determined by the question “*How old was your baby when you stopped breastfeeding?*”. For the children that were still being breastfed at the nine-month interview, or for whom information on breastfeeding duration was missing at that time point, information about breastfeeding duration was obtained from the 31-month interview. The description of exclusive breastfeeding duration used the information collected when the children were nine months old and was determined by the question “*How long did you exclusively breastfeed? By exclusively I mean feeding baby only breast milk (including expressed breast milk) and not any water, milk formula, other liquids or solid foods*”.

The duration of exclusive breastfeeding (in months) reported by the mothers was corrected for the information about the child’s feeding reported when they were six weeks old and by the age of introduction of foods or drinks reported when they were nine months old. At the six-week interview the mothers were asked how they were feeding their babies (only breast milk; mainly breast milk but has also received some water-based drinks, only formula; formula and breast milk, others). At the nine-month interview each mother was asked how old their child was when they first tried a list of 25 food items (infant milk formula or milk; baby rice; baby breakfast cereal; other cereal; bread or toast; rusks; biscuits; vegetables; fruit; meat; fish; eggs; puddings; nuts or peanut butter; shellfish; soy foods; sweets; chocolate; hot chips; potato chips/crisps; fruit juices; herbal drinks; tea; coffee; soft drinks). This food list was designed by an experienced dietitian (CRW) who selected the food items based on the Food and Nutrition Guidelines for 0–2 year-olds⁷ and foods and beverages

commonly fed to infants from a previous study which assessed prevalence of the dietary intake and nutritional status of an ethnically diverse sample of 6–23 month-olds.¹⁴

Covariates

Maternal and household variables identified as potentially influencing any and exclusive breastfeeding duration were examined. Information on self-prioritised ethnicity, age, parity, pregnancy planning, education and household deprivation were collected at the face-to-face antenatal maternal interview.

Maternal self-prioritised ethnicities were gathered at the most detailed level possible, and were then coded into six Level 1 categories following the Statistics NZ coding criteria; (1) European, (2) Māori, (3) Pacific Peoples, (4) Asian, (5) Middle Eastern, Latin American and African (MELAA), and (6) other, with MELAA and other then combined for analysis purposes.

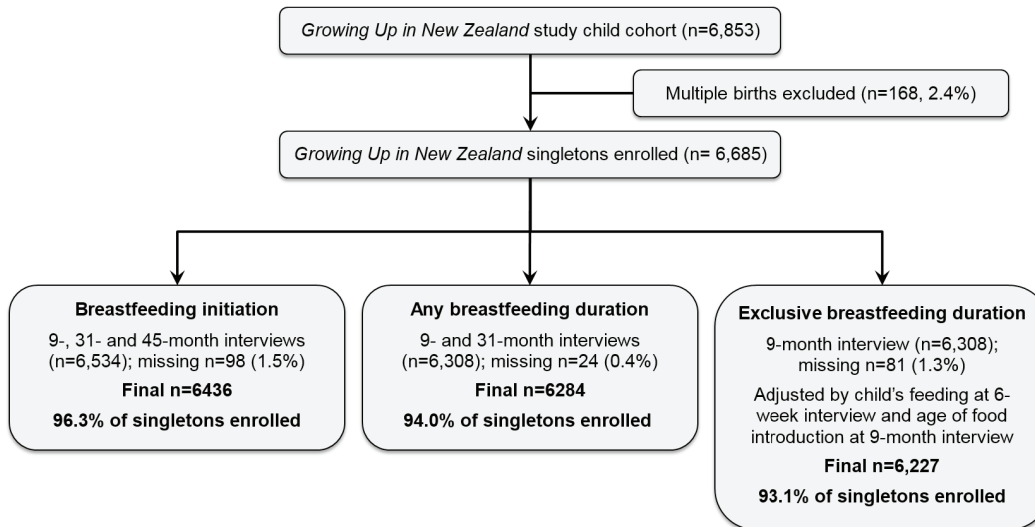
Household deprivation was measured using NZDep06, which combines nine socioeconomic characteristics from 2006 census data collected at aggregations of approximately 100 people and assigned to individual households based on geo-coded address data.¹⁵

Statistical analyses

Proportions, medians and interquartile ranges (IQR) were calculated. Twins or triplets and children for whom data describing BF indicators or covariates were missing were excluded from the analysis. The rates of any and exclusive breastfeeding duration presented included children who were never breastfed.

For analysis purposes, binary outcome variables for breastfeeding duration were created: <6 vs ≥6 months for any breastfeeding; and <4 vs ≥4 months for exclusive breastfeeding. Poisson regression with robust variance was used to estimate the relationship between the covariates and breastfeeding outcomes. Unadjusted and adjusted relative risk (RR) with 95% confidence intervals (CI) were presented. Analyses were performed using IBM SPSS Statistic 22 software. All P values were two-tailed and P<0.05 was the level of significance.

Figure 1: Number of children enrolled in the *Growing Up in New Zealand* cohort study included in each of the breastfeeding indicator estimates.



Results

Study population and breastfeeding initiation

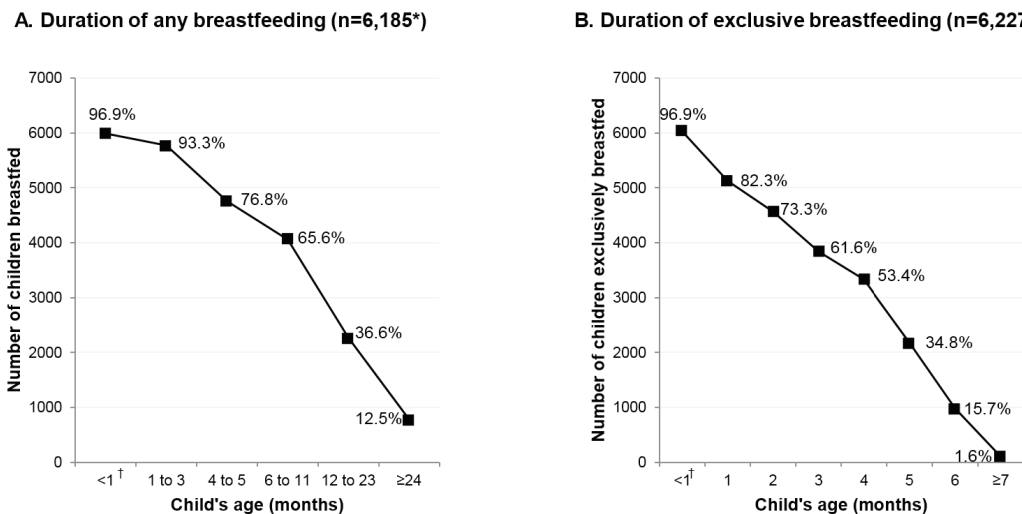
From the 6,853 children enrolled, 6,685 (97.6%) were singletons. Information about breastfeeding initiation, any and exclusive breastfeeding duration were available for 96.3%, 94.0% and 93.1% of these 6,685 children, respectively. Breastfeeding was not initiated for 193 (3%) of the children (Figure 1). Rates of breastfeeding initiation varied by maternal ethnicity: Māori (95.4%), Pacific (94.7%), European (97.6%), Asian (98.0%) and others (99.6%); ($P < 0.001$). Compared to children of European mothers, children

whose mothers were of Māori (RR=1.89, 95% CI 1.32–2.72) or Pacific (RR=2.16, 95% CI 1.53–3.04) ethnicity were less likely to have breastfeeding initiated.

Any and exclusive breastfeeding duration

Median (IQR) duration of any and exclusive breastfeeding were seven (4.0–12.0) and four months (2.0–5.0), respectively. At age six months, one and two years, 65.6%, 36.6% and 12.5%, respectively, of the children were still being breastfed. At age four and six months 53.4%, and 15.7% of the children, respectively, were being exclusively breastfed (Figure 2).

Figure 2: Duration of any breastfeeding (A) and exclusive breastfeeding (B), as defined by parental report.



*Does not include 99 children still being breastfed at nine months but for whom subsequent information on breastfeeding duration was not available.

†The age category '<1 month' included children breastfed for 1–14 days.

Rates of any and exclusive breastfeeding by maternal demographics

Maternal age, education, pregnancy planning and parity were independently associated with the duration of any breastfeeding (Table 1). The likelihood of any breastfeeding for ≥6 months was increased

for children of women: who were aged 20–29 (RR=1.26, 95% CI 1.10–1.45) or ≥30 (RR=1.40, 95% CI 1.22–1.61) versus <20 years old; who had a tertiary (RR=1.11, 95% CI 1.06–1.59) versus secondary or less education; for whom this was a planned (RR=1.11, 95% CI 1.06–1.15) pregnancy; or was their first child (RR=1.04, 95% CI 1.00–1.08).

Table 1: Associations of maternal and household demographics with duration of any breastfeeding.

Maternal and household demographics	Any breastfeeding for six months or more		Adjusted and unadjusted relative rates for breastfeeding for six months or more		
	Yes N=1,934 n (%)	No N=4,157 n (%)	Unadjusted RR (95% CI)	Adjusted RR (95% CI)	Forest plot of adjusted RR
Self-prioritised ethnicity					
European	2,364 (71)	985 (29)	1.00	1.00	
Māori	492 (61)	311 (39)	0.87 (0.82–0.92)	0.98 (0.92–1.04)	
Pacific	518 (64)	398 (36)	0.90 (0.85–0.95)	1.02 (0.95–1.08)	
Asian	6,220 (69)	274 (31)	0.98 (0.94–1.03)	1.01 (0.96–1.06)	
Other*	153 (72)	61 (28)	1.01 (0.93–1.10)	1.02 (0.94–1.12)	
Age group					
<20	123 (45)	149 (55)	1.00	1.00	
20–29	1,475 (63)	863 (37)	1.39 (1.22–1.60)	1.26 (1.10–1.45)	
≥30	2,559 (74)	922 (26)	1.63 (1.42–1.86)	1.40 (1.22–1.61)	
Education					
Secondary level or lower	1,077 (60)	706 (40)	1.00	1.00	
Tertiary	3,074 (72)	1,218 (29)	1.18 (1.14–1.24)	1.11 (1.06–1.59)	
Pregnancy planning					
Unplanned	1,412 (61)	897 (39)	1.00	1.00	
Planned	2,733 (73)	1,025 (27)	1.19 (1.14–1.23)	1.11 (1.06–1.15)	
Parity					
First Child	1,708 (66)	889 (34)	1.00	1.00	
Subsequent child	2,449 (70)	1,041 (30)	1.07 (1.03–1.10)	1.04 (1.00–1.08)	
Household deprivation[†]					
1 to 3 (least deprived)	1,136 (73)	427 (27)	1.00	1.00	
4 to 7	1,576 (69)	702 (31)	0.95 (0.91–0.99)	0.98 (0.94–1.02)	
8 to 10 (most deprived)	1,444 (64)	804 (36)	0.88 (0.85–0.92)	0.97 (0.92–1.01)	

*Other includes Middle Eastern, Latin American and African.

[†]Area-level socio-economic deprivation was measured using the NZ Index of Deprivation, Dep 1,2 & 3 = least deprived, Dep 8,9 & 10 = most deprived households.¹⁵

RR – Relative Risk; CI – confidence interval.

Interactions not included in this model. Missing number of cases for: self-prioritised ethnicity (103), Age group (86), education (104), pregnancy planning (116), parity (93), household deprivation (88).

Table 2: Associations of maternal and household demographics with duration of exclusive breastfeeding.

Maternal and household demographics	Exclusive breastfeeding for four months or more		Adjusted and unadjusted relative rates for exclusive breastfeeding for four months or more		
	Yes N = 3,328 n (%)	No N = 2,706 n (%)	Unadjusted RR (95% CI)	Adjusted RR (95% CI)	Forest plot of adjusted RR
Self-prioritised ethnicity					
European	2,047 (62)	1,283 (38)	1.00	1.00	
Māori	345 (44)	448 (56)	0.71 (0.46–0.65)	0.80 (0.73–0.87)	
Pacific	393 (49)	414 (51)	0.79 (0.73–0.85)	0.90 (0.83–0.98)	
Asian	417 (47)	462 (53)	0.77 (0.72–0.83)	0.80 (0.74–0.86)	
Other*	120 (56)	94 (44)	0.91 (0.81–1.03)	0.93 (0.82–1.04)	
Age group					
<20	94 (31)	177 (66)	1.00	1.00	
20–29	1,152 (50)	1,169 (50)	1.46 (1.23–1.74)	1.24 (1.04–1.49)	
≥30	2,085 (61)	1,360 (39)	1.78 (1.50–2.11)	1.36 (1.14–1.62)	
Education					
Secondary level or lower	830 (47)	937 (53)	1.00	1.00	
Tertiary	2,494 (59)	1,761 (41)	1.25 (1.18–1.32)	1.14 (1.08–1.21)	
Pregnancy planning					
Unplanned	1,069 (47)	1,211 (53)	1.00	1.00	
Planned	2,250 (60)	1,485 (40)	1.28 (1.22–1.35)	1.14 (1.08–1.21)	
Parity					
First child	1,310 (51)	1,260 (49)	1.00	1.00	
Subsequent child	2,018 (58)	1,446 (42)	1.14 (1.09–1.20)	1.12 (1.07–1.18)	
Household deprivation[†]					
1 to 3 (least deprived)	949 (61)	606 (39)	1.00	1.00	
4 to 7	1,273 (56)	983 (44)	0.92 (0.88–0.98)	0.98 (0.93–1.04)	
8 to 10 (most deprived)	1,105 (50)	1,116 (50)	0.81 (0.77–0.86)	0.97 (0.91–1.03)	

*Other includes Middle Eastern, Latin American, and African.

[†]Area-level socio-economic deprivation was measured using the NZ Index of Deprivation, Dep 1,2 & 3 = least deprived, Dep 8,9 & 10 = most deprived households.¹⁵ RR – Relative Risk; CI – confidence interval.

Interactions not included in this model. Missing number of cases for: self-prioritised ethnicity (103), Age group (86), education (104), pregnancy planning (116), parity (93), household deprivation (88).

Maternal ethnicity, age, education, pregnancy planning and parity were independently associated with the duration of exclusive breastfeeding (Table 2). The likelihood of exclusive breastfeeding for ≥4 months was decreased for children of women of Māori (RR=0.80, 95% CI 0.73–0.87), Pacific (RR=0.90, 95% CI 0.83–0.98) or Asian (RR=0.80, 95% CI 0.74–0.86) compared with European ethnicity. The likelihood of

exclusive breastfeeding for ≥4 months was increased for children of women: who were aged 20–29 (RR=1.24, 95% CI 1.04–1.49) or ≥30 (RR=1.36, 95% CI 1.14–1.62) versus <20 years old; who had tertiary (RR=1.14, 95% CI 1.08–1.21) compared with secondary or less education; for whom this was a planned pregnancy (RR=1.14, 95% CI 1.08–1.21); or was their first child (RR=1.31, 95% CI 1.17–1.47).

Discussion

This is the first description of breastfeeding indicators in a New Zealand sample generalisable to the national birth cohort. Breastfeeding was initiated for 97% of children, 16% were exclusively breastfed at age six months, 37% were breastfed for ≥ 12 months and 13% for ≥ 24 months. First-born children, those from unplanned pregnancies, and those whose mothers were younger (< 20 years old) or less educated were at greater risk of having a shorter duration of any or exclusive breastfeeding. In addition, children of mothers of Māori, Pacific or Asian ethnicity were at increased risk of a shorter duration of exclusive breastfeeding. For most of the associations found, the effect size was greater than 10%, which is a clinically and statistically significant magnitude when we consider the generalisability of the cohort. According to Gigerenzer (2008),¹⁶ interpretation of the RRs is dependent on the study parameters and the outcomes under investigation.

It is important to highlight the differences in methodologies used when we compare our data describing breastfeeding in preceding months with global information on breastfeeding provided by WHO/UNICEF, which included studies that collected information describing breastfeeding in the preceding 24 hours.¹ The percentage of children ever breastfed (97%) and children breastfed for one year or beyond (37%) within our cohort was higher than average rates reported for high-income countries globally ($\sim 82\%$ and $< 20\%$, respectively).¹ However, according to the *World Breastfeeding Trend Initiative*,¹⁷ a tool developed to monitor the *WHO Global Strategy for Infant and Young Child Feeding*,³ the median duration of breastfeeding in New Zealand (seven months) ranks in the lowest quartile globally for this indicator.

Breastfeeding rates found in our study were higher or comparable to rates reported in representative surveys and cohort studies that used retrospective assessments in high-income countries.^{18–24} The proportion of children ever breastfed in our study (97%) was higher than reported for Belgium (82%),¹⁸ Canada (72%),²² the US (79%)²³ and England (70% and 76%).^{20,21} Cohort studies conducted in Australia,²⁴ and with a group

of Somali and Iraqi mothers from Norway,¹⁹ reported rates of breastfeeding initiation similar to our cohort: 91% and 93%, respectively. While comparisons are restricted by the use of different age cut-offs, the duration of any and exclusive breastfeeding were longer in our cohort than reported for England,^{20,21} the US,²³ Belgium,¹⁸ Canada,²² Norway²¹ and Australia.²⁴ Only for the indicator ‘percentage of children exclusively breastfed at age six months’ was there a higher rate reported in the US in 2011 (19%)²³ than in our cohort (16%).

Rates of breastfeeding initiation were comparable, and duration of any and exclusive breastfeeding duration were longer, in our cohort compared with two other contemporary New Zealand child cohort studies: the NZ Asthma and Allergy Cohort Study from Christchurch and Wellington (13% exclusively breastfed at 4–5 months, 31% any breastfeeding at age 12 months),²⁵ and in the Pacific Islands Families Cohort, from Auckland (9% exclusively breastfed at age six months, 29% any breastfeeding at age 12 months).²⁶ Only limited direct comparisons of our data with breastfeeding data collected routinely in New Zealand are possible. In New Zealand, breastfeeding status is reported when mothers and their young infants are discharged from their LMC, which usually occurs during the first weeks of infancy.²⁷ Because the *Plunket system* of well child care provision collects data on breastfeeding status up until the child is six months old, the median duration of breastfeeding cannot be estimated. The percentage of children exclusively breastfed at six months within our cohort (16%) was higher than that reported by the *Plunket System* in 2011 (12%).⁸

Similar to our observations, previous cohort studies have reported shorter breastfeeding^{18,20,22,24} and exclusive breastfeeding duration²² among younger mothers. In our cohort, children with older siblings were more likely to be breastfed and exclusively breastfeed for longer. Longer duration of any breastfeeding among children with siblings was also reported in cohort studies in Australia and England.^{20,24} The association observed of higher maternal education with longer duration of any and exclusive breastfeeding is consistent with observations

reported previously in Australia, Canada, England and the US.^{20,22,24,28} In addition to the experience gained from having previous children, older women have more opportunity to have completed their formal education.²⁹ Women with more education tend to have more access to family and social supports, which enable breastfeeding to be continued, for example, access to more flexible work arrangements and the capacity to return to part-time employment.²⁸

Similar to our findings, previous cross-sectional studies have reported associations between unplanned pregnancies and shorter breastfeeding duration.²⁹⁻³¹ Planning for a pregnancy is believed to involve thinking beforehand about how the baby will be fed, which can facilitate the mothers' commitment to breastfeeding.^{29,31} A cross-sectional study that included data from 18 developing countries showed that maternal attitude towards the pregnancy is an independent predictor of breastfeeding duration.³⁰ However, longitudinal studies are required to understand the causal inferences and relationships between pregnancy intentions, prenatal care behaviours and subsequent pregnancy and infancy outcomes, including breastfeeding.³⁰

Previously reported associations of maternal ethnicity with breastfeeding patterns differ from those observed in this study. We observed lower rates of breastfeeding initiation among children whose mothers were of Māori or Pacific ethnicity, and shorter duration of exclusive breastfeeding among children whose mothers were of Māori, Pacific or Asian ethnicity. These findings differ from previous cohort studies from England²⁰ and the UK¹⁹ that showed higher rates of breastfeeding initiation in non-European ethnic groups. Data from the US²⁸ and from one cohort study conducted in north England¹⁹ found no association between maternal ethnicity and exclusive breastfeeding duration. Differences in breastfeeding patterns between ethnic groups are influenced by both cultural contexts and country of residence.^{18-21,26} Further investigations on barriers to breastfeeding initiation and exclusivity among Māori, Pacific and Asian mothers within our cohort are necessary

in order to guide specific interventions for these population groups.

Breastfeeding practices are affected by historical, socioeconomic, cultural and individual factors.⁹ Improving breastfeeding practices requires supportive measures at different levels, including legal and policy directives, social support, women's employment conditions, access to healthcare and healthcare provider knowledge and skills to support breastfeeding.⁹ Future studies will investigate barriers to breastfeeding initiation and duration, with the aim to developing interventions aimed at improving breastfeeding practices in New Zealand. A meta-analysis conducted by The Lancet Breastfeeding Series Group identified that combined health system and community interventions could increase exclusive breastfeeding 2.5-fold.⁹

Strengths of our study include its antenatal recruitment and representativeness of the contemporary New Zealand birth cohort. Study weaknesses include the potential for recall bias in the description of breastfeeding duration. However, previous studies have shown that maternal recall of breastfeeding initiation and duration offers a valid and reliable estimate for recall periods of three years or less.³² A number of studies have reported the inaccuracy of EBF duration estimation when information is collected retrospectively,³²⁻³⁵ indicating that simply asking mothers how long they exclusively breastfed may not be valid.³⁶ Some authors³⁵⁻³⁷ have advocated for the use of accrual methods to evaluate extent of EBF in prospective studies. In our study, in order to minimise recall bias for EBF, we adjusted the information on reported exclusive breastfeeding duration by the reported age of introduction of solids and liquids. Although we may have captured the introduction of water to the child's diet at the six-week interview, we did not specifically ask the age of water introduction at the nine-month interview. Due to this aspect, the estimate of EBF to age six months that was adjusted for age of food introduction could still be an overestimate, potentially including together the children that were exclusively and those predominantly breastfed to age six months.

Conclusions and policy implications

The rate of breastfeeding initiation in New Zealand is favourable compared to most high-income countries and comparable to many low- and middle-income countries.¹ Currently in New Zealand a large proportion of children do not achieve the international recommendations for duration of breastfeeding or exclusive breastfeeding. Clearly New Zealand has some work to do to achieve the global nutrition target of at least 50% EBF to six months of age.³⁸ Perez-Escamilla & Sellen (2015), based on overwhelming

evidence that breastfeeding has health and economic benefits for families and society, suggest that access to breastfeeding protection and support is a human right in the context of social justice and equity.³⁹ Therefore, any social, economic, political, legal or biomedical factors that prevent women from implementing their choice and right to breastfeed need to be considered through an equity lens. When planning and evaluating the strategies necessary to support, protect and promote breastfeeding in New Zealand, it is important to take the inequalities observed in breastfeeding practices into account and target interventions to at-risk groups.

Competing interests:

Nil.

Acknowledgements:

Most importantly, we would like to acknowledge the children and the families who are part of the *Growing Up in New Zealand* study. We would also like to acknowledge the initial funders, in particular the New Zealand Ministry of Social Development, supported by the Health Research Council as well as the ongoing support from Auckland UniServices and The University of Auckland. We acknowledge all the members of the *Growing Up in New Zealand* team, including those members and managers of the operational, data, communications, community and quality aspects of the study. We thank the ongoing support and advice provided by our Kaitiaki Group and our national and international Scientific Advisory Group and we also acknowledge the members of the Morton Consortium responsible for planning and design of this study in the development phase. We thank Catherine Gilchrist for review of the manuscript.

Author information:

Teresa Castro, Paediatrics and Growing Up in New Zealand, University of Auckland, Auckland; Cameron Grant, Paediatrics, Growing Up in New Zealand and Centre for Longitudinal Research—He Ara ki Mua; Clare Wall, Discipline of Nutrition and Dietetics, University of Auckland, Auckland; Michaela Welch, Medicine, Boston University, United States; Emma Marks, Growing Up in New Zealand, University of Auckland, Auckland; Courtney Fleming, Auckland District Health Board, Auckland; Juliana Teixeira, Nutrition, University of Sao Paulo, Brazil; Dinusha Bandara, Growing Up in New Zealand, and Centre for Longitudinal Research—He Ara ki Mua, University of Auckland, Auckland; Sarah Berry, Growing Up in New Zealand, University of Auckland, Auckland; Susan Morton, Growing Up in New Zealand, and Centre for Longitudinal Research—He Ara ki Mua, University of Auckland, Auckland.

Corresponding author:

Dr Cameron Grant, Paediatrics, University of Auckland, Grafton Road, Auckland 1142.
cc.grant@auckland.ac.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7426>

REFERENCES:

1. Victora CG, Bhal R, Barros AJD, et al. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *The Lancet*. 2016; 387:475–87.
2. World Health Organization: United Nations Children's Fund. Global Strategy for Infant and Young Child Feeding. Geneva, Switzerland: World Health Organization; 2003.
3. World Health Organization: United Nations Children's Fund. Indicators for assessing infant and young child feeding practices: Part 1-Definitions. Geneva, Switzerland: World Health Organization; 2008.
4. Organisation for Economic Co-operation and Development. Doing better for children. Organisation for Economic Co-operation and Development; 2009.
5. World Health Organization. Global data bank on infant and young child feeding [online]. 2016. Available at <http://www.who.int/nutrition/databases/infantfeeding/en/> Accessed 7 September 2016.
6. Bosi ATB, Eriksen KG, Sobko T, et al. Breastfeeding practices and policies in WHO European Region Member States. *Public Health Nutr*. 2015; 19(4):753–64.
7. Ministry of Health. 2008. Food and Nutrition Guidelines for Healthy Infants and Toddlers (Aged 0–2): A background paper (4th ed) – Partially Revised December 2012. Wellington: Ministry of Health.
8. Ministry of Health. The health status of children and young people in New Zealand. Dunedin: New Zealand Ministry of Health, 2013.
9. Rollins NC, Bhandari N, Hajeebhoy N, et al. Why invest, and what it will take to improve breastfeeding practices? *Lancet*. 2016; 387:491–504.
10. Morton SMB, Ramke J, Kinloch J, Grant CC, Atatoa Carr P, Leeson H, Chi Lun Lee A, Robinson E. Growing Up in New Zealand cohort alignment with all New Zealand births. *Aust NZ J Public Health*. 2014; Online; doi: 10.1111/1753-6405.12220
11. Morton SMB, Atatoa Carr P, Grant CC, et al. Growing Up in New Zealand: A longitudinal study of New Zealand children and their families. Now we are Two: Describing our first 1000 days. Auckland: Auckland: Growing Up in New Zealand, 2014.
12. Morton SM, Ramke J, Kinloch J, et al. Growing Up in New Zealand cohort alignment with all New Zealand births. *Aust NZ J Public Health*. 2015; 39(1):82–87.
13. World Health Organization (1991) Indicators for Assessing Breast Feeding Practices. WHO/CDD/SER/91.14. Geneva:WHO.
14. Wall CR, Brunt DR, Grant CC. Ethnic variance in iron status: is it related to dietary intake? *Public Health Nutr*. 2009; 12:1413–1421.
15. Salmond C, Crampton P, Atkinson J (2007). NZDep2006 Index of Deprivation. Wellington. University of Otago.
16. Gigerenzer G, Gaissmaier W, Kurz-Milcke E, Schwartz LM, Woloshin S. Helping Doctors and Patients Make Sense of Health Statistics. *Psychol Sci Public Interest*. 2007; 8(2):53–96.
17. Baby Friendly Hospital Initiative Hong Kong Association: United Nations Children's Fund. The World Breastfeeding Trends Initiative (WBTi). Hong Kong, China; 2012.
18. Robert E, Coppieters Y, Swennen B, Dramaix M. Breastfeeding duration: A survival analysis- Data from a Regional Immunization Survey. *Biomed Research International*. 2014; doi: 10.1155/2014/529790.
19. Santorelli G, Petherick E, Waiblinger D, et al. Ethnic differences in the initiation and duration of breast feeding-Results from the Born in Bradford Birth Cohort Study. *Paediatr Perinat Epidemiol*. 2013; (27):388–92.
20. Griffiths LJ, Tate AR, Desateux C and the Millennium Cohort Study Child Health Group. The contribution of parental and community ethnicity to breastfeeding practices: evidence from the Millennium Cohort. *Int J Epidemiol*. 2005; 34:1378–86.
21. Grewal NK, Andersen LF, Sellen D, et al. Breastfeeding and complementary feeding practices in the first 6 months of life among Norwegian-Somali and Norwegian-Iraqi infants: the InnBaKost survey. *Public Health Nutr*. 2015; 19(4):703–15.
22. Dubois L, Girard M. Social determinants of initiation, duration and exclusivity of breastfeeding at the population level: the results from the Longitudinal Study of Child Development in Quebec (ELDEQ 1998–2002). *Can J Public Health*. 2003; 94(4):300–5.
23. Centers for Disease and Control. Breastfeeding Report Card- United States/2014. Atlanta: CDC, 2014.
24. Baxter J, Cooklin AR. Which mothers wean their babies prematurely from full

- breastfeeding? An Australian cohort study. *Acta Paediatr.* 2009; 98:1274–7.
25. Silvers KM, Frampton CM, Wickens K, et al. Breastfeeding protects against adverse respiratory outcomes at 15 months of age. *Matern Child Nutr.* 2009; 5:243–50.
 26. Schluter PJ, Carter S, Percival T. Exclusive and any breast-feeding rates of Pacific infants in Auckland: data from the Pacific Islands First Two Years of Life study. *Public Health Nutr.* 2006; 9(6):692–99.
 27. Ministry of Health. 2012. Report on Maternity, 2010. Wellington: Ministry of Health.
 28. Pitonyak JS, Jessop AB, Pontiggia L, Crivelli-Kovach A. Life course factors associated with initiation and continuation of exclusive breastfeeding. *Matern Child Health J.* 2016; 20:240–9.
 29. Houghton J, Gregorio D, Perez-Escamilla R. Factors associated with breastfeeding duration among Connecticut Special Supplemental Nutrition Program for Women, Infants and Children (WIC) participants. *J Hum Lact.* 2010; 26(3):266–73.
 30. Hromi-Fiedler A, Perez-Escamilla R. Unintended pregnancies are associated with less likelihood of prolonged breast-feeding: an analysis of 18 Demographic and Health Surveys. *Public Health Nutr.* 2006; 9(3):306–12.
 31. Taylor JS, Cabral HJ. Are women with an unintended pregnancy less likely to breastfeed? *J Fam Pract.* 2002; 51:431–6.
 32. Li R, Scanlon KS, Serdula MK. The validity of maternal recall of breastfeeding practice. *Nutrition Reviews;* 63(4):103–10.
 33. Burhnam L, Buczek M, Braun N, Feldman-Winter L, Chen N, Merewood A. Determining length of breastfeeding exclusivity: validity of maternal report 2 years after birth. *J Hum Lact.* 2014; 30(2):190–4.
 34. Gillespie B, d'Arcy H, Schwartz K, Bobo JK, Foxman B. Recall of age of weaning and other breastfeeding variables. *Int Breastfeed J.* 2006; 9:1–4.
 35. Agampodi SB, Fernando S, Dharmaratne SD, Agampodi TC. Duration of exclusive breastfeeding; validity of retrospective assessment at nine months of age. *BMC Pediatrics.* 2011; 14:80. doi: 10.1186/1471-2431-11-80.
 36. Greiner T. Exclusive breastfeeding: measurement and indicators. *Int Breastfeed J.* 2014; 20(9):18. doi: 10.1186/1746-4358-9-18.
 37. Abdel-Hady DM, El-Gilany A. Calculating exclusive breastfeeding rates: comparing dietary “24-hour recall” with recall “since birth” methods. *Breastfeed Med.* 2016; 11(10):514–8.
 38. World Health Organization: United Nations Children’s Fund. Global Nutrition Targets 2025- Breastfeeding Policy Brief. Geneva, Switzerland: World Health Organization; 2014.
 39. Perez-Escamilla, Sellen D. Equity in breastfeeding: where do we go from here? *J Hum Lact.* 2015; 31(1):12–4.

Teaching quality improvement to medical students: over a decade of experience

Michelle R Wise, Bridget Kool, Lynn Sadler, Roshini Peiris-John, Gillian Robb, Susan Wells

ABSTRACT

AIM: To describe how we incorporate experiential quality improvement (QI) learning at the University of Auckland by integrating a clinical audit project into the Year 6 obstetrics and gynaecology clinical attachment.

METHODS: Students gain insight into the relevance of QI while engaged in day-to-day clinical work. Students work with a clinical supervisor to identify an area for potential improvement, set a standard of care, measure current practice, investigate reasons for deviation from the standard and make real-world suggestions to close the gap between best evidence and observed practice.

RESULTS: Since 2004, over 1,250 projects have been completed, and two journal articles published. Many of the student projects result in actual improvements to clinical processes of care, and lead to strengthening of academic and service provider learning networks and partnerships.

CONCLUSIONS: Performing a hands-on project within the constraints and context of a busy women's health service is a feasible and effective method of teaching QI. Medical schools have an integral role to play in ensuring future healthcare professionals are equipped with QI knowledge, skills and attitudes. Experiential QI learning enhances clinical teaching and training, and is important in preparing future clinicians to incorporate QI into their daily practice.

Clinician engagement in ongoing health service quality improvement (QI) is an important component of improving clinical outcomes. Integrating QI in undergraduate and postgraduate medical education to equip clinicians with the knowledge and skills to improve care is increasingly acknowledged as important. A number of international medical accreditation bodies now include QI as a core competency, necessitating medical education programmes to develop authentic curricula in this area.¹⁻⁴ The New Zealand and Australian Curriculum Framework for Junior Doctors includes clinical audit as a core element of all pre-vocational training.^{5,6} Like many other professional colleges, clinical audit is also a mandatory requirement set out by the Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG).⁷

A growing number of universities worldwide are offering QI learning opportunities to medical learners, although there is significant heterogeneity in educational content, teaching methods, students targeted and learning outcomes.⁸ Undergraduate curricula vary from a few lectures (the majority involving fewer than 10 contact hours and often consisting of a single session) to the undertaking of a QI project.⁹ Postgraduate curricula targeting junior doctors often involve multiple encounters within existing core rotations, standalone sessions or elective rotations with participation in QI projects.^{8,10} The primary objective in most QI curricula is learning the principles and methods of QI, with common educational content including QI science and skills, safety incident analysis and systems thinking.⁸ Assessments of QI

curricula have reported improvements in learner's knowledge and self-efficacy, with lesser emphasis on changes in behaviour and clinical process.⁸ Incorporating experiential learning that is fully integrated into day-to-day clinical work has been suggested as a way to improve generalisability to future practice settings and lead to change in clinical practice.¹¹

To date, the most promising form of experiential learning in undergraduate QI curricula combines classroom learning with practical projects.¹² The QI projects commonly include elements of root-cause or safety incident analysis, a chart audit and reflective practice.¹³ Students are assigned to interprofessional QI teams^{14,15} or to community settings,¹⁶⁻¹⁸ or undertake mentored projects.¹⁹ Difficulties in achieving a balance between both didactic and experiential learning exist amidst competing educational demands of medical students. However, the QI project approach is consistent with adult learning principles, where learners are engaged in actual QI practice and the projects have the potential for improving clinical processes of care.¹³

Typical QI projects require iterative cycles of implementation and evaluation. However, longitudinal learning attachments and integrating QI learning into day-to-day clinical work are often not feasible.¹¹ The competing educational demands on medical students require projects to be kept simple and workload appropriate.²⁰ The aim of this paper was to describe how we incorporate experiential QI learning at the University of Auckland by integrating a clinical audit project into the Year 6 obstetrics and gynaecology clinical attachment.

Methods

At the University of Auckland, we provide classroom and web-based QI science and skills learning during the six-year undergraduate medical curriculum. Since 2004, we have also incorporated experiential QI learning by integrating a clinical audit project into final-year medical students' obstetrics and gynaecology (O&G) clinical attachment across nine hospitals.

The improvement sciences theory and skills learning commences in Year 3 where the focus is on patient safety. Medical

students are allocated into interprofessional teams with third-year nursing, pharmacy and optometry students for a two-day module which consists of face-to-face lectures covering teamwork in healthcare, understanding healthcare disparities, error and violation, human factors (such as practitioner fatigue) and measuring intervention impacts (include revenge effects). The module culminates in conducting a root cause analysis following an adverse event, and the process of open disclosure. Students in Year 4 have six hours of lectures on the critical appraisal of epidemiological evidence. In Year 5, students complete an eight-hour online self-directed module on clinical audit and improvement frameworks, sampling and measurement strategies, and QI science tools to enable them to display, describe and learn from clinical data.

In Year 6, students apply their improvement science skills to conduct a clinical audit project on a topic of their choice during their clinical attachment in O&G. The Year 6 QI programme is designed to help students view a health service through the lens of QI dimensions and the concepts of harm, waste and variation. Starting with the research evidence for best practice, students follow the audit cycle (see Figure 1).

Working with a clinical supervisor, the students identify an area for potential improvement and set an evidence-based standard of care. They conduct part of a simple clinical audit to measure current practice and investigate reasons for any deviation from the standard. Students put into practice basic approaches to measurement in QI, including identifying appropriate measures and sampling methods, collecting, analysing and interpreting data, and describing and displaying variation. Students work alone or in pairs and are allocated one academic half-day per week to complete the project. Students are encouraged to discuss their findings with the relevant stakeholders to inform their interpretation of the findings and to develop a suggested action plan. Although the QIPs are carried out during a short timeframe, Year 6 students do learn QI skills beyond conducting a clinical audit. Students are expected to discuss their findings with the relevant service providers, interpret their

Figure 1: The clinical audit cycle.



Figure credit: Health Quality and Safety Commission, New Zealand.

findings by describing the potential root causes and how they fit in the context of the service, and provide change ideas and recommendations of possible solutions that the service could implement. They also identify areas for further investigation. The students present to the local clinical team for discussion. We are aware of many service changes that have been made by clinicians as a result of the student presentations over the years. We then encourage future students to re-audit to check if the change/s has resulted in any improvement, thus closing the loop. The projects are formally assessed based on a written report and an oral presentation to their peers, QI faculty and where possible the clinical team.

Results

As at July 2016, over 1,250 projects have been completed, with two publications arising from this work so far incorporating multiple audits from multiple sites,^{21–22} and a thesis dissertation. The projects cover a broad range of obstetrics and gynaecology topics. They range from being condition-specific (eg, adherence to clinical indications for elective caesarean section before 39 weeks; prophylactic Anti-D administration for Rhesus (D) negative pregnant women) to general processes of care and practice (eg, adherence to handwashing policies; screening for family violence on admission). The topics chosen most often by students

over the last 14 years include post-operative thromboprophylaxis (n=39 projects), induction of labour (n=53), colposcopy (n=98) and antenatal screening (for infections, congenital anomalies, gestational diabetes, anaemia, etc.) (n>100). A clinical audit project example is provided in Table 1.

A formal evaluation of the QI Programme was conducted in 2014 when a shorter clinical rotation in O&G (five weeks to four) was to be implemented. Details of the formal evaluation are reported elsewhere.²³ This evaluation was funded by a University of Auckland Learning Enhancement Grant. In summary, the QI programme was seen by clinicians as a valuable way for Year 6 medical students to apply their knowledge and skills into practice, by performing a hands-on project within the constraints and context of a busy women's health service. Additionally, most clinicians agreed the QIP has provided value to the O&G service and the organisation. Students reported gaining useful insights into QI and agreed that the skills learnt would be important for their future.

However, the evaluation revealed that there was significant variation in data collection and in programme implementation across the eight teaching sites. In addition, challenges were faced by students in selecting a topic that fulfilled project criteria, was relevant to the clinical supervisor and/or the service, and was

Table 1: Example of a clinical audit project.

Topic selection	Preterm birth complicates 7% of New Zealand births, and accounts for 85% of perinatal morbidity and mortality; antenatal corticosteroids reduce adverse outcomes
Standard of care	Criterion (antenatal corticosteroids are given to women at risk of preterm birth 24+0–34+6 weeks); target (100%); allowable exceptions (imminent birth)
Survey current practice and compare against standard	Process indicator (receipt of antenatal corticosteroids); study population (every second woman who gave birth from 24+0–34+6 in 2013 at Auckland Hospital, exclusion: if patient declined); sample size (106); variables (type of maternity caregiver, gestational age)
Display and interpret findings	85% of women received antenatal corticosteroids; presented data using a pareto diagram and an Ishikawa diagram
Develop an action plan	Local teaching sessions on clinical guidelines; add clinical guideline to orientation of new staff; link to clinical guideline within electronic medical record; add date/time of antenatal corticosteroid doses to electronic medical record; ensure clear documentation of gestational age

achievable in the time frame. The findings of the evaluation led to the development and implementation of project-wide and site-specific solutions to ensure learning outcomes continue to be met. This has been largely achieved by developing new resources and making processes more efficient. All resources are now easily accessible to students and supervisors through the university education online portal, such as Year 5 teaching resources, a how-to guide, all previous project reports and a discussion board of ‘tips and hints’ from previous students. A fifteen minute ‘QI Project Overview’ video was developed, highlighting the key components of the clinical audit, the process to follow, timelines and expectations. A ‘topic selection form’ was developed to help ensure students select an appropriate topic and methodology (eg, standard, sample size, variables, etc.) and requires signoff from the supervisor. A ‘written report template’ was created to ensure reports are concise and relevant to stakeholders. Clinical supervisors at each hospital keep an updated list of potential topics, and have created a repository for written reports accessible to hospital clinicians. Hospital QI staff established processes to enable quicker and easier access to medical records.

In advance of the attachment, a welcome email was sent to students providing a link to a university education online portal. The key process steps are summarised below:

- Week 1: Orientation and topic selection
 - Students and supervisors watch “QI project overview” video, and review available resources.
 - Students receive an encrypted memory stick (to protect patient privacy) for data collection.
 - Students meet with supervisor, identify topic and complete Topic Selection Form.
- Week 2–3: Conduct project
 - Supervisor signs off Topic Selection Form with specific feedback to students.
 - Students collect and analyse data.
 - Students discuss findings with stakeholders and brainstorm barriers and enablers.
- Week 4: Complete project
 - Students prepare and submit three-page written report.
 - Students prepare and deliver a 15-minute oral presentation and answer questions.

The QI curriculum meets the University of Auckland graduate learning outcome for students to identify feasible strategies to improve health that incorporate the broader determinants of health at community and population level.²⁴ It also meets the Australasian Medical Council (AMC) accreditation

standard regarding QI, which requires students to be able to describe a systems approach to improving the quality and safety of healthcare.³ The 2005 AMC report noted *“the team was impressed especially by the root cause analysis of sentinel events undertaken in Year 3 in a multidisciplinary training environment, and the Year 6 quality assurance project.”*

We are committed to disseminating what we have learned through the delivery of our QI teaching programme. Recent efforts have included presentations at local faculty medical education rounds, and facilitation of a QI teaching workshop at the 2014 AMEE (An International Association for Medical Education) conference in Milan, Italy, by two of the authors (MRW and BK). We have appointed a part-time research coordinator (RP-J) to assist with resource development, research outputs and collaboration across all sites.

Discussion

Performing a hands-on project within the constraints and context of a busy O&G service is a feasible and effective method of teaching QI and provides value to the health service. Experiential QI learning, as described here, enhances clinical teaching and training. By integrating clinical audit projects within clinical attachments, medical schools can equip future clinicians with the necessary knowledge and skills to incorporate QI into their daily practice and improve clinical practice.

The Institute of Medicine’s ‘Learning Healthcare System’ report described an approach for integrating clinical research and clinical medicine.²⁵ This has subsequently evolved into a broader concept of a learning health system where all stakeholders can securely, effectively and efficiently contribute, share and use data to create knowledge and improve health outcomes. We plan to support a learning network of clinical supervisors and university academics with a view to enhancing QI training, benchmarking service performance across regional hospitals, sharing ideas for improvement and engaging in academic research.

The importance of developing capability of clinical trainers in QI, transforming them

into teachers of QI, has been previously highlighted as a way forward in improving QI in medical education.¹³ The learning network will facilitate this. We are building faculty capacity by involving more clinicians in student projects, and providing support from the academic department and hospital QI staff to guide them in supervising students to learn the required skills. Specifically, when clinicians first become involved in the project, we provide one-on-one tutoring in clinical audit methodology and mentor them with assessment, by co-evaluating student reports and presentations. We then provide ongoing feedback on the topics, helping them appropriately frame the topic as audit (not research). Informal feedback has been that clinicians feel more confident as time goes on to be able to provide students with adequate support and guidance. As they start using the students’ findings to drive implementation of solutions to improve service, they become more aware of the utility of the student projects, and thus motivated to become more involved in future projects. An unexpected finding in our formal evaluation of the QI programme was that clinicians felt they were learning QI skills from the students. This is important as the New Zealand Medical Council requires all clinicians to perform an annual clinical audit of their own medical practice in order to recertify.²⁶

Together with clinician supervisors, we are also working towards documenting changes in clinical processes and patient outcomes that occurred as a result of student projects. Future research could look at whether focus on specific topics could impact the quality of care delivered in O&G, as was shown in improved health outcomes for patients with diabetes at community practices where medical students and their preceptors completed QI projects.¹⁶

Although students are encouraged to learn and apply QI principles beyond conducting a clinical audit, we acknowledge that it is challenging to achieve improvements in service and patient outcomes using small projects conducted in short blocks. That being said, we have worked closely with clinicians over the years to direct the student reports to the clinicians who have governance over the areas studied, and to engage with hospital-wide QI departments,

in order to facilitate using the findings to improve service. One way to overcome this could be the team-based approach adopted by some postgraduate programmes in which individual junior doctors work on one part of an overall project, building on work done by their predecessors and a quality initiative over time.^{27,28}

Our QI programme is undergoing continuous improvement. We continue to look for ways to modify the clinical audit project in order to reduce student workload. Our supervisors encourage students to choose only one standard, one or two measures, and a few stratifying variables; we are considering assessing the project proposal only, rather than the completed project; and we are exploring with other departments to collaborate with O&G to enable the clinical audit to be conducted over more than one placement, which would extend the project timeframe. We also want to engage clinicians at more sites to improve knowledge translation, for example, getting the findings from student projects back out to stakeholders who can use them to make a difference in their services. Clinicians who see improvements in patient outcomes will be more likely to seek out continuing QI education for themselves and to supervise future students.

Another challenge we are addressing is that of assessment. At the moment individual students are assessed for their QI competency by evaluating their group

project presentation. There may be more effective ways of assessing competency and we may add other objective measures of student knowledge such as the QIKAT-R,²⁹ or modify our assessment scheme to a validated tool.³⁰ We plan to survey postgraduate trainees to assess retention of QI knowledge and skills, and investigate whether the projects increased QI capability so that learners were able to improve health service performance in their future roles. The survey will also gauge whether involvement in these projects enhanced exposure to rigorous research methods and structured reporting. As previously reported, this may potentially inspire health professionals to be engaged with academic medicine during their career.³¹

Medical schools have an integral role to play in ensuring future healthcare professionals are equipped with QI knowledge, skills and attitudes so that they can contribute towards improved clinical practice and patient outcomes. We provide a combination of didactic and experiential QI learning opportunities. The literature notes numerous benefits of experiential learning, although learning real-life QI practices is comparatively new. Incorporating clinical audit projects within short clinical attachments should be considered a feasible and effective method of QI education to medical students. The authors are happy to be contacted to share resources, consult with faculty or facilitate workshops.

Competing interests:

Dr Wells reports grants from Health Research Council of New Zealand, grants from National Heart Foundation of New Zealand, grants from The Stevenson Foundation, grants from Roche Diagnostics Ltd, outside the submitted work.

Acknowledgements:

Prof Peter Stone and A/Prof John Buchanan for having the foresight to embed QI teaching in the undergraduate medical curriculum at University of Auckland; Michelle Carvalho for her ongoing administrative support of the medical students in the Department of Obstetrics and Gynaecology; Faith Mahony for her assistance in conducting the 2014 formal evaluation of the QI programme; and Year 6 medical students Prasadi Jayasinghe and Cathy Zhong, on whose 2014 clinical audit project formed the example provided in the paper.

Author information:

Michelle Wise, Epidemiology and Biostatistics, School of Public Health, FMHS, University of Auckland, Auckland; Bridget Kool, Epidemiology and Biostatistics, School of Public Health, FMHS, University of Auckland, Auckland; Lynn Sadler, Epidemiology and Biostatistics, School of Public Health, FMHS, University of Auckland, Auckland;
Roshini Peiris-John, Epidemiology and Biostatistics, School of Public Health, FMHS, University of Auckland, Auckland; Gillian Robb, Epidemiology and Biostatistics, School of Public Health, FMHS, University of Auckland, Auckland;
Susan Wells, Epidemiology and Biostatistics, School of Public Health, FMHS, University of Auckland, Auckland.

Corresponding author:

Dr Michelle R Wise, Senior Lecturer, Department of Obstetrics and Gynaecology, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, Auckland 1142.
m.wise@auckland.ac.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7427>

REFERENCES:

1. Accreditation Council for Graduate Medical Education. Common Program Requirements: General Competencies. Chicago, Illinois; 2007.
2. General Medical Council. Tomorrow's Doctors London: 2009 [cited 2016 Aug 29]. Available from: http://www.ub.edu/medicina_unitateducaciomedica/documentos/Tomorrows-Doctors_2009.pdf
3. Australian Medical Council. Standards for assessment and accreditation of primary medical programs. Kingston; 2012.
4. Frank JR, Snell L, Sherbino J, editors. CanMEDS 2015 Physician Competency Framework. Ottawa: Royal College of Physicians and Surgeons of Canada; 2015.
5. Graham IS, Gleason AJ, Keogh GW, et al. Australian Curriculum Framework for Junior Doctors. *Med J Aust.* 2007; 186(7 Suppl):S14–9.
6. Medical Council of New Zealand. New Zealand Curriculum Framework for Prevocational Medical Training. Wellington; 2014 [cited 2016 February 16]. Available from: <http://www.mcnz.org.nz/assets/News-and-Publications/NZCF26Feb2014.pdf>
7. Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG). The CPD Curriculum Framework [cited 2017 April 22]. Available from: http://www.ranzcog.edu.au/RANZCOG_SITE/media/RANZCOG-MEDIA/CPD/CPD-Framework-Table-ON-LINE-RANZCOG-website.pdf
8. Wong BM, Etchells EE, Kuper, et al. Teaching quality improvement and patient safety to trainees: a systematic review. *Acad Med.* 2010; 85(9):1425–39.
9. Windish DM, Reed DA, Boonyasai RT, et al. Methodological rigor of quality improvement curricula for physician trainees: a systematic review and recommendations for change. *Acad Med.* 2009; 84(12):1677–92.
10. Jones AC, Shipman SA, Ogrinc G. Key characteristics of successful quality improvement curricula in physician education: a realist review. *BMJ Qual Saf.* 2015; 24(1):77–88.
11. Wong BM. Reporting on Patient Safety and Quality Improvement Education: Designing Projects for Opti-

- mal Dissemination. *J Grad Med Educ.* 2015; 7(4):513–6.
12. Health Foundation. Evidence scan: quality improvement training for healthcare professionals. London, UK; 2012.
 13. Wong BM, Levinson W, Shojanian KG. Quality improvement in medical education: current state and future directions. *Med Educ.* 2012; 46(1):107–19.
 14. Chessman AW, Bellack JP, Lahoz MR, et al. Students add value to learning organizations: the Medical University of South Carolina experience. *Qual Manag Health Care.* 1998; 6:38–43.
 15. Hall LW HL, Cox KR, Deane K, et al. Linking health professional learners and health care workers on action-based improvement teams. *Qual Manag Health Care.* 2009; 18:194–201.
 16. Gould BE, Grey MR, Huntington CG, et al. Improving patient care outcomes by teaching quality improvement to medical students in community-based practices. *Acad Med.* 2002; 77(10):1011–8.
 17. Knapp M, Bennett NM, Plumb J, Robinson J. Community-based quality improvement for the health professions: balancing benefits for communities and students. *J Interprofessional Care.* 2000; 14:119–30.
 18. Mak DB, Plant AJ. Reducing unmet needs: a pre-vocational medical training program in public health medicine and primary health care in remote Australia. *Aust J Rural Health.* 2005; 13:183–90.
 19. Tartaglia KM, Walker C. Effectiveness of a quality improvement curriculum for medical students. *Med Educ Online.* 2015; 20.
 20. Gould BE, O’Connell MT, Russell MT, et al. Teaching quality measurement and improvement, cost-effectiveness, and patient satisfaction in undergraduate medical education: the UME-21 experience. *Fam Med.* 2004; 36:Suppl:S57–62.
 21. Wise M, Sadler L, Ekeroma A. Chlamydia trachomatis screening in pregnancy in New Zealand: translation of national guidelines into practice. *J Prim Health Care.* 2015; 7(1):65–70.
 22. Lilic N, Jayanatha K, Stone P. Routine postnatal use of the Kleihauer-Betke test for anti-D administration in rhesus D-incompatible pregnancies: a medical student quality improvement project. *Jt Comm J Qual Patient Saf.* 2013; 39(7):324–7.
 23. Kool B, Wise MR, Peiris-John R, Sadler L, Mahony F, Wells S. Is the delivery of a quality improvement education programme in obstetrics and gynaecology for final year medical students feasible and still effective in a shortened time frame? *BMC Med Educ.* 2017; 17(1):91.
 24. Faculty of Medical and Health Sciences. Phase 3 Guidebook Auckland: Univeristy of Auckland; 2015 [cited 2016 August 29]. Available from: <http://mbchb.auckland.ac.nz>
 25. Institute of Medicine. The Learning Healthcare System: Workshop Summary. Washington, DC; 2007.
 26. Medical Council of New Zealand. Recertification and continuing professional development booklet. Wellington; 2014.
 27. Neuspiel D, Hyman D, Lane M. Quality improvement and patient safety in the paediatric ambulatory setting: current knowledge and implications for residency training. *Pediatr Clin North Am.* 2009; 56:935–51.
 28. Tess AV, Yang JJ, Smith CC, et al. Combining Clinical Microsystems and an Experiential Quality Improvement Curriculum to Improve Residency Education in Internal Medicine. *Acad Med.* 2009; 84:326–34.
 29. Singh MK, Ogrinc G, Cox KR, et al. The Quality Improvement Knowledge Application Tool Revised (QIKAT-R). *Acad Med.* 2014; 89(10):1386–91.
 30. Leenstra JL, Beckman TJ, Reed DA, et al. Validation of a method for assessing resident physicians’ quality improvement proposals. *J Gen Intern Med.* 2007; 22(9):1330–4.
 31. Lawson McLean A, Saunders C, Velu PP, et al. Twelve tips for teachers to encourage student engagement in academic medicine. *Med Teach.* 2013; 35:549–54.

Deaf New Zealand Sign Language users' access to healthcare

Joanne Witko, Pauline Boyles, Kirsten Smiler, Rachel McKee

ABSTRACT

AIMS: The research described was undertaken as part of a Sub-Regional Disability Strategy 2017–2022 across the Wairarapa, Hutt Valley and Capital and Coast District Health Boards (DHBs). The aim was to investigate deaf New Zealand Sign Language (NZSL) users' quality of access to health services. Findings have formed the basis for developing a 'NZSL plan' for DHBs in the Wellington sub-region.

METHODS: Qualitative data was collected from 56 deaf participants and family members about their experiences of healthcare services via focus group, individual interviews and online survey, which were thematically analysed. Contextual perspective was gained from 57 healthcare professionals at five meetings. Two professionals were interviewed, and 65 staff responded to an online survey. A deaf steering group co-designed the framework and methods, and validated findings.

RESULTS: Key issues reported across the health system include: inconsistent interpreter provision; lack of informed consent for treatment via communication in NZSL; limited access to general health information in NZSL and the reduced ability of deaf patients to understand and comply with treatment options. This problematic communication with NZSL users echoes international evidence and other documented local evidence for patients with limited English proficiency.

CONCLUSION: Deaf NZSL users face multiple barriers to equitable healthcare, stemming from linguistic and educational factors and inaccessible service delivery. These need to be addressed through policy and training for healthcare personnel that enable effective systemic responses to NZSL users. Deaf participants emphasise that recognition of their identity as members of a language community is central to improving their healthcare experiences.

International studies show that deaf sign language users encounter barriers to healthcare and have worse outcomes than the general population. Contributing factors are limited health literacy,¹ health practitioners' unfamiliarity with the implications of deafness and the background of sign language users, and insufficient provision of sign language interpreters within healthcare systems.^{2–4} Most sign language users have been deaf since infancy, and the resulting disruption to language acquisition typically has far-reaching developmental and educational impacts. Internationally, the prevalence of pre-lingual deafness is about 7:10,000.⁵ The deaf NZSL community is estimated at approximately 4,500.⁶ In New Zealand prior to 1980, sign language was censured by schools and society as a means

of communication. Intensive pedagogical focus on the mastery of speech was at the expense of a comprehensive education for many children.⁷ Deaf children tended to sign to each other and thus NZSL began as an underground language, which has developed through intergenerational networks of deaf people who claim a cultural identity.⁸ Today, human rights measures—particularly the United Nations Convention on the Rights of Persons with Disabilities (UNCRPD, 2009)—have led the education system to recognise the importance of sign language to deaf people's access to society, yet not all deaf children have timely access to NZSL, and educational disadvantage persists for this population.^{9,10} Childhood deafness restricts incidental learning of common cultural knowledge that hearing children absorb

through conversation and media. Exclusion from spoken information is compounded by literacy delays associated with pre-lingual deafness,¹⁰ and literacy itself is strongly associated with the capacity to make use of health-related information.¹¹ Restricted access to communication throughout a deaf person's life can result in a 'fund-of-information-deficit'¹ that compromises healthcare outcomes.¹² For example, family medical history is a risk factor for various conditions, but many deaf people have been excluded from conversations about health issues among family members. Deaf people with limited health literacy exercise less autonomy in healthcare settings, may have limited understanding of healthcare delivery systems and are less compliant when they do not understand physician instructions. This results in negative outcomes and higher costs for patients and providers.¹⁵ Compromised access to the determinants of health—employment, social networks, participation in local community and society¹⁶—and systemic barriers to accessing health information and services contribute to poor health and wellbeing outcomes for deaf people.¹⁷ For example, a UK study found deaf people had above average rates of risk factors for cardiovascular disease, hypertension and diabetes, and high rates of self-reported depression.² Deaf people may delay using primary health services due to communication difficulties, and instead present to emergency or specialist services when a condition is worse. A US study found that deaf adults are less likely to visit a doctor, due in part to dissatisfaction with communication and their perception that hospital emergency departments (ED) provide better accessibility than primary care doctors.¹⁸

Effective communication in healthcare settings contributes to good patient outcomes, and this requires recognition of linguistic and cultural differences.¹⁷ A Dutch study of communication between deaf people and healthcare staff revealed that professionals were generally unaware of linguistic and cultural differences associated with being deaf, assuming spoken Dutch to be the preferred language of deaf patients when it was actually sign language, and practitioners rated the quality of communication more positively than their deaf patients.⁴ A UK survey found 77% of British

Sign Language users reported difficulties communicating with hospital staff, and 33% left consultations with their family doctor unsure about medication instructions and subsequently took the wrong dose. Many deaf people were reluctant to admit they had not understood their GP, which may explain why 87% of GPs in this study believed that they could communicate effectively with their deaf patients through lip-reading.¹⁹ In fact, lip-reading requires proficiency in the spoken language, and the capacity to lip-read is reduced by factors such as being ill, tired, anxious, poor lighting, facial hair or hands partially covering the lips, or visual barriers (eg, a partition).¹⁷

Patient comprehension underpins compliance with medical instructions, and interpreters can play a critical role in this. A US study found that the use of professional interpreters in consultations resulted in higher patient compliance and engagement in preventative programmes.²⁰ A UK study of deaf and hearing impaired people's communication preferences in hospital consultations found that 50% preferred an interpreter, whereas 43% preferred a consultation with a signing health professional if available, and 7% agreed to accept communication using speech, as long as the doctor was 'deaf aware'.²¹ Deaf people often go without interpreters at health appointments because they are difficult to find at short notice,⁴ or an interpreter is not offered. A recent Australian study reports hospitals not consistently providing interpreters, especially in emergency departments, although the need for a qualified interpreter in this situation is high. In the absence of an interpreter, communication often relies on writing, gesturing, lip-reading or mediation by family and friends.²² However, research suggests that around 50% of the information translated by an unqualified interpreter or family member is misinterpreted or omitted, leading to poor comprehension and reduced compliance with medication instructions.¹⁹ Inadequate interpretation is compounded by deaf individuals' gaps in health knowledge and limited ability to use written sources of information.²³ Poor communication can lead to an increase in invasive and unnecessary procedures that increase the risk of complications and length of inpatient stays.²⁴

The World Federation of the Deaf advocates that access to sign language is fundamental to realising human rights for deaf citizens.²⁵ Recognition of this right underpins the intent of the NZSL Act 2006² The UNCRPD (Article 25) protects the right of persons with disabilities to access the highest attainable standards of health service provision and care without discrimination.²⁶ New Zealand has ratified this convention and is accordingly obliged to implement (on an incremental basis) these measures in domestic law and policy. A 2003 cabinet paper on the NZSL Bill noted policies for DHBs must be comprehensive, specify the use of qualified NZSL interpreters and that DHB services and information are accessible.²⁷ A 2013 Human Rights Commission enquiry into barriers for NZSL users found that while two-thirds of DHBs had policies for provision of interpreters, a reluctance of DHBs to book interpreters (for various reasons) was reportedly common.²⁸ This was regarded as a breach of the Code of Health and Disability Services Consumers' Rights which guarantees the right to effective communication, to receive full information, to make an informed choice and to give informed consent, and to have cultural needs taken into account.²⁹ A key recommendation was that DHBs develop comprehensive NZSL interpreting and translation policies giving greater visibility to existing expectations (previously noted) for all DHBs.²⁸ The NZSL Board Action plan (2016–2018) lists 'access to services and information in NZSL' as a priority, and will monitor the existence of NZSL policies within 'core government information and services' such as DHBs.³⁰

Methods

The objectives of the study were to (i) identify accessibility issues from the perspective of deaf NZSL users in general, mental health and addiction secondary services; (ii) document the perspectives of healthcare personnel working with deaf people; (iii) use the findings as a basis for developing NZSL policy for the sub-region. The research project team, under the leadership of the Director of Disability Strategy and Performance, included a mental health portfolio lead, and a project expert lead

contracted by the Service Integration and Development Unit who is a hearing NZSL user (the first author). A steering group comprising mainly deaf consumer representatives provided cultural perspective on data collection methods, analysis and recommendations. Third and fourth authors provided support with methodology, analysis and reporting. Ethical approval was obtained as per Capital and Coast District Health Board policy.³

Qualitative methods were used to enquire into how deaf people experience healthcare services, and how healthcare staff perceive their interactions with deaf people. Data collection from deaf people and their family members was mainly through focus groups (32 participants) and individual interviews (nine participants), facilitated in NZSL by hearing and deaf NZSL users. A semi-structured interview schedule encouraged discussion of personal experiences of health services; a qualified NZSL interpreter translated from NZSL to spoken English within the group discussion. This was audio recorded and later transcribed for analysis. Fifteen responses to an online survey provided supporting data.

Healthcare staff were invited to discuss their interactions with deaf NZSL users. Since this is not a frequent encounter for most professionals, the project lead raised awareness of the topic by facilitating several team education and training sessions. Various departments, managers and individuals known to have experience of working with deaf people were invited. In total, 57 professional staff attended five meetings where the topic of NZSL users in health services was discussed, eliciting perspective from those participants. Two professionals were subsequently interviewed, and 65 staff responded to an online survey.

The first and third authors independently read the transcribed interviews, survey data and notes to develop a thematic coding scheme, which identified recurring issues. This article reports key findings about deaf NZSL users' interactions with healthcare staff and systems. Due to space constraints, we do not discuss access to mental health or first response emergency services.

Findings

Sensory barriers

Managing health appointments presents challenges for deaf people who do not use the phone. Participants reported that going to a health service in person is the only way they could make or change an appointment or request an interpreter, and that negotiating these encounters through speech and/or writing is difficult. Digital communication modes (email, texting, Video Interpreting Service) were noted as useful alternatives, however these are not commonly used in most healthcare settings, and they require a level of confidence in written English, digital literacy and internet access that is not available to many deaf people. Another sensory barrier noted was the requirement to speak (and hear) through an intercom to enter a delivery ward after hours, causing delays to entry. Without visual alert systems, medical waiting rooms (eg, outpatient clinics) present difficulties for deaf people; a common strategy they report is watching reception staff intently to try to discern when their name is being called. Even in cases when a deaf person informs reception staff of being deaf and asks to be alerted, effective accommodation is rare. One person describes an experience at a hospital:

“I think I missed the call of our name, as I was focused on my son. We’d been waiting for hours, watching every single movement from the medical staff in case I might see them calling my name, lip-reading from a distance”.

Use of interpreters

Professional interpreters play a crucial role in facilitating communication between deaf NZSL users and healthcare personnel, by increasing the deaf person’s capacity to provide and understand information relevant to diagnosis and treatment recommendations. Participants reported many instances in which interpreters were not provided, leaving them feeling disempowered. Interviews with DHB staff and deaf NZSL users revealed that staff seemed unaware of the risks of not using professional interpreters and regularly used communication strategies such as writing (problematic, given literacy levels among deaf NZSL users), gesturing or asking an accompanying family member or friend to mediate communication. DHB staff also

reported using texting and email to communicate with deaf patients about health information. A deaf participant described their response to the use of written communication in a consultation: “

I don’t get the complete information, there’s not much detail or elaboration, and they don’t want to write everything down on a piece of paper... I need to be aware of all the information because it’s my body and it’s my right to know what’s going on and what they’re giving me”.

Some deaf people felt that they were expected to rely on family members as interpreters, even though they were neither fluent enough in NZSL nor necessarily able or inclined to render medical information accurately and fully to the deaf person. One person recounted:

“You don’t get the information you want, plus it’s like going through four different people. I have to explain it to my mum, they explain to the nurse, then the nurse to my mum and my mum to me, then me to my (deaf) partner!”

Professionals were concerned about the current financial constraints of the DHBs and often chose family and friends to mediate communication, without an awareness of the risks of doing so. When family members are not present, this leaves deaf people without an interpreter and in a vulnerable position. Several deaf people told of medical staff asking their children to interpret for them; deaf participants felt strongly that such practices were inappropriate and in breach of their right to accessible healthcare.

It was noted that family members acting as interpreters, even when initiated by a deaf person, can restrict free exchange of information. One family member commented:

“Although my mum prefers us to interpret for her at her hospital appointments it’s striking that the doctor never asked if she would like an interpreter. I also wondered if the doctor should have been asking questions related to the side effects of medication but didn’t because it would’ve been embarrassing, as I’m her son.”

On the other hand, some deaf people reported preferring a family member for reasons such as being of an older generation unaccustomed to interpreters, discomfort

with an interpreter's presence at sensitive appointments such as an STI consultation, or feeling more comfortable communicating through someone familiar. Other participants mentioned instances when a health service had duly booked an interpreter, but they felt it was not really necessary, such as a routine mammogram.

During inpatient stays, interpreters are seldom present and NZSL users may experience stress due to exclusion from communication in the ward. One participant recalled:

"I didn't understand what was going on, there was nobody at all talking to me, I was just waiting. It was very lonely".

While participants described some nurses as 'deaf friendly', by taking time to write things down, gesture and improvise strategies to communicate, one person commented that *"some nurses freaked out and didn't know how to communicate with me. I was constantly having to explain to staff how to communicate with me as a deaf person"*.

Shift handovers and doctor conferences conducted in front of deaf patients created frustration and anxiety:

"While other patients could be involved, all I could do was watch them talk about me while pointing to things on the chart—very frustrating."

At the same time, professional participants in the study expressed willingness to learn more about NZSL users to improve their communication strategies. Both deaf and professional participants reported varied practices for arranging interpreters, and noted problems with availability. Some deaf people and a DHB staff member reported bypassing interpreter booking agencies to directly book freelance interpreters. In these instances, the deaf person or staff member felt they had more control over the booking process. Where appointments were pre-arranged, there was usually time to match an interpreter with the appointment. In unplanned or urgent presentations however, interpreters were difficult to arrange and consultations often proceeded without one in both primary and secondary care settings. Hospital staff described difficulties coordinating interpreter presence with doctor availability, and unclear lines of authority to book interpreters.

Provision of an interpreter for a deaf family member accompanying a patient was raised as a grey area for accessibility, such as the deaf spouse of a hearing woman in labour, or the deaf parent of a child patient. One deaf participant reported:

"When my wife was pregnant and we went to hospital there was no interpreter provided because I am 'only the husband'. We lobbied for that with the hospital and they provided it, but it shouldn't be up to the patient to lobby for their rights".

Consent and treatment compliance

Deaf participants described being in ED or a ward and feeling that staff made no attempt to explain procedures that were about to happen or to ascertain consent. One participant recalled:

"They took me to theatre to put antibiotics into my heart but I wasn't aware of what they were going to do so I told them I wasn't happy to go ahead until I fully understood what was going on. I was uneasy that night, until the next day they explained with an interpreter, then I was okay to go ahead with the procedure".

While incidents like this are relatively rare, deaf people often do not have the confidence (or are too ill) to question their care and advocate for full communication. Instead, they accept procedures with little understanding about what is going on, often leading to considerable distress. Staff acknowledged that fully informed consent for procedures was not always obtained. Time constraints were also cited by staff as an obstacle to good practice; one staff member involved in administering ECGs said she never really gained full consent from her deaf patients or knew whether they used NZSL because, *"with a 15 minute appointment slot you don't have the time to find out"*. Another reason was that they simply did not know how to gain informed consent from a deaf patient, or appreciate the risks of not doing so.

Deaf participants reported discontinuing treatment when they did not understand why they needed to take medication long-term or undergo follow-up procedures (such as regular blood tests). Reluctance to attend repeat procedures is perhaps exacerbated by difficult communication at each encounter. In one case, a deaf woman suffered a stroke after assuming that she only needed to take the prescribed blood

pressure medication until it ran out, as with antibiotics. The resulting stroke required inpatient and outpatient rehabilitation over a number of years. This miscommunication occurred at a GP where no interpreter was used. This case demonstrates the risks of not communicating with a patient in their first language and the high costs to individuals and the DHB associated with a lack of understanding and thus compliance with treatment recommendations.

Access to printed health information

Many deaf participants had struggled to understand printed health information such as brochures or instructions. Some asked family members to explain written material, while others could not access it at all. Many alluded to gaps in their health literacy and English literacy, indicating the need for information to be translated into NZSL: *“I read it again and again and sometimes understand it... Deaf people miss out and don't know about things such as high blood pressure and the link to having a stroke, for example, they don't understand this”*.

Cultural recognition

Deaf participants expressed the importance of healthcare staff recognising their identity as culturally deaf sign language users. When deaf people perceive this recognition, they describe feeling more satisfied with a health consultation even without an interpreter. Many described instances when they felt devalued by comments or behaviours from health professionals that reflected a medicalised view of their deafness as a deficit or infirmity. A common annoyance was a sign reading ‘hearing impaired’ placed above their bed. Most participants rejected this label, and said they would prefer a sign reading, ‘NZSL user’, or ‘deaf’. They noted that staff’s response to the sign by speaking loudly is unhelpful.

Another example of deficit framing occurred during a newborn hearing screening, when a screener told a deaf mother that her baby had passed the test. She reflected:

“What if the baby had been deaf? Would that be a fail? Am I a failure as a deaf person? Little things like this affect me as it shows a cultural attitude of negativity towards deaf people that needs to change”.

Negative consequences for treatment can ensue when a deaf person is not viewed within a sociocultural context.³¹ One example reported involved a standardised assessment tool being used to determine a deaf person’s capacity for independent functioning. Clinicians concluded that because the deaf person was unable to answer certain questions, he had a reduced capacity for functioning. With awareness of this person’s educational and language background, his performance on an English-based test might have been understood in terms of language and cultural difference rather than a functional capacity deficit.

Discussion

Barriers exist at multiple points for deaf NZSL users in health services, echoing issues described for deaf people in similarly economically developed countries.³² Deaf NZSL users report difficulty making and managing appointments at services that rely on auditory interfaces. For various reasons, professional interpreters are often absent in consultations, and assumptions are made by practitioners that lip-reading, writing and family/friend ‘interpreters’ will suffice. Although well-intentioned, staff are not always aware of the risks of not communicating information in NZSL to obtain informed consent for procedures, whereas others are aware but cite time and fiscal pressures as systemic factors leading to non-ideal practices. The data indicate that the logistics of timely and consistent provision of NZSL interpreters in hospitals are complex (not fully canvassed here), and can result in deaf people waiting longer for treatment or accepting partial communication, especially for emergency presentations and inpatient stays. Use of existing video remote interpreting services has not been exploited as a potential solution. Lastly, deaf people report that they do not always follow treatment recommendations as a result of poor communication in consultations, compounded by health literacy gaps.

Inadequate provision of interpreters in healthcare is not unique to NZSL users, but mirrors local practice generally: a 2011 study of clinicians’ use of interpreters with limited English proficiency (LEP) patients in Wellington region DHBs concluded that they,

*“do perceive there to be clinical risk associated with the communication difficulties that they face with LEP patients, but despite this, rarely use trained interpreters. There is a clear mismatch between actual practice and the relatively high levels of awareness of policy, methods of accessing interpreters and the significance of communication difficulties for quality of care”.*³³ As also found in that study, clearly a mix of systemic factors compound deaf people’s disadvantaged position in healthcare, including staff attitudes and awareness, lack of policy and/or staff capacity to comply with it, budgetary constraints and workforce issues with NZSL interpreters.

Importantly, deaf participants framed their healthcare experiences in terms of not feeling recognised as NZSL users with a distinct language, sensory and cultural profile. Their accounts support international evidence that recognition of linguistic and cultural difference as a component of quality healthcare applies to sign language communities as much as to other minority language groups.

Conclusion

Accessing healthcare services, understanding information and interacting with healthcare personnel is problematic for

many deaf NZSL users, partly due to health literacy and English literacy disadvantages, as well as a lack of provision for communication in their preferred modality of NZSL. Healthcare personnel are generally unfamiliar with the perspective of deaf people as members of a socio-cultural group and language minority, and the study finds that they inconsistently use professional NZSL interpreters to address language barriers. In part, systemic barriers hinder better use of interpreting services; the absence of clear DHB policy, strategy and resourcing means healthcare professionals may be ill-equipped to respond to deaf people presenting in health services. Findings from the study show that widespread use of ad hoc communication strategies (eg, lip-reading, writing, gesturing) pose demonstrated risks to health outcomes for deaf patients. Evidence from the study supports the DHBs to proceed with developing a NZSL five-year plan (in progress, involving a co-design approach between a deaf advisory group and the DHBs) for the sub-region. This research exemplifies issues likely to exist in other DHB regions and healthcare services and possible avenues to better ensuring deaf NZSL users’ right to access equitable healthcare.

Competing interests:

Nil.

Acknowledgements:

Members of the steering group that guided this research; Anthony Sammons, Kellye Bensley, Lyneen Allen-Kuklinski, Shannon Krogmann, Gale Cull, Wenda Walton, Vivien Oakshott and the Wellington Deaf community for sharing their stories.

Author information:

Joanne Witko, Project Lead for the ‘NZSL in Health’ project, Office of Disability Strategy and Performance 3DHB, Wairarapa, Hutt Valley and Capital and Coast District Health Board and Psychotherapist, Te-Upoko-me-te-Whatu-o-Te-Ika, Mental Health, Addictions & Intellectual Disability Service; Pauline Boyles, Director of Disability Strategy and Performance, Wairarapa, Hutt Valley and Capital and Coast District Health Boards; Kirsten Smiler, Postdoctoral Scholar, School of Government, Victoria University of Wellington; Rachel McKee, Associate Professor, NZSL Studies, School of Linguistics and Applied Language Studies, Victoria University of Wellington.

Corresponding author:

Joanne Witko, Strategy, Innovation and Performance, Capital and Coast District Health Board, Riddiford Street, Wellington 6021.
joanne.witko@sidu.org.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7428>

REFERENCES:

1. Pollard RQ, Barnett S. Health-Related Vocabulary Knowledge Among Deaf Adults. *Rehabil Psychol*. 2009; 54;2:182–5.
2. Emond A, Ridd M, Sutherland H, Allsop L, et al. The current health of the signing Deaf community in the UK compared with the general population: a cross-sectional study *BMJ Open* 2015; 5:1:eDD6668.
3. Royal National Institute for Deaf People. A Simple Cure: A national report into deaf and hard of hearing people's experiences of the National Health Service. 2004; London: Royal National Institute for Deaf People.
4. Smeijers AS, Pfau R. Towards a Treatment for Treatment: On communication between General Practitioners and Their Deaf Patients. *The Sign Language Translator and Interpreter* 2009; 3:1:1–14.
5. Fellingner J, Holzinger D, Pollard R. Mental health of deaf people. *Lancet* 2012; 379:1037–44.
6. Dugdale PO. Being Deaf in New Zealand: A Case Study of the Wellington Deaf Community. 2000. Unpublished PhD Thesis. Victoria University of Wellington.
7. McKee R. People of the Eye: Stories from the Deaf World. 2001. Wellington : Bridget Williams Books.
8. Collins-Ahlgren M. Aspects of New Zealand Sign Language. 1989. Unpublished PhD Thesis. Victoria University of Wellington.
9. Fitzgerald & Associates Scoping support for New Zealand Sign Language users accessing the curriculum. Part II: A New Zealand Overview. Report prepared for the NZ Ministry of Education. 2010 Retrieved from <http://www.educationcounts.govt.nz/publications/>
10. Powell D, Hyde M. Deaf education in New Zealand: Where we have been and where we are going. *Deafness and Education International*. 2014; 16:3:129–145.
11. Dewalt DA, Berkman ND, Sheridan S, Lohr KN, et al. Literacy and health outcomes: a systematic review of the literature. *J Gen Intern Med*. 2004; 19:12:1228–39.
12. Barnett S, McKee M, Smith SR, Pearson TA. Deaf Sign Language Users, Health Inequalities, and Public Health: Opportunity for Social Justice. *Prev Chronic Dis*. 2011 Mar; 8:2:A45.
13. McEwen E, Anton-Culver H. The medical communication of deaf patients. *J Fam Practitioners*, 1988; 26:3:289–91.
14. Steinberg AG, Barnett S, Meador HE, Wiggins EA, et al. Health Care System. Accessibility: Experiences and Perceptions of Deaf People. *J Gen Intern Med*. 2006; 21:3:260–6.
15. Mohay HA, Kleinig DF. Providing medical care for deaf patients. *Med J Australia*. 1991; 155:498–9.
16. WHO. The Determinants of Health. Retrieved from <http://www.who.int/hia/evidence/doh/en/>
17. Harmer L. Health care delivery and deaf people: practice, problems, and recommendations for change. *J Deaf Stud Deaf Educ*. 1999; 2:73–110.
18. Barnett S, McKee M, Smith SR, Pearson TA. Deaf Sign Language Users, Health Inequalities, and Public Health: Opportunity for Social Justice. *Prev Chronic Dis*. 2011; 8:2:A45.
19. Alexander A, Ladd P, Powell S. (2012). Deafness might damage your health. *Lancet*. 2012; 379:9820:979–81.
20. MacKinney T, Walters GD, Bird GL, Nattinger AB. Improvements in Preventive Care and Communication for Deaf Patients: Results of a Novel Primary Health Care Program. *J Gen Intern Med*. 1995; 10:3:133–7.
21. Middleton A, Turner GH, Bitner-Glindzicz M, Lewis P, et al. Preferences for communication in clinic from deaf people: a cross-sectional study. *J Eval Clin Pract*. 2010; 16:4:811–7.
22. Lowrie M. An Enquiry into access to AUSLAN interpreters in Victorian Hospitals. 2014. Deaf Victoria, Australia.
23. Napier J, Kidd MR. English literacy as a barrier to health care information for deaf people who use Auslan. *Aust Fam Physician*. 2013; 42:12:896–9.
24. Flores G. (2005). The impact of Medical Interpreter Services on the Quality of Health Care: A systematic review. *Med Care Res Rev*. 2005; 62:3:255–99.
25. Hauland H, Allen C. Deaf people and human rights. 2009. Helsinki: World Federation of the Deaf.
26. UNCRPD. Convention on the rights of persons with disabilities. 2006. Retrieved from <http://www.un.org/disabilities/default.asp?navid=15&pid=150>
27. Office of the Minister of Disability Issues. (2003). Cabinet paper on the New Zealand Sign Language Bill. Wellington: Office for Disability Issues.
28. Human Rights Commission. A New Era in the Right to Sign - He Houhanga Rongo te Tika Ki Te Reo Turi. Report of the New Zealand Sign Language Enquiry. 2013. Wellington: Human Rights Commission.

29. <http://www.hdc.org.nz/the-act--code/the-code-of-rights>
30. Office for Disability Issues. New Zealand Sign Language Board Action Plan 2016-2018. Wellington: Office for Disability Issues. <http://www.odi.govt.nz/new-zealand-sign-language-nzsl/>
31. Phillips BA. Bringing culture to the forefront: Formulating diagnostic impressions of deaf and hard of hearing people at times of medical crisis. *Professional Psychology: Research and Practice*, 1997; 27(2):137-144.
32. Kuenburg A, Fellingner P, Fellingner J. Health Care Access Among Deaf People. *J Deaf Stud Deaf Educ*. 2016; 21:1:1-10.
33. Gray B, Stanley J, Stubbe M, Hilder J. Communication difficulties with limited English proficiency patients: clinician perceptions of clinical risk and patterns of use of interpreters. *NZ Med J*. 2011; 124:1342:23-38.

A New Zealand platform to enable genetic investigation of adverse drug reactions

Simran DS Maggo, Eng Wee Chua, Paul Chin, Simone Cree, John Pearson, Matt Doogue, Martin A Kennedy

ABSTRACT

A multitude of factors can affect drug response in individuals. It is now well established that variations in genes, especially those coding for drug metabolising enzymes, can alter the pharmacokinetic and/or pharmacodynamic profile of a drug, impacting on efficacy and often resulting in drug-induced toxicity. The UDRUGS study is an initiative from the Carney Centre for Pharmacogenomics to biobank DNA and store associated clinical data from patients who have suffered rare and/or serious adverse drug reactions (ADRs). The aim is to provide a genetic explanation of drug-induced ADRs using methods ranging from Sanger sequencing to whole exome and whole genome sequencing. Participants for the UDRUGS study are recruited from various sources, mainly via referral through clinicians working in Canterbury District Health Board, but also from district health boards across New Zealand. Participants have also self-referred to us from word-of-mouth communication between participants. We have recruited various ADRs across most drug classes. Where possible, we have conducted genetic analyses in single or a cohort of cases to identify known and novel genetic association(s) to offer an explanation to why the ADR occurred. Any genetic results relevant to the ADR are communicated back to the referring clinician and/or participant. In conclusion, we have developed a programme for studying the genetic basis of severe, rare or unusual ADR cases resulting from pharmacological treatment. Genomic analyses could eventually identify most genetic variants that predispose to ADRs, enabling *a priori* detection of such variants with high throughput DNA tests.

An adverse drug reaction (ADR) is defined by the World Health Organization as any response to a drug which is noxious, unintended, and which occurs at doses used for prophylaxis, diagnosis and therapy.¹ Adverse drug reactions cause significant morbidity and mortality, with up to 100,000 annual deaths reported in the US in 2014, with an additional 800,000 ADRs reported as serious (resulted in hospitalisation, were life-threatening, led to a disability or resulted in a congenital anomaly).² In Christchurch ADRs were reported to be primarily responsible for about 19% of admissions to general medicine and partially contribute to a further 9% of admissions.³ Data from the UK showed that hospitalisations due to ADRs are estimated to cost the health system over £637 million annually,⁴ and about half are considered preventable.^{5,6} Hence strategies to identify patients at risk of ADRs offer opportunities for both health gains and cost savings.

Some risk factors contributing to the development of ADRs are known, including age, sex, renal function, body composition, co-medications, existing diseases, smoking and/or alcohol consumption. However, these only account for some of the risk and many patients present with idiosyncratic ADRs where the above mentioned factors cannot provide a plausible explanation, and in some cases genetic causes have been identified. A small proportion of ADRs are immune-mediated hypersensitivity reactions often labelled as drug allergies. Some immune-mediated hypersensitivity ADRs have a significant association with particular human leukocyte antigen (HLA) alleles. Examples include an increased risk of abacavir-induced hypersensitivity in patients carrying at least one *HLA-B*57:01* allele,⁷ and an increased risk of carbamazepine-induced Stevens-Johnson syndrome (SJS) in patients carrying at least one copy of *HLA-B*15:02*.⁸ The association of

*HLA-B*15:02* is reported to be strongest in individuals of Asian ancestry, particularly South East Asians, where this variant is reported as common, ie, occurs at a frequency of >1% in the population.⁹ In contrast, carbamazepine-induced SJS is rare in individuals of European ancestry, and is associated with a different HLA allele (*HLA-A*31:01*).^{9,10}

Most ADRs are due to the extended or non-specific pharmacological properties of a drug. The risks increase with drug dose and there is substantial inter-individual variability in the pharmacokinetics and pharmacodynamics limiting our ability to predict individual risk. Some ADRs have been found to have significant association with particular genes. Examples include an increased risk of hematopoietic toxicity associated with genetic variants, which reduce thiopurine S-methyltransferase (TPMT) activity, increased risk of simvastatin-induced muscle myopathy with specific variants in the *SLCO1B1* gene and various ADRs related to reduced CYP2D6 activity.^{11–13} However, many more ADRs have not been subject to genetic studies.

This report describes a New Zealand initiative to systematically collect samples from people who have had ADRs to characterise known or identify novel genetic variants associated with rare, unusual and severe ADRs. The pilot work described in this report was prompted by the strong genetic component observed for serious ADRs, such as those of abacavir, azathioprine and carbamazepine,^{14–17} for which pharmacogenetics has proven clinical utility. Several other observations also informed our study design. First, sequencing of many human genomes has revealed that each individual has a high load of singleton variants, some of which may impact pharmacogenes.^{18–20} This suggests that the overall contribution of low frequency gene variants towards inter-individual differences in drug response may be greater than previously thought. Second, international collaborative efforts between multiple centres have emerged as a possible solution to the as yet unresolved questions in pharmacogenomics research. Notable examples are projects hosted by the Pharmacogenomics Knowledge Base (PharmGKB; <http://www.pharmgkb.org/page/projects>), and the International Serious Adverse Event Consortium (iSAEC; <http://www.saeconsortium.org/>). To contribute to these important initiatives, the essential

prerequisite is an effective means of accruing blood and DNA samples from ADR cases that are phenotypically well characterised. Taking statin-myopathy as an example, it is important to define myopathy using standardised guidelines so that it is not missed, or incorrectly classified as another statin-muscle ADR such as myalgia, rhabdomyolysis or necrotising autoimmune myopathy.²¹ Third, high throughput next-generation sequencing technology, which enables simultaneous sequencing of a large number of genes or even entire genomes, has proven to be a powerful tool in the discovery of causal variants for rare diseases.^{22,23} The success of this technology in Mendelian disease gene discovery suggests it will have similar value in identifying rare, functionally relevant pharmacogenetic variants.

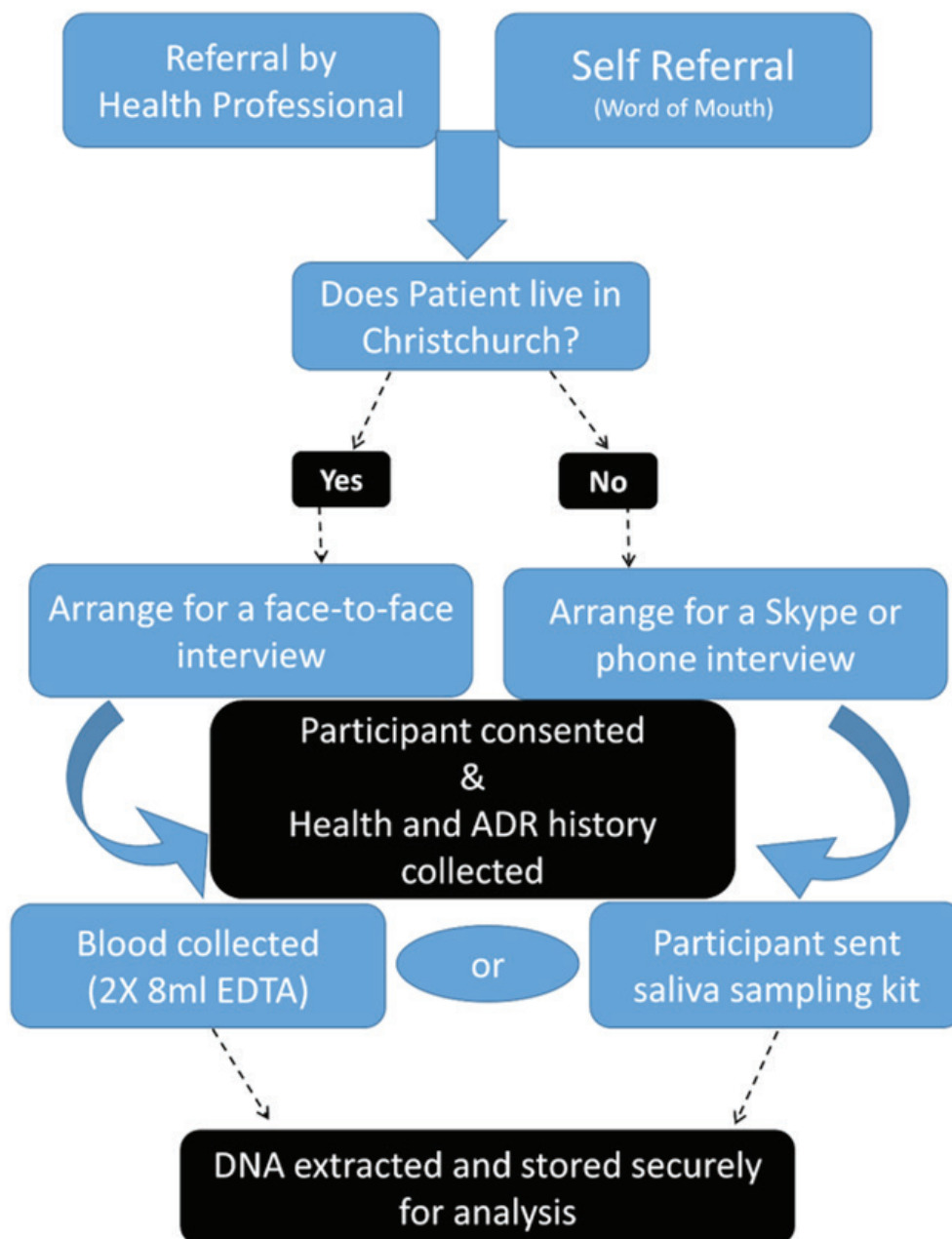
On this background we established a programme called UDRUGS (Understanding Adverse Drug Reactions Using Genomic Sequencing), with two major goals: (1) to establish a DNA bank linked to clinical information of patients who have experienced severe ADRs or exhibited aberrant response to pharmacological treatment, and (2) to explore the range of variations in known pharmacogenes that may contribute to the observed phenotypes.

Methods

Patient recruitment

The programme was approved by the Southern Health and Disability Ethics Committee (New Zealand). Participant recruitment is an active ongoing process. Participants are recruited from various sources (Figure 1). To date these have been mainly via referral through clinicians working in Canterbury District Health Board, but also from clinicians in Southern, Capital & Coast, and Auckland District Health Boards. Other sources have been patient self-referrals resulting from word-of-mouth communication between patients. The programme includes studies of specific cohorts, that is, patients who have experienced a specific ADR or unusual drug response. For example, we recruited a group of patients who preferentially methylate thiopurine compounds into 6-methylmercaptopyrimidine (6-MMP), as evidenced by a high 6-MMP/6-TGN ratio (6-thioguanine nucleotides) (>20), and applied various approaches to evaluate possible genetic underpinnings of this phenotype.²⁴

Figure 1: Patient recruitment into the UDRUGS study.



Consenting, sample collection and storage

Written consent is sought from all patients referred to the UDRUGS study, and is obtained by a researcher or collaborating clinician. Consent includes provision for extensive whole genome analyses, long-term storage of blood and DNA samples, access to hospital medical records, contact with family members or relatives where necessary, and sharing of research data and samples with local or international collaborators. The possibility of incidental genetic

findings is discussed with the patient at time of consenting. For patients unable to attend a face-to-face meeting, a telephone or Skype meeting is arranged to discuss and complete the consent form with a UDRUGS study coordinator. After consent is obtained, peripheral blood in two 8ml EDTA tubes is collected and frozen at -20°C until required for DNA extraction. If a study participant is unable to provide a blood sample, a saliva sample is collected via an Oragene DNA (OG-500) kit (DNA Genotek Inc.).^{25,26} In this case, participants are sent an Oragene kit

via courier, with a return courier envelope to return the saliva kit to the Carney Centre for Pharmacogenomics. Saliva samples are stable at room temperature for at least two years,²⁵ and on extraction, DNA quantity and quality is sufficient to use in standard polymerase chain reaction (PCR) as well as next-generation sequencing, eg, whole exome sequencing. Extracted DNA is aliquoted and stored at -80 °C until required for analysis. All samples are assigned a UDRUGS code and spreadsheets linking the codes to participants' details are password protected and stored securely.

Medical history questionnaire

The questionnaire was designed by adapting the Pharmacist's Workup of Drug Therapy, and is therefore suitable for universal documentation of ADRs.²⁷ The questionnaire consists of two parts: the first part is answered by the participant at the time of consenting and blood collection with the help of a UDRUGS study coordinator. The second part of the questionnaire is completed by the referring clinician. Participants' demographics, disease and medication histories, detailed account of the adverse drug reaction(s), and objective data (eg, blood test results including concentrations of the suspected drug) are documented. Causality of adverse drug reactions is evaluated using the Naranjo Algorithm.²⁸ For participants unable to attend a face-to-face meeting, the health questionnaire is completed over the phone by the UDRUGS study coordinator.

Genetic analysis

Various genetic analysis techniques are employed. Sanger sequencing of candidate genes—those related to drug metabolism in particular—has been the principal route of analysis. Increasingly with multiple candidate genes potentially involved, a more comprehensive approach is adopted. This entails whole exome sequencing (WES), which enables simultaneous sequencing of all protein coding regions (exome) within the human genome. However, whole genome sequencing (WGS) is becoming more tractable and affordable, and we will begin testing this approach for analysis of UDRUGS cases in the near future. Functional effects of novel or rare genetic variants that may be clinically important are analysed using computer prediction algorithms or software

such as Annotate Variation (ANNOVAR),²⁹ Combined Annotation Dependent Depletion (CADD)³⁰ and the SeattleSeq Annotation server.³¹ However, proof of causality would require replication in other cohorts and more extensive laboratory-based studies.

Long-term storage and contribution to international consortia

Where no immediate genetic analysis is possible because of limitations in current technology or genetics understanding, the participants' blood, saliva and extracted DNA samples are stored at -80 °C for future analysis or contribution to appropriate international studies.

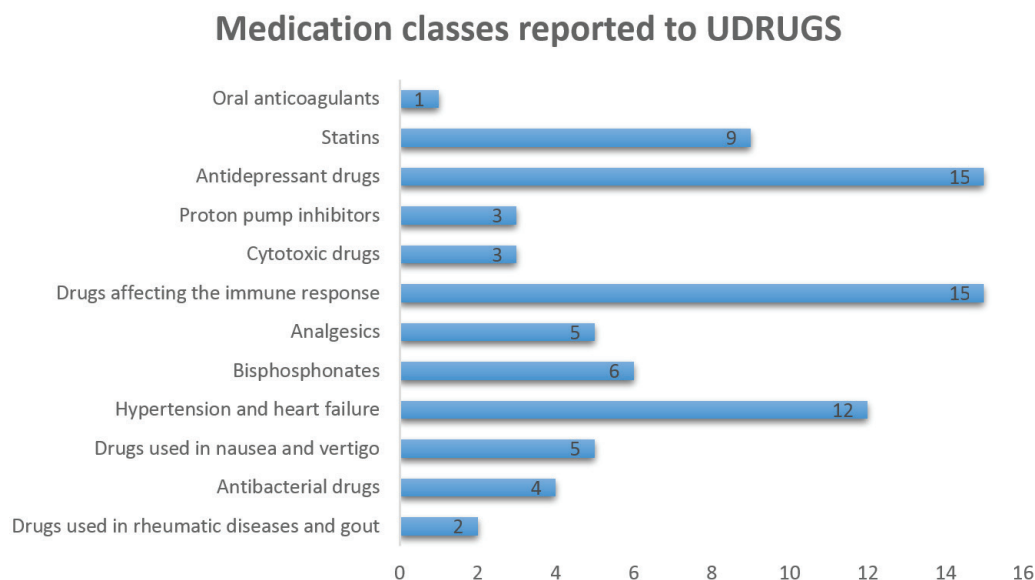
Return of results

We communicate relevant research findings back to the participants and/or the referring clinician, emphasising that the findings are from a research laboratory. Where requested, we can also provide a list of medications for which genetic findings may be important. This list is largely based on the therapeutic guidelines formulated by Swen et al 2011,³² and recently, guidelines are available from the Clinical Pharmacogenetics Implementation Consortium (CPIC).^{33,34}

Results and discussion

To date we have collected 80 cases (with stored DNA samples and clinical histories) thus far in the exploratory phase of this study (Figure 2). We utilised broad inclusion criteria and collected ADR cases, which seemed more likely to have a genetic underpinning. We did not exclude patients taking interacting drugs or patients with multiple medical conditions, as these factors need to be taken into account during any genetic association analyses in the future.

In collaboration with clinical colleagues, it has been possible to recruit case series of patients who have had specific ADRs. The first example of this includes the aforementioned whole exome study of 12 patients who exhibited extreme therapeutic resistance to azathioprine or 6-mercaptopurine, termed "extreme shunters".²⁴ In addition to WES, array-based comparative genomic hybridisation (aCGH) was also carried out to identify potential genes with copy number variations (CNVs). Novel genetic variants

Figure 2: Horizontal bars show the current number of UDRUGS cases per medication class.

identified in four genes associated with thiopurine metabolism were considered, but no significant variants were identified as being associated with treatment resistance in this study.²⁴ Similarly, we have recently recruited eight patients defined as “statin intolerant”. These selected patients all have a history of persistent muscle myalgia, even after being challenged on two different statins, at varying doses. It has been previously reported that up to 50% of patients initiated on statin therapy are non-compliant after the first year, and ADRs or fear of ADRs are two of the major reasons associated with this high rate of non-adherence to statins.^{35–37} Therefore, identifying genetic variants associated with statin-induced muscle myalgia will allow clinicians to consider a different lipid lowering agent or introduce statin intolerance management strategies earlier.^{38,39} We have conducted WES on these eight patients, and these data are currently being analysed. Although these studies are small, the data can ultimately be combined with that from other groups, and underlying genetic patterns relevant to the ADR may become apparent.

Currently we are actively recruiting patients who have a thorough medical history of drug-induced hyponatremia, particularly induced by drugs such as

thiazide diuretics, SSRIs and proton pump inhibitors (PPIs). Furthermore, we are looking to recruit patients with a history of PPI-induced hypomagnesaemia and/or interstitial nephritis. The aim is to identify genetic variants which predispose to these specific ADRs.

Biobanking of samples is essential for contemporary or future personalised medicine research, and is being carried out elsewhere to facilitate the genetic analyses of ADRs.^{40,41} The preliminary findings that we have obtained and reported^{24,42} show that this is a viable route towards uncovering novel genetic components of severe ADRs or unusual drug response. Future work with UDRUGS will focus on establishing more systematic processes for patient recruitment, and in this regard, exploring the use of e-prescribing systems that are being introduced into district health boards in this country may be an efficient way to identify rare, severe or unusual ADRs. With the fast developing pace of genomic technologies, we are also trialling new DNA sequencing methods and developing assays that can be efficiently applied to incoming samples. Finally, where possible, studies to evaluate the likely functional effects of any relevant genetic variants identified will be conducted.

Conclusion

We have developed a programme for studying severe, rare or unusual ADR cases resulting from pharmacological treatment—the high-risk group where genetic factors may be more likely to be implicated. This has entailed recruiting participants via various routes, documenting phenotype data, collecting and storing blood samples, exploring the range of appropriate genetic analyses, assessing the clinical relevance of genetic findings based on *in silico* predictions

or available literature, and returning results to participants in an easily understood format. Genomic analyses should eventually identify most genetic variants that predispose to ADRs, enabling *a priori* detection of such variants with high throughput DNA tests. Establishing UDRUGS, a New Zealand DNA bank that can receive ADR samples and associated clinical data, will ensure we contribute to and benefit from such research. We welcome approaches from colleagues with problematic ADR cases that may contribute to this goal.

Competing interests:

Dr Doogue reports grants from Health Research Council during the conduct of the study; Dr Doogue is employed by CDHB as a clinical pharmacologist with responsibilities including adverse drug reactions and medicines governance. Dr Doogue's work includes general medicine, and patients with adverse drug reactions are admitted under general medicine.

Dr Doogue is a member of the Health Quality and Safety Commission, Medicines Safety Expert Advisory Group.

Acknowledgements:

Dr Eng Wee Chua initiated the UDRUGs study as part of his PhD project. Dr Chua and Dr Maggo should be considered joint first authors of this publication.

The Carney centre for Pharmacogenomics and salary for research fellow Dr Simran Maggo is funded by the Jim and Mary Carney Charitable Trust. Work on UDRUGS is also supported by a project grant from the Health Research Council of New Zealand. We thank the clinicians and patients who have supported this work.

Author information:

Simran DS Maggo, Research Fellow, Carney Centre for Pharmacogenomics and Department of Pathology, University of Otago, Christchurch; Eng Wee Chua, Lecturer, Faculty of Pharmacy, Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia;

Paul Chin, Clinical Pharmacologist, Department of Medicine, University of Otago, Christchurch; Simone Cree, Research Fellow, Carney Centre for Pharmacogenomics and Department of Pathology, University of Otago, Christchurch;

John Pearson, Senior Research Fellow & Biostatistician, Biostatistics and Computational Biology Unit, University of Otago, Christchurch; Matthew Doogue, Clinical Pharmacologist, Department of Medicine, University of Otago, Christchurch;

Martin A Kennedy, Department of Pathology, University of Otago, Christchurch.

Corresponding author:

Dr Simran Maggo, Pathology, University of Otago, 2 Riccarton Avenue, Christchurch 8140.
simran.maggo@otago.ac.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7429>

REFERENCES:

1. (WHO) WHO. International Drug Monitoring: The role of national centres. Geneva 1972.
2. FDA. FAERS Reporting by Patient Outcomes by Year. 2015 [updated November 2015; cited 17/08/2016]; Available from: <http://www.fda.gov/drugs/guidancecompliance-regulatoryinformation/surveillance/adversedrug-effects/ucm070461.htm>
3. McLachlan CY, Yi M, Ling A, et al. Adverse drug events are a major cause of acute medical admission. *Intern Med J.* 2014;44(7):633-8. doi: 10.1111/imj.12455.
4. Davies EC, Green CF, Taylor S, et al. Adverse drug reactions in hospital in-patients: a prospective analysis of 3695 patient-episodes. *PloS one.* 2009; 4(2):e4439. doi: 10.1371/journal.pone.0004439.
5. Hakkarainen KM, Hedna K, Petzold M, et al. Percentage of patients with preventable adverse drug reactions and preventability of adverse drug reactions—a meta-analysis. *PloS one.* 2012; 7(3):e33236. doi: 10.1371/journal.pone.0033236.
6. Sultana J, Cutroneo P, Trifiro G. Clinical and economic burden of adverse drug reactions. *J Pharmacol Pharmacother.* 2013; 4(Suppl 1):S73–7. doi: 10.4103/0976-500X.120957.
7. Hetherington S, Hughes AR, Mosteller M, et al. Genetic variations in HLA-B region and hypersensitivity reactions to abacavir. *Lancet.* 2002; 359(9312):1121–2. doi: 10.1016/S0140-6736(02)08158-8.
8. Kulkantrakorn K, Tassaneeyakul W, Tiamkao S, et al. HLA-B*1502 strongly predicts carbamazepine-induced Stevens-Johnson syndrome and toxic epidermal necrolysis in Thai patients with neuropathic pain. *Pain Pract.* 2012; 12(3):202–8. doi:10.1111/j.1533-2500.2011.00479.x.
9. Amstutz U, Shear NH, Rieder MJ, et al. Recommendations for HLA-B*15:02 and HLA-A*31:01 genetic testing to reduce the risk of carbamazepine-induced hypersensitivity reactions. *Epilepsia.* 2014; 55(4):496–506. doi: 10.1111/epi.12564.
10. Yip VL, Pirmohamed M. The HLA-A*31:01 allele: influence on carbamazepine treatment. *Pharmgenomics Pers Med.* 2017; 10:29–38. doi: 10.2147/PGPM.S108598.
11. Pirmohamed M, Park BK. Cytochrome P450 enzyme polymorphisms and adverse drug reactions. *Toxicology.* 2003; 192(1):23–32.
12. Link E, Parish S, Armitage J, et al. SLCO1B1 variants and statin-induced myopathy—a genome-wide study. *The New England journal of medicine.* 2008; 359(8):789–99. doi: 10.1056/NEJMoa0801936.
13. Tamm R, Magi R, Tremmel R, et al. Polymorphic variation in TPMT is the principal determinant of TPMT phenotype: A meta-analysis of three genome-wide association studies. *Clin Pharmacol Ther.* 2017; 101(5):684–95. doi: 10.1002/cpt.540.
14. Chung BH, Kim JH, Choi BS, et al. Clinical significance of the presence of anti-human leukocyte antigen-donor specific antibody in kidney transplant recipients with allograft dysfunction. *Korean J Intern Med.* 2016. doi: 10.3904/kjim.2016.107.
15. Mallal S, Nolan D, Witt C, et al. Association between presence of HLA-B*5701, HLA-DR7, and HLA-DQ3 and hypersensitivity to HIV-1 reverse-transcriptase inhibitor abacavir. *Lancet.* 2002; 359(9308):727–32.
16. Priest VL, Begg EJ, Gardiner SJ, et al. Pharmacoeconomic analyses of azathioprine, methotrexate and prospective pharmacogenetic testing for the management of inflammatory bowel disease. *Pharmacoeconomics.* 2006; 24(8):767–81.
17. Gardiner SJ, Gearry RB, Barclay ML, et al. Two cases of thiopurine methyltransferase (TPMT) deficiency—a lucky save and a near miss with azathioprine. *Br J Clin Pharmacol.* 2006; 62(4):473–6. doi:10.1111/j.1365-2125.2005.02474.x.
18. Gordon AS, Tabor HK, Johnson AD, et al. Quantifying rare, deleterious variation in 12 human cytochrome P450 drug-metabolism genes in a large-scale exome dataset. *Hum Mol Genet.* 2014; 23(8):1957–63. doi: 10.1093/hmg/ddt588.
19. Nelson MR, Wegmann D, Ehm MG, et al. An abundance of rare functional variants in 202 drug target genes sequenced in 14,002 people. *Science.* 2012; 337(6090):100–4. doi: 10.1126/science.1217876.
20. Lek M, Karczewski KJ, Minikel EV, et al. Analysis of protein-coding genetic variation in 60,706 humans. *Nature.* 2016; 536(7616):285–91. doi: 10.1038/nature19057.
21. Alfirevic A, Neely D, Armitage J, et al. Phenotype standardization for statin-induced myotoxicity. *Clin Pharmacol Ther.* 2014; 96(4):470–6. doi: 10.1038/clpt.2014.121.

22. Koboldt DC, Steinberg KM, Larson DE, et al. The next-generation sequencing revolution and its impact on genomics. *Cell*. 2013; 155(1):27–38. doi: 10.1016/j.cell.2013.09.006.
23. Maggo SD, Savage RL, Kennedy MA. Impact of New Genomic Technologies on Understanding Adverse Drug Reactions. *Clin Pharmacokinet*. 2016; 55(4):419–36. doi: 10.1007/s40262-015-0324-9.
24. Chua EW, Cree S, Barclay ML, et al. Exome sequencing and array-based comparative genomic hybridisation analysis of preferential 6-methylmercaptopurine producers. *Pharmacogenomics J*. 2015; 15(5):414–21. doi: 10.1038/tj.2015.9.
25. Genotek D. DNA collections kits. 2017 [cited 2017 31st July]; Available from: <http://www.dnagenotek.com/ROW/products/OG500.html>
26. Nunes AP, Oliveira IO, Santos BR, et al. Quality of DNA extracted from saliva samples collected with the Oragene DNA self-collection kit. *BMC Med Res Methodol*. 2012; 12:65. doi: 10.1186/1471-2288-12-65.
27. Strand LM, Cipolle RJ, Morley PC. Documenting the clinical pharmacist's activities: back to basics. *Drug Intell Clin Pharm*. 1988; 22(1):63–7.
28. Naranjo CA, Busto U, Sellers EM, et al. A method for estimating the probability of adverse drug reactions. *Clin Pharmacol Ther*. 1981; 30(2):239–45.
29. Wang K, Li M, Hakonarson H. ANNOVAR: functional annotation of genetic variants from high-throughput sequencing data. *Nucleic Acids Res*. 2010; 38(16):e164. doi: 10.1093/nar/gkq603.
30. Kircher M, Witten DM, Jain P, et al. A general framework for estimating the relative pathogenicity of human genetic variants. *Nature genetics*. 2014; 46(3):310–5. doi: 10.1038/ng.2892.
31. Institute EP-NHGR. SeattleSeq Annotation. 2017 [updated 31st July 2017; cited 2017 31st July]; Available from: <http://snp.gs.washington.edu/SeattleSeqAnnotation138/index.jsp>
32. Swen JJ, Nijenhuis M, de Boer A, et al. Pharmacogenetics: from bench to byte—an update of guidelines. *Clin Pharmacol Ther*. 2011; 89(5):662–73. doi: 10.1038/clpt.2011.34.
33. Caudle KE, Klein TE, Hoffman JM, et al. Incorporation of pharmacogenomics into routine clinical practice: the Clinical Pharmacogenetics Implementation Consortium (CPIC) guideline development process. *Curr Drug Metab*. 2014; 15(2):209–17.
34. Relling MV, Klein TE. CPIC: Clinical Pharmacogenetics Implementation Consortium of the Pharmacogenomics Research Network. *Clin Pharmacol Ther*. 2011; 89(3):464–7. doi: 10.1038/clpt.2010.279.
35. McGinnis B, Olson KL, Magid D, et al. Factors related to adherence to statin therapy. *The Annals of pharmacotherapy*. 2007; 41(11):1805–11. doi: 10.1345/aph.1K209.
36. Harrison TN, Derose SF, Cheetham TC, et al. Primary nonadherence to statin therapy: patients' perceptions. *Am J Manag Care*. 2013; 19(4):e133-9.
37. Casula M, Tragni E, Catapano AL. Adherence to lipid-lowering treatment: the patient perspective. *Patient Prefer Adherence*. 2012; 6:805–14. doi: 10.2147/PPA.S29092.
38. BPACNZ. Investigating myalgia in patients taking statins. 2014 [cited 2016 15th September 2016]; Available from: <http://www.bpac.org.nz/BT/2014/August/myalgia.aspx>
39. Mancini GB, Baker S, Bergeron J, et al. Diagnosis, Prevention, and Management of Statin Adverse Effects and Intolerance: Canadian Consensus Working Group Update (2016). *Can J Cardiol*. 2016; 2(7 Suppl):S35–65. doi: 10.1016/j.cjca.2016.01.003.
40. Ross CJ, Visscher H, Sistonen J, et al. The Canadian Pharmacogenomics Network for Drug Safety: a model for safety pharmacology. *Thyroid*. 2010; 20(7):681–7. doi: 10.1089/thy.2010.1642.
41. van der Wouden CH, Swen JJ, Samwald M, et al. A brighter future for the implementation of pharmacogenomic testing. *Eur J Hum Genet*. 2016. doi: 10.1038/ejhg.2016.116.
42. Chua EW, Foulds J, Miller AL, Kennedy MA. Novel CYP2D6 and CYP2C19 variants identified in a patient with adverse reactions towards venlafaxine monotherapy and dual therapy with nortriptyline and fluoxetine. *Pharmacogenet Genomics*. 2013; 23(9):494–7. doi:10.1097/FPC.0b013e328363688d.

Acupuncture, ACC and the Medicines Act

Daniel J Ryan

ABSTRACT

Acupuncture is covered under the Accident Compensation (Liability to Pay or Contribute to Cost of Treatment) Regulations 2003, and is therefore eligible for Accident Compensation Corporation payments for the treatment of personal injuries. This study searched New Zealand acupuncturists' websites for therapeutic claims that may breach Section 58(1)(a) of the Medicines Act. A search of acupuncturists' websites shows that many claim to be able to treat a wide range of conditions, despite a lack of evidence showing the efficacy of acupuncture in the treatment of those conditions. Practitioners and owners of websites likely to be in breach of the Medicines Act include many committee members from acupuncture's professional bodies.

Acupuncture is one of the most popular forms of 'alternative' health therapy in New Zealand.¹

It is often used for the treatment of personal injuries, and New Zealand's Accident Compensation Corporation (ACC) spent \$30 million on acupuncture treatments from July 2015 to June 2016 (S Melville, personal communication, July 26, 2016), an increase of \$4 million from \$26 million the year before.²

Gilbey et al noted there are around 17,000 published articles on acupuncture. They concluded that acupuncture may be efficacious for some types of pain relief, as well as for nausea and vomiting.³ However, the authors of this meta-review noted that there was "insufficient evidence to make positive recommendations", and suggested that the quality of the studies, even for these conditions, was poor. The UK's National Institute for Health and Care Excellence (NICE) no longer recommends using acupuncture for the treatment of any health conditions other than for some types of headaches (K Summerscales, personal communication, December 6, 2017).⁴

Further, concepts such as qi, meridians and acupuncture points are not based on evidence,⁵ and it generally does not matter where acupuncture needles are inserted.⁶ There is also an issue of positive acupuncture publication bias in a number

of countries.⁷ Many acupuncture studies will show small improvements, but any small improvement could well be due to the placebo effect⁸—particularly when subjects and practitioners are not blinded.⁹

The two main professional bodies for acupuncture are Acupuncture NZ and The NZ Acupuncture Standards Authority (NZASA). Between them they oversee the majority of registered acupuncturists for New Zealand. If an acupuncturist is registered with either of these two bodies, they can receive ACC payments under the Accident Compensation (Liability to Pay or Contribute to Cost of Treatment) Regulations 2003.¹⁰ In rare instances, osteopaths and physiotherapists who perform acupuncture are registered with the Physiotherapy Board, the Osteopathic Council or the Medical Council of New Zealand.

Acupuncturists are unable to lodge ACC claims directly, and require a medical GP, osteopath or physiotherapist to do this on their behalf. In 2015, GPs logged the majority (53%) of ACC claims that resulted in acupuncture treatment, out of a total of 58,681 claims.¹¹

Acupuncture NZ advocates that acupuncture, "can be used to treat an enormous variety of conditions from sporting injuries to digestive upsets or even the common cold".¹² They also have a list of conditions which can be treated with

acupuncture based on “recommendations from the World Health Organization”.¹³ However, O’Sullivan et al explain that the WHO report “was withdrawn in March 2014, in response to substantial evidence contradicting the WHO’s advice”. They found 27% of Australian acupuncturists’ websites quote from withdrawn WHO evidence.¹⁴ The quoted statement from Acupuncture NZ is therefore misleading, and misleading claims from practitioners have been successfully challenged in the past. From October 2012 to April 2016, 14 Advertising Standards Authority (ASA) complaints were laid against acupuncturists for making inappropriate claims regarding the therapeutic benefits of their treatment. All but one of the complaints were upheld or settled.¹⁵

Despite this, there appear to be no studies examining the explicit or implicit claims made on New Zealand-based acupuncture websites which might have the potential to mislead first-time users of acupuncture services.

The purpose of the current study was therefore to investigate the websites of acupuncture practitioners in New Zealand, regarding direct or indirect claims of being able to assist with conditions that are listed under Section 58(1)(a) of the Medicines Act to see if therapeutic claims that may be considered inappropriate are a feature of the websites of companies and professionals offering acupuncture services.

Methods

In August 2016, Daniel Ryan searched for the websites of acupuncture clinics that were potentially in breach of Section 58(1)(a) of the Medicines Act. This section of the Medicines Act was chosen as it prohibits the publication of advertisements claiming the ability to prevent, mitigate or cure a range of serious diseases:

“Subject to section 60, no person shall publish, or cause or permit to be published, any medical advertisement that—directly or by implication claims, indicates, or suggests that medicines of the description, or medical devices of the kind, or the method of treatment, advertised will prevent, alleviate, or cure any disease, or prevent, reduce, or terminate any physiological condition

specified, or belonging to a class of disease or physiological condition specified, in Part 1 of Schedule 1;...

Alcoholism, Appendicitis, Arteriosclerosis, Arthritis, Baldness, ‘Blood pressure, disorders of’, ‘Bust, underdevelopment of’, Cancer, Cataract, ‘Central nervous system, disorders of’, Diabetes, Diphtheria, Dropsy, Epilepsy, Gallstones, kidney stones, bladder stones, Gangrene, Glaucoma, Goitre, Heart disease, Infertility, Leukemia, ‘Menopause, disorders of’, ‘Menstrual flow, disorders of’, Mental disorders, Nephritis, Pernicious anaemia, Pleurisy, Pneumonia, Poliomyelitis, ‘Prostate gland, disorders of’, Septicaemia, Sexual impotence, Smallpox, Tetanus, Thrombosis, Trachoma, Tuberculosis, Tumours, Typhoid Fever, Ulcers of the gastro-intestinal tract, Venereal diseases.”^{16,17}

Two advanced searches were used to find any of the terms in the Medicines Act listed above, along with the word ‘acupuncture’; these focused on New Zealand sites only, and removed any mention of ‘animal’ or ‘vet’. Two searches were required because there is a maximum limit on the number of characters in the text of a Google search. Sponsored links and social media pages were not included. A targeted search against websites where Acupuncture NZ or NZASA Council members were listed as practitioners was also included. The two search queries used were:

site:nz acupuncture Alcoholism OR Appendicitis OR Arteriosclerosis OR Arthritis OR Baldness OR ‘Blood pressure’ OR Bust OR Cancer OR Cataract OR ‘Central nervous system’ OR Diabetes OR Diphtheria OR Dropsy OR Epilepsy OR Gallstones OR ‘kidney stones’ OR bladder stones OR Gangrene OR Glaucoma OR Goitre OR ‘Heart disease’ OR Infertility OR Leukemia -animal -vet

site:nz acupuncture ‘Mental disorder’ OR Menopause OR Menstrual OR Nephritis OR Pernicious OR anaemia OR Pleurisy OR Pneumonia OR Poliomyelitis OR ‘Prostate gland’ OR Septicaemia OR ‘Sexual impotence’ OR Smallpox OR Tetanus OR Thrombosis OR Trachoma OR Tuberculosis OR Tumours OR ‘Typhoid Fever’ OR ‘Ulcers gastro-intestinal tract’ OR Venereal -animal -vet

For each site that appeared in the search results for containing a reference to one or more of the relevant medical conditions, a

manual reading was performed of the site’s pages to ensure that a therapeutic claim was definitely being made, and if the claim was in the form of a testimonial. Words whose use were deemed to constitute ‘therapeutic’ claims were ‘prevent’, ‘alleviate’, ‘cure’, ‘reduce’ and ‘terminate’. Website content was explored by clicking every link that was found, up to a maximum of 40 pages and documents per website. This exploration was aided by a Google Chrome plugin called MultiHighlighter, which highlighted the relevant medical terms.

This search was in no way exhaustive; as the flow diagram (Figure 1) shows, 98 sites were found from the Google search, and a further eight were independently investigated because they belonged to board members of either the NZASA or Acupuncture NZ. One website was removed as it was not a New Zealand company despite using a .nz domain. Three sites were removed as the companies were no longer in business. After these exclusions, 102 eligible websites remained.

These 102 websites were monitored periodically from August 2016 until January 2017 for any changes; during this time, only minor changes were noticed. One business had closed their website during this period, and so the site was removed from the list.

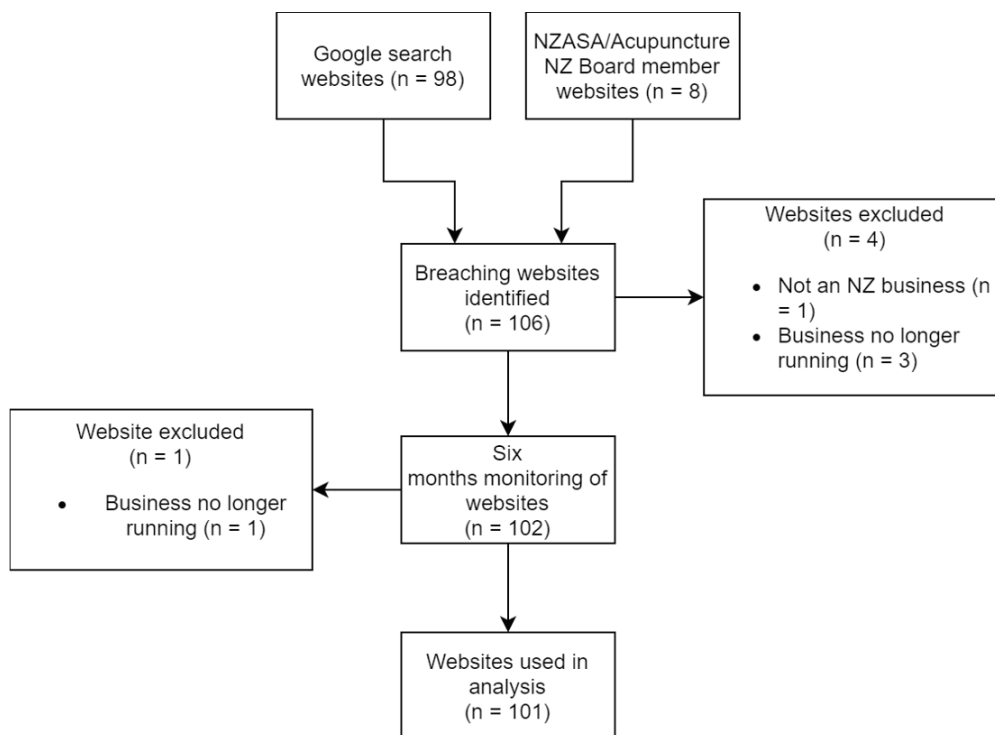
To work out the total percentage of likely breaching websites found for Acupuncture NZ and the NZASA, the total number of New Zealand acupuncturists’ websites needed to be estimated. To calculate this estimate, website URLs and business names were collected from member listings on the websites of Acupuncture NZ and the NZASA. Missing website URLs were filled in by manually looking up business names in a Google search. Duplicated businesses, websites that didn’t mention acupuncture, non-New Zealand businesses and websites that were offline were all removed. The NZASA’s records don’t require practitioners to enter a practice name, which may skew some of the results.

Results

The result of this effort was a list of 101 websites for acupuncture clinics based in New Zealand where at least one claim was made to be able to prevent, treat or cure a condition listed in the Medicines Act under Schedule 1. The majority of acupuncturists were registered with a New Zealand professional organisation 98/101 (97%), with some being registered to more than one.

Of the registered acupuncturists, Acupuncture NZ’s members accounted for

Figure 1: Flow diagram of acupuncture website search.



72 of the 101 websites that were found, the NZ Acupuncture Standards Authority's members were responsible for 28 websites, the Physiotherapy Board's members owned four websites, the Osteopathic Council's members owned four websites and the Medical Council of New Zealand's members owned a single website.

There are 44 terms listed in Part 1, Schedule 1 of the Medicines Act. Of the websites surveyed, 35/44 (80%) terms were used as shown on Table 1. The three most frequently occurring claims likely in breach of Section 58 of the Medicines Act were (1) treating/preventing mental illness; (2) treating/preventing infertility; and (3) treating/preventing arthritis. Combined, these occurred on 74/101 (73%) of the websites in this study. Furthermore, 34/101 (34%) of the websites had health testimonials, which are prohibited by the Medicines

Act,¹⁶ and were likely in breach of Section 58 of the Act.

There are 426 acupuncture businesses which have been listed with Acupuncture NZ by its members, and 217 of those businesses have a New Zealand acupuncture website advertising their services. Similarly, there are 100 acupuncture businesses listed NZASA by their members, and 72 associated business websites. Removing duplicate businesses, this gives a total of 274 acupuncture websites advertising the services of New Zealand acupuncturists as shown on Figure 2.

Six of the nine Acupuncture NZ council members' websites (year 2015/2016) and two of the five NZASA executive board members' websites (year 2015/2016) were likely in breach of the Act.

The data collected for this study is available on request from The Society for Science Based Healthcare.

Figure 2: Flow diagram of total estimated New Zealand registered acupuncture websites.

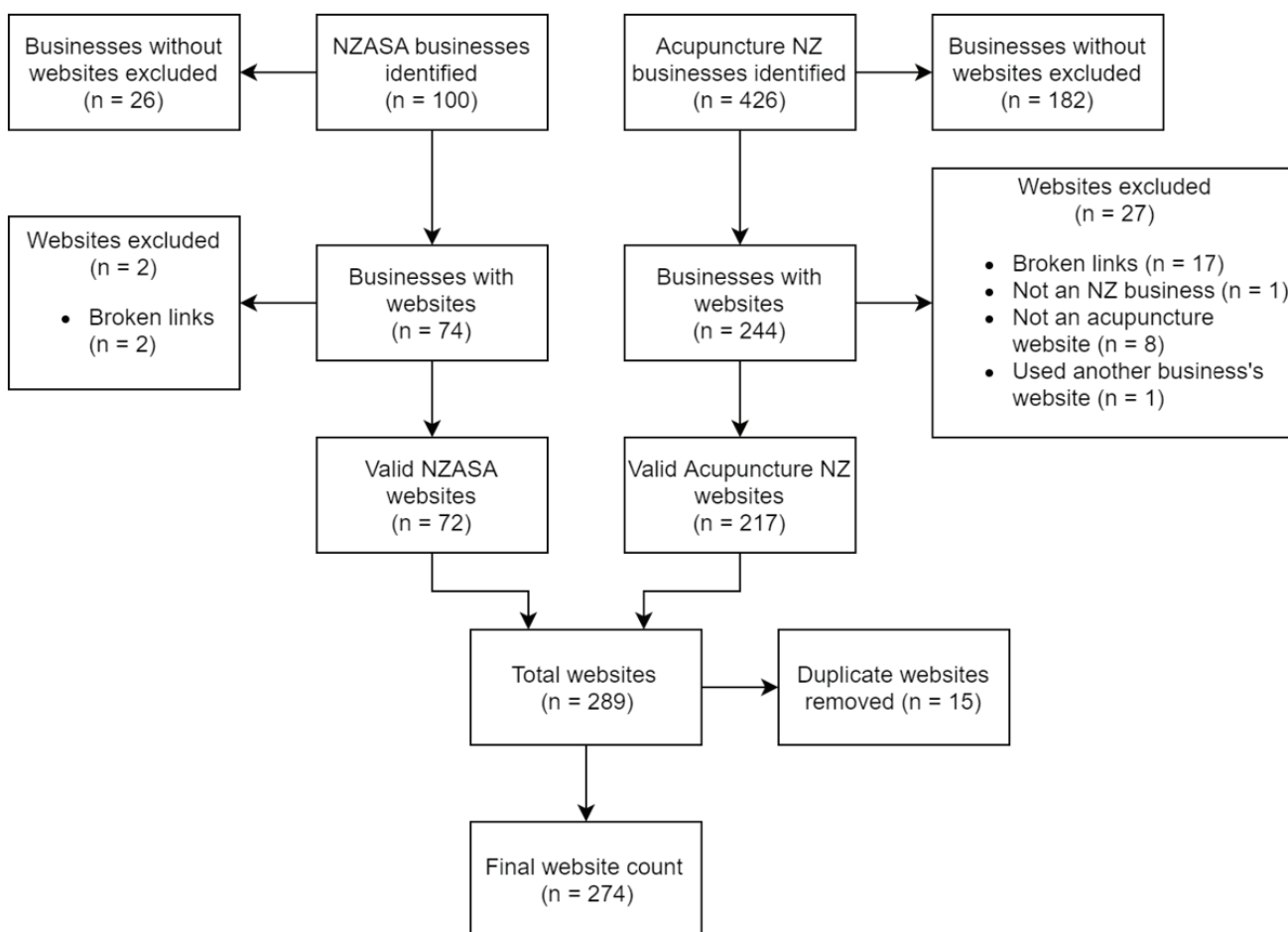


Table 1: The list of terms used, along with the number of websites with claims likely in breach of the Act.

Terms	Count	Percent
Mental disorders	97	96.0%
Infertility	85	84.2%
Arthritis	74	73.3%
Blood pressure	69	68.3%
Menstrual flow	55	54.5%
Central nervous system	45	44.6%
Sexual impotence	38	37.6%
Prostate gland	36	35.6%
Menopause	34	33.7%
Ulcers of the gastro-intestinal tract	30	29.7%
Diabetes	23	22.8%
Thrombosis	19	18.8%
Alcoholism	15	14.9%
Heart disease	14	13.9%
Pernicious anaemia	13	12.9%
Dropsy	13	12.9%
Tumours	12	11.9%
Cancer	11	10.9%
Baldness	9	8.9%
Gallstones	9	8.9%
Pleurisy	7	6.9%
Venereal diseases	6	5.9%
Epilepsy	6	5.9%
Poliomyelitis	5	5.0%
Bladder stones	5	5.0%
Kidney stones	4	4.0%
Arteriosclerosis	4	4.0%
Cataract	4	4.0%
Goitre	2	2.0%
Glaucoma	1	1.0%
Leukemia	1	1.0%
Nephritis	1	1.0%
Appendicitis	1	1.0%
Pneumonia	1	1.0%
Gangrene	1	1.0%

Unique terms: 35, Total terms: 576, Median terms: 7, Standard Deviation: 3.99

Discussion

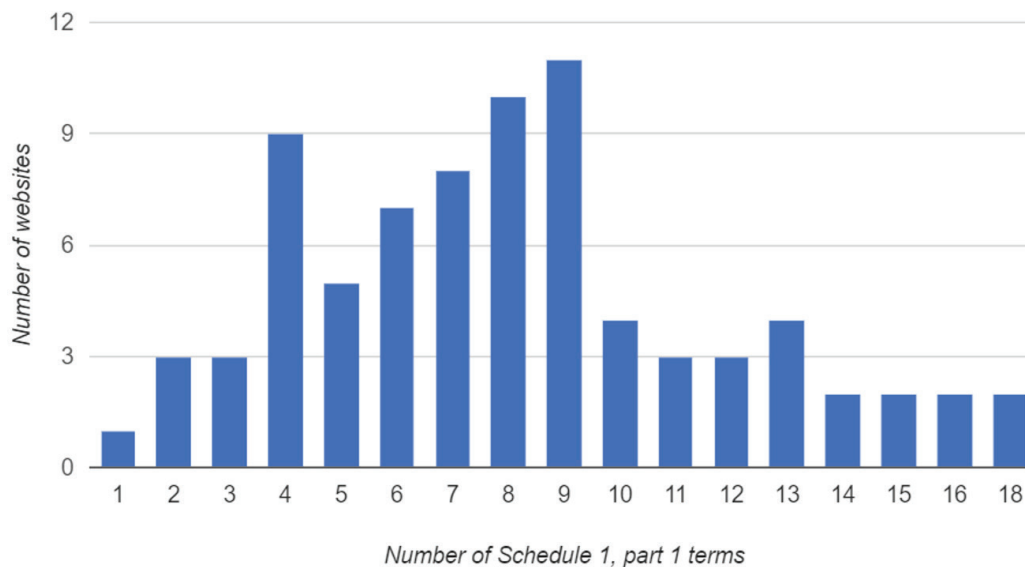
Of the businesses with websites that were surveyed, at least 39% of the sites belonging to NZASA-registered acupuncturists and 33% of websites of Acupuncture NZ-registered acupuncturists appeared to be in breach of the Act. This is a lower boundary, and is likely to be somewhat lower than the real total, as the search for websites making claims was not exhaustive. In addition, some of the sites that were checked may have produced false negatives due to misspellings of the medical conditions that were being searched for.

Acupuncturists who are eligible for ACC payments accounted for 97/101 (96%) of the total, as their practitioners were registered with a professional organisation which is recognised by ACC. ACC has rules regarding its ability to limit funding of providers under the Accident Compensation Regulations 2003. In funding any provider, these regulations require that ACC recognises any acupuncturist as long as they are a member of either Acupuncture NZ or NZASA, and have a current Annual Practising Certificate (APC). The expectation is that those professional bodies are responsible—through their registration and APC renewal processes—for providing ACC with an assurance that their members are safe and fit to practise. Where there are concerns about a practitioner, ACC can investigate and put in place certain conditions around invoicing, but they are ultimately reliant on these professional associations to suspend or remove APCs when it comes to formally ending any funding relationship (K Eland, personal communication, May, 2016).

ACC has conducted two reviews of acupuncture since 2011, finding limited or little evidence of effectiveness that it can help with mental health issues, and either inconclusive or insufficient evidence of benefit for musculoskeletal pain other than some positive evidence for chronic neck and shoulder pain.^{18,19}

The evidence presented in this report shows that New Zealand acupuncturists routinely claim much wider benefits for their practices than is justified by the evidence, or allowed by law. Acupuncture NZ and the NZASA appear to be failing to protect New Zealanders from potentially harmful misinformation.

Figure 3: Number of websites that are likely in breach of the Act plotted against the number of Schedule 1 conditions mentioned.



In the interests of public safety, these New Zealand professional bodies must take steps to ensure their members are abiding by the rules they agreed to follow when they joined. Both Acupuncture NZ and the NZASA should give clear direction to acupuncturists to remove any terms used in their advertising that breach Section 58(1)(a) of the Medicines Act, as well as claims to help any health condition where rigorous evidence of the efficacy of acupuncture treatment is lacking. Sanctions or suspension of membership would be an appropriate measure to take, until such

time as the owners of these websites ensure any therapeutic claims being made on their website are compliant with all the relevant rules and regulations of New Zealand. The least compliant acupuncturists may warrant referral to Medsafe or the Commerce Commission for prosecution.

Further study

It is hoped that new technologies such as Machine Learning may be employed in a future study to automate the process of finding websites that are in breach of the Medicines Act, not only for acupuncture but also for other types of alternative medicine.

Competing interests:

Nil.

Acknowledgements:

I would also like to thank Dr Jonathan Broadbent (PhD), Mark Honeychurch, Dr Alison Campbell (PhD), Dr Michael Foley (PhD) and Mark Hanna for their feedback on the manuscript.

Author information:

Daniel J Ryan, Committee Member, Society for Science Based Healthcare, Wellington.

Corresponding author:

Daniel J Ryan, Committee Member, Society for Science Based Healthcare, 78 Mercury Way, Whitby, Porirua, Wellington 5024.
daniel.ryan@sbh.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7430>

REFERENCES:

1. Pollock K. Alternative health therapies - Asian alternative-health therapies [Internet]. 2011 [cited 1 June 2017]. Te Ara - the Encyclopedia of New Zealand. Available from: <http://www.teara.govt.nz/en/alternative-health-therapies/page-5>
2. 2014/2015 ACC acupuncture spending [Internet]. 2017 [cited 18 February 2017]. FYI. Available from: <http://fyi.org.nz/request/3043/response/10026/attach/html/2/Response%20OIA%20Hanna%20M%20Acupuncture%20spending.pdf.html>
3. Gilbey A, Ernst E, Tani K. A systematic review of reviews of systematic reviews of acupuncture. *Focus on Alternative and Complementary Therapies*. 2013; 18:8–18.
4. Exercise not acupuncture for people with low back pain says NICE in draft guidance. The National Institute for Health and Care Excellence [Internet]. The National Institute for Health and Care Excellence. 2016 [cited 20 February 2017]. Available from: <http://www.nice.org.uk/news/press-and-media/exercise-not-acupuncture-for-people-with-low-back-pain-says-nice-in-draft-guidance>
5. Andrew AC, Agatha CP, Belinda AJ, ØRjan MG, Richard H, Steve C, Peter WM, Helene LM. Electrical properties of acupuncture points and meridians: A systematic review. *Bioelectromagnetics*. 2008; 29(4):245–256.
6. David GH. Integrative oncology: really the best of both worlds? *Nature Reviews Cancer*. 2014; 14:692–700.
7. Vickers A, Goyal N, Harland R, Rees R. Do Certain Countries Produce Only Positive Results? A Systematic Review of Controlled Trials. *Controlled Clinical Trials*. 1998; 19(2):159–166.
8. Colquhoun D, Novella SP. Acupuncture is a theatrical placebo: the end of a myth. *Anesthesia & Analgesia*. 2013; 116(6):1360–1363.
9. Miller LE, Stewart ME. The blind leading the blind: use and misuse of blinding in randomized controlled trials. *Contemp Clin Trials*. 2011; 32(2):240–3.
10. Section 3 of the Accident Compensation (Liability to Pay or Contribute to Cost of Treatment) Regulations 2003 [Internet]. Legislation.govt.nz. 2017 [cited 24 February 2017]. Available from: <http://www.legislation.govt.nz/regulation/public/2003/0388/2.0/DLM235783.html>
11. Patel A, Toossi V. Traditional Chinese medicine practitioners in New Zealand: differences associated with being a practitioner in New Zealand compared to China. *NZ Med J*. 2016. Available from: <http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1444-28-october-2016/7045>
12. Frequently Asked Questions [Internet]. 2017 [cited 6 August 2017]. Acupuncture NZ. Available from: <http://www.acupuncture.org.nz/what-is-acupuncture/frequently-asked-questins>
13. Conditions [Internet]. 2017 [cited 6 August 2017]. Acupuncture NZ. Available from: <http://www.acupuncture.org.nz/what-is-acupuncture/what-conditions-can-be-treated-with-acupuncture/>
14. O'Sullivan J, Mar CD, Barnes A, Marron LJ. Acupuncture in Australia. *Focus on Alternative and Complementary Therapies*. 2016; 21:22–24.
15. ASA complaints against acupuncturists that were making bad therapeutic claims [Internet]. ASA - Advertising Standards Authority. 2017 [cited 18 February 2017]. Available from: <http://www.asa.co.nz/backend/documents/2016/05/13/16029.pdf> http://old.asa.co.nz/display.php?ascb_number=15428 http://old.asa.co.nz/display.php?ascb_number=15384 http://old.asa.co.nz/display.php?ascb_number=15255 http://old.asa.co.nz/display.php?ascb_number=15251 http://old.asa.co.nz/display.php?ascb_number=14646 http://old.asa.co.nz/display.php?ascb_number=14645 http://old.asa.co.nz/display.php?ascb_number=14536 http://old.asa.co.nz/display.php?ascb_number=14461 http://old.asa.co.nz/display.php?ascb_number=14405 http://old.asa.co.nz/display.php?ascb_number=14316 http://old.asa.co.nz/display.php?ascb_number=14192 http://old.asa.co.nz/display.php?ascb_number=13448 http://old.asa.co.nz/display.php?ascb_number=12493
16. Section 58 of the Medicines Act 1981 [Internet]. Legislation.govt.nz. 2017 [cited 18 February 2017]. Available from: www.legislation.govt.nz/act/public/1981/0118/latest/DLM56046.html
17. Part 1, Schedule 1, Section 58 of the Medicines Act 1981 [Internet]. Legislation.govt.nz. 2017 [cited 18 February 2017]. Available from: www.legislation.govt.nz/act/public/1981/0118/latest/DLM57001.html

18. Dara S. Effectiveness of acupuncture in selected mental health conditions. [Internet]. Accident Compensation Corporation. Auckland 2014. Available from: <http://www.acc.co.nz/assets/research/Evidence-based-review-acupuncture-and-mental-health-conditions.pdf>
19. Accident Compensation Corporation. Pragmatic Evidence Based Review: The efficacy of acupuncture in the management of musculoskeletal pain. Accident Compensation Corporation. Auckland 2011. Available from: <http://fyi.org.nz/request/1800/response/6586/attach/5/2%20%20120113Acupuncture-Final%20extrev%20Redacted.pdf>

Exposure to respirable crystalline silica in the construction industry—do we have a problem?

David McLean, Bill Glass, Andrea 't Mannetje, Jeroen Douwes

ABSTRACT

AIMS: To assess personal exposure to respirable dust and respirable crystalline silica (RCS) in New Zealand construction workers.

METHODS: In a pilot study, 39 personal samples were collected from a cross-section of workers engaged in a range of tasks performed on construction sites that were expected to entail exposure to respirable crystalline silica. Nine static samples were taken at locations adjacent to these tasks. Particle size-selective sampling heads were used to collect the respirable fraction of airborne particulates. Dust concentrations were determined gravimetrically, while crystalline silica was analysed using x-ray diffraction.

RESULTS: Almost half of the personal crystalline silica samples exceeded the New Zealand Workplace Exposure Standard (NZ WES), while 56% exceeded the more stringent international recommendation (ACGIH TLV). The tasks associated with the highest RCS levels were concrete grinding and cutting. Two of four static samples collected close to (silica-containing) Linea board cutting exceeded the ACGIH TLV for RCS, indicating the potential for bystander exposure.

CONCLUSIONS: A large proportion of workers performing common tasks in the construction industry may be exposed to levels of respirable dust and crystalline silica exceeding national standards and international recommendations. These results suggest that workers in this industry may be at risk of developing silica-related diseases, including silicosis, lung cancer, COPD and chronic renal disease. Action is required to improve dust control to reduce silica exposure and the associated health risks.

Although often thought of as an historical issue, respirable crystalline silica (RCS) remains one of the most common occupational exposures worldwide.^{1,2} Silicosis is a progressive and irreversible fibrotic lung disease, which can be either acute after very high exposure to RCS or chronic, manifesting 10 to 30 years after continued exposure at lower levels. RCS exposure occurs mainly in occupational settings (including construction) wherever materials such as rock, gravel, sand, concrete or brick, or the newer high-silica content building materials such as Linea board, are mechanically broken down through cutting, grinding, crushing, drilling or abrasive blasting. In the past there has been high silica exposure in the New Zealand mining industry, with 1,576 pensions

awarded between 1915 and 1938 to miners suffering from silicosis.³ RCS was classified by the International Agency for Research on Cancer as an IARC Group 1 carcinogen (sufficient evidence of carcinogenicity in humans) in 1997,⁴⁻⁶ and more recently evidence has been accumulating showing that it is also associated with autoimmune conditions, including rheumatoid arthritis, scleroderma, Sjögren's syndrome and systemic lupus erythematosus,^{7,8} and with chronic renal disease.⁹ For this reason the New Zealand Workplace Exposure Standard (NZ WES) for RCS was updated to 0.1mg/m³ as recently as 2016,¹⁰ and there is an even more stringent internationally-recommended exposure limit (ACGIH TLV) of 0.025mg/m³.^{11,12}

Studies on work-related RCS exposures in New Zealand workers are rare. A survey of

high-risk extractive industries (ie, quarries, lime works, gold mines and tunnels) in the late 1990s found average (geometric mean) levels of RCS ranging from 0.05mg/m³ in quarries to 0.09mg/m³ in tunnels, with highs of 0.57mg/m³ and 1.29mg/m³ respectively,¹³ but current levels of exposure (and associated health risks) in the New Zealand construction industry are not known. We report here the results of a pilot study of respirable dust and silica exposure levels in construction workers involved in the post-earthquake Canterbury rebuild.

Methods

Construction companies working in the Christchurch rebuild were invited to participate in this pilot study of respirable dust and respirable crystalline silica exposure. In those that agreed, personal respirable dust samples were taken on 39 workers engaged in a range of tasks performed on construction sites that were expected to entail silica exposure. These tasks included: driving Bobcats and diggers; jackhammering, polishing, grinding, drilling or crushing concrete; cutting concrete or Linea board; and general labouring. All samples were taken according to the method AS2985, 2009 Workplace Atmospheres-Method for Sampling and Gravimetric Determination of Respirable Dust using portable pumps set at an airflow rate of 2.2 l/min attached to Higgins Dewell cyclones (Casella) and 25mm PVC filters. Sampling time varied according to work requirements, but most samples taken were for periods of five to eight hours. Using the same method and equipment nine static or area samples were also collected in locations adjacent to the above tasks.

Gravimetric measurements were conducted using a Sartorius balance with a resolution of 0.01mg to calculate respirable dust levels. Crystalline silica was analysed on each filter using x-ray diffraction (XRD) according to the US National Institute of Occupational Safety and Health Manual of Analytical Methods; Method 7500 to calculate airborne silica concentrations. The limit of detection for crystalline silica was 5µg. When filters were overloaded, analyses were conducted as if samples

were solid powders rather than dust on filters. Five personal samples (representing general labouring tasks (n=3), drilling (n=2), jackhammering (n=1) and digging (n=1)) had very low dust levels so these were not assayed for crystalline silica. For the descriptive analyses of silica content in these cases it was assumed that silica levels were half of the lowest observed concentrations in this study, ie, 1.05mcg/m³. As dust and RCS exposure data followed a log-normal distribution, results were expressed as geometric means (GM) and geometric standard deviations (GSD), stratified by work task.

Results

The personal respirable dust levels ranged from below the limit of detection to 47.4mg/m³, with 12 of the 39 samples (ie, 31%) exceeding the NZ WES and the ACGIH TLV recommendation of 3mg/m³. The majority of samples exceeding this limit were collected from workers polishing or grinding concrete, with average (GM) levels of 4.2 and 5.5mg/m³ respectively. The average levels were considerably lower for other tasks, although samples taken from workers drilling concrete and cutting Linea board both included samples above 3mg/m³. None of the nine static or area samples exceeded the respirable dust WES. The personal RCS levels ranged from below the limit of detection to a maximum of 4.767mg/m³. In total, 17 of the personal samples (44%) exceeded the NZ WES of 0.1mg/m³, while 22 (56%) exceeded the ACGIH TLV of 0.025mg/m³ (Table 1). The highest levels were observed in concrete polishers and grinders with average (GM) concentrations of 0.306mg/m³ and 0.657mg/m³ respectively. While the levels measured in personal sampling varied widely, even within similar tasks, each task assessed included at least one sample in excess of the ACGIH TLV and in most the NZ WES was also exceeded at least once. None of the nine static RCS samples exceeded the NZ WES, but two out of four samples taken near the cutting of silica-containing Linea board did exceed the ACGIH TLV, indicating the potential for bystander exposure.

Table 1: Personal respirable crystalline silica levels by work task.

Work task	N	GM (mg/m ³)	GSD	Range (mg/m ³)	>NZ-WES (%)	>ACGIH-TLV (%)
Grinding concrete	10	0.657	5.4	0.012–3.207	90	90
Polishing concrete	5	0.306	21.3	0.003–4.767	60	80
Cutting concrete	1	0.100	-	-	100	100
Crushing concrete	3	0.026	4.7	0.004–0.074	0	67
Cutting Linea board	4	0.017	11.2	0.002–0.486	25	25
Bobcat/digger driving	5	0.009	8.0	0.001–0.143	20	40
Drilling concrete	4	0.007*	23.7	0.001–0.762	25	25
Jackhammering	2	0.006	12.4	0.001–0.037	0	50
General labouring	5	0.004*	10.2	0.001–0.222	20	20

N=Number of samples. GM= Geometric mean. GSD=Geometric standard deviation.

*Non-detectable samples, assumed to be 0.001mg/m³, were included in the calculation of the GM.

Discussion

This study has shown that both respirable dust and crystalline silica exposures in workers conducting common tasks in the construction sector regularly exceed the NZ WES and the ACGIH TLV recommendation. Our results may not be representative of average levels of exposure in all construction workers, but the silica levels measured are consistent with those reported for workers performing similar construction industry tasks in studies from other countries.^{14–16} In addition, a recent large cross-sectional survey of the Australian working population has shown that 80% (95% CI 69.1, 90.9) of construction workers have some exposure to RCS, and that 61.8% (95% CI 48.6, 75.1) have exposures in excess of the WES.¹⁷ From the results of our study, the exposure levels reported in other countries, and the Australian exposure prevalence, estimates it would be reasonable to assume that a substantial proportion of the New Zealand construction industry workforce experiences excessive exposure to RCS. This is of concern given that the construction industry is the fifth largest sector in the New Zealand economy, employing >8% of the workforce with more than 193,000 workers.¹⁸

When we compared the results of this survey with the current NZ WES of 0.1mg/m³ for RCS we found that 44% of samples

exceeded this standard. Occupational exposure limits are set in most countries after review of the available epidemiological evidence and consultation with industry. For example, the Australian exposure standard was reduced from 0.2 to 0.1mg/m³ in 2004, the UK Workplace Exposure Limit was reduced from 0.3 to 0.1mg/m³ in 2006, and in the US the enforceable Occupational Safety and Health Administration Permissible Exposure Limit was set at 0.05mg/m³ in 2016. It should be noted that the methods available for sampling and analysis of personal worker exposures have an effective lower order of detection of 0.02mg/m³ for quartz and 0.04mg/m³ for cristobalite.¹⁹ Although the NZ WES was updated in 2016,¹⁰ it has not taken full account of the current epidemiological evidence showing significant adverse health effects at much lower levels. For example, a risk assessment based on international epidemiological data has indicated that 45 years of occupational exposure at the current NZ WES of 0.1mg/m³ is associated with a lifetime excess mortality risk of 13 to 60 per 1,000 workers for lung cancer, 11 to 83 per 1,000 for silicosis and non-malignant respiratory disease, and 39 per 1,000 for renal disease.^{11,12} The excess lung cancer risk in particular is much higher than the residual risk of 1 per 10,000 to 1 per 1,000,000 that is generally considered “acceptable” by organisations that perform quantitative risk assessment to derive occu-

pational exposure limits.^{20,21} We consider it more appropriate, therefore, to compare these results with the more stringent ACGIH TLV of 0.025mg/m³, which was exceeded by 56% of the samples taken. Even at this level of exposure, risks remain, with lifetime mortality risk estimated to range from 3 to 23 per 1,000 workers for lung cancer, 4 to 22 per 1,000 for silicosis and non-malignant respiratory disease and 25 per 1,000 for renal disease.¹¹ While studies on work-related exposures and health outcomes in New Zealand construction workers are rare, there is some evidence of increased risk of lung cancer in this sector. One New Zealand study of mortality by occupation found an increased risk of lung cancer (SIR=130, 95% CI 120–433) in bricklayers and carpenters.²² A more recent New Zealand population-based lung cancer case-control study showed that, after adjustment for smoking, both builder's labourers (OR=3.2,

95% CI 1.4–7.1) and bricklayers/stonemasons (OR=5.7, 95% CI 1.2–25.8) had significantly increased risk of lung cancer.²³

In conclusion, in addition to the well-recognised risk to workers in mining, quarrying and sandblasting, this pilot study has shown that a sizeable proportion of workers performing routine tasks in the construction industry in New Zealand are also likely to be exposed to RCS levels that exceed national and international standards. These results suggest that workers in this industry may be at risk of developing silica-related diseases, including silicosis, lung cancer, COPD and chronic renal disease, and that there is a need for action to reduce exposure levels. As with all occupational diseases, the silica-related health effects are largely preventable provided that airborne exposures are minimised and/or workers are adequately protected against inhalation.

Competing interests:

Nil.

Acknowledgements:

This study was funded by WorkSafe New Zealand, and the sampling and analysis were conducted by K2 Environmental.

Author information:

David McLean, Senior Research Officer, Centre for Public Health Research, Massey University; Bill Glass, Professorial Research Fellow, Centre for Public Health Research, Massey University and Departmental Medical Practitioner, WorkSafe New Zealand; Andrea 't Mannelje, Associate Professor, Centre for Public Health Research, Massey University; Jeroen Douwes, Professor and Director, Centre for Public Health Research, Massey University.

Corresponding author:

Dr David McLean, Centre for Public Health Research, Massey University, PO Box 756, Wellington.

d.j.mclean@massey.ac.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7431>

REFERENCES:

1. Steenland K. One agent, many diseases: Exposure-response data and comparative risks of different outcomes following silica exposure. *Am J Ind Med.* 2005; 48:16–23.
2. Leung CC, Tak Sun Yu I, Chen W. Silicosis. *Lancet.* 2012; 379:2008–18.
3. Silicosis: Report of the Interdepartmental Committee, Bulletin No 57, Department of
4. International Agency for Research on Cancer. Monographs on the evaluation of silicates, coal dust and

- para-aramid fibrils. Vol. 68. Lyon: World Health Organisation, IARC, 1997.
5. Cassidy A, 't Mannetje A, van Tongeren M, et al. Occupational exposure to crystalline silica and risk of lung cancer. A multicentre Case-Control study in Europe. *Epidemiology*. 2007; 18(1):36–43.
 6. International Agency for Research on Cancer (IARC): A Review of Human Carcinogens. Part C: Arsenic, Metals, Fibres and Dusts. Lyon. France: IARC, 2012.
 7. Parks C, Conrad K, Cooper G. Occupational exposure to crystalline silica and autoimmune disease. *Environ Health Perspect*. 1999; 107(5):793–802.
 8. Pollard KM. Silica, Silicosis, and Autoimmunity. *Front Immunol*. 2016; 7(97).
 9. Millerick-May M, Schrauben S, Reilly M, Rosenman K. Silicosis and chronic renal disease. *Am J Ind Med*. 2015; 58:730–6.
 10. Workplace Exposure Standards and Biological Exposure Indices. June 2016, 8th Edition, Work-Safe New Zealand/Mahi Haumarua Aotearoa.
 11. Occupational Safety and Health Administration (OSHA). Occupational exposure to respirable crystalline silica-Review of health effects literature and preliminary quantitative risk assessment. OSHA-2010-0034.
 12. American Conference of Governmental Industrial Hygienists: 2015 Threshold Limit Values and Biological Exposure Indices. Cincinnati, Ohio. ACGIH, 2015.
 13. Glass WI, McLean D, Armstrong R, et al. Respiratory health and silica dust levels in the Extractive Industry. Occupational Safety and Health Service, Department of Labour. Occupational Health Report Series, Number 9, 2003.
 14. Van Deursen E, Pronk A, Spaan S, et al. Quartz and respirable dust in the Dutch construction industry: a baseline exposure assessment as part of a multidimensional intervention approach. *Ann Occup Hyg*. 2014; 58:724–38.
 15. Beaudry C, Lavoué J, Sauvé J-F, et al. Occupational exposure to silica in construction workers: A literature-based exposure database. *J Occup Environ Hyg*. 2013; 10:71–77.
 16. Sauvé J-F, Beaudry C, Begin D, et al. Statistical modelling of crystalline silica exposure by trade in the construction industry using a database compiled from the literature. *J Environ Monit*. 2012; 14:2512–20.
 17. Si S, Carey R, Reid A, et al. The Australian Workforce Exposures Study: Prevalence of occupational exposure to respirable crystalline silica. *Ann Occup Hyg*. 2016; 60(5):631–7.
 18. <http://www.worksafe.govt.nz/worksafe/research/health-and-safety-data/pdf-and-documents-library/construction-sector-by-numbers.pdf>
 19. UK Health and Safety Laboratory. Methods for the determination of hazardous substances. 101 Crystalline silica in respirable airborne dusts. HSE Books. Feb 2005.
 20. Bolt HM, Huici-Montagud A. Strategy of the scientific committee on occupational exposure limits (SCOEL) in the derivation of occupational exposure limits for carcinogens and mutagens. *Arch Toxicol*. 2008; 82(1): 61–64.
 21. Wheeler MW, Park RM, Bailer AJ, et al. Historical context and recent advances in exposure-response estimation for deriving occupational exposure limits. *J Occup Environ Hyg*. 2015; 12:S7–S17.
 22. Firth HM, Cooke KR, Herbison GP. Male cancer incidence by occupation: New Zealand 1972–84. *Int J Epidemiol*. 1996; 25(1):14–21.
 23. Corbin M, McLean D, 't Mannetje A, et al. Lung cancer and occupation: A New Zealand cancer registry-based case-control study. *Am J Ind Med*. 2011; 54:89–101.

Physician advocacy in Western medicine: a 21st century challenge

Philip Bagshaw, Pauline Barnett

ABSTRACT

Physician advocacy occurs when doctors speak up for the health and healthcare of patients and communities. Historically, this was strong in some Western countries with doctors finding that it enhanced their authority, prestige and power. But it weakened in the 20th century when the biomedical model of health triumphed and medicine became a dominant profession.

In the second part of the 20th century, this dominance was threatened by political, technological and socioeconomic forces. These weakened medicine's state support, brought it under managerial control and undermined the social contract on which trust between doctors and the community was based. Defence of the profession was assumed by medical colleges, societies and associations. They had some success in retaining professional autonomy but did not undertake open advocacy, particularly on social justice issues, and did not therefore enhance their standing in the community.

Opinion is divided on the level of advocacy that it is ethically proper for the medical profession to employ. Some contend doctors should only advise authorities when expert opinion is requested. Others contend doctors should speak out proactively on all health issues, and that collective action of this type is a hallmark of professionalism. This lack of consensus needs to be debated.

Recent developments such as clinical leadership have not revitalised physician advocacy. However, continued deterioration of the UK National Health Service has led some English medical colleges to take up open advocacy in its defence. It is to be seen whether medical colleges elsewhere follow suit, as and when their healthcare systems are similarly threatened.

What is physician advocacy?

Physician advocacy (PA) occurs when doctors speak up for the health and healthcare of patients and communities.¹ There is an extensive scientific literature on PA, originating mostly in the US, UK and some commonwealth countries, much of which is relevant to New Zealand. There is, however, continuing debate on the role of PA and how it should be incorporated into professional practice.²⁻⁵

PA is a complex topic, for which no accepted model has been published.⁶ We see it as best described under three domains: subjects; methods; and moral/ethical tensions. The **subjects** are traditionally divided into cases (doctors advocating for the needs of individuals or group of patients; a role now frequently assumed by nurses

and others) and causes (public health specialists and others advocating for population health measures).⁶

The **methods** of PA can be divided into three distinct types: policy advice, and what we call here 'closed' and 'open' PA. First, there is policy advice which is requested by governments and other organisations from medical experts. Second, is closed PA when doctors advocate for cases or causes with authorities behind closed doors, largely unknown to the general public. Lastly, there is open PA, when doctors advocate with authorities for cases and causes employing open communication, frequently using the public media.⁶ The 21st century challenge is to decide which aspects of PA are legitimate functions, if not obligations, of medical professionalism.

The **moral and ethical tensions** frequently associated with PA reflect the balance between, at one end of the spectrum, complete doctor self-interest and pure altruism at the other end,⁷ between the relative power of physician advocates and the beneficiaries of advocacy,⁸ and the balance between individual and group benefit arising from advocacy.⁹ This last commonly involves the issue of distributive justice and how resources can be allocated to ensure equity within populations.

The rise and fall of physician advocacy

PA has a long history in Western medicine, mentioned first in Hippocratic writings.¹⁰ In modern times, it began in Europe, including the UK, then spread to North America but changed with prevailing culture and circumstances.^{6,11} During the 19th century, when medical treatments were often ineffective or harmful, doctors built up the trust of the community by frequently engaging in PA on issues concerning both cases and causes.¹² This added to their authority and status, giving them some degree of autonomy over their own area of work. Rudolf Virchow was the embodiment of 19th century PA.¹³ Besides being one of the fathers of modern pathology he was also a man who led an “extraordinarily civic-oriented life”,⁶ and frequently engaged in both open and closed PA. He famously captured the spirit of the time when he said “Medicine is a social science, and politics nothing but medicine on a grand scale.”¹⁴

During the first half of the 20th century, the situation changed. Medicine became more scientific, effective and safe, and a period began that became known as the “golden age of doctoring”.^{15,16} It was also the time, however, when PA started to decline. Medicine had become a dominant profession with (i) state-protected authority over its own area of work; (ii) control over associated areas of work; and (iii) massive public support and trust.¹⁷ As explained by Richard and Sylvia Cruess,¹⁸ this last was underpinned by a strong social contract, a largely implicit agreement between the medical profession and society about the expectations and obligations each had of the other. With the success of the individual biomedical model of health, doctors, as purveyors of effective medical care,

occupied a powerful and secure position in society. In this environment, doctors saw PA as unnecessary, undesirable and politically risky, and tended to withdraw from participation.^{19,20}

The exception to the withdrawal from PA was the persistence of advocacy on some key public health issues of the time. This included, in the second half of the 20th century, physician advocacy against nuclear weapons²¹ and tobacco use,²² and into the 21st century advocacy for regulation of obesogenic products²³ and addressing climate change and health.²⁴ These initiatives, while generally led by public health physicians, have recently become increasingly multidisciplinary. When clinicians chose to become involved, they were encouraged by the perception that these issues could be seen as ‘safely distant’ from areas of employment and clinical practice. In our view, they did not, therefore, invite the risks to personal position that raising patient safety issues, challenging local resource decisions or criticising institutional management might carry.

Challenges to medicine and the profession’s response

In the second half of the 20th century, the dominance of the medical profession was challenged by countervailing political, technological and socioeconomic forces.¹⁵ First, there was increased demand for healthcare, fuelled by developing technology, increased medicalisation and consumer preferences. This was accompanied by governments and private sector organisations, such as insurance companies, making funds available to meet this demand. By the 1970s, ‘New Right’ ideologies had become dominant, endorsing a corporate or managerialist model for managing the tension between growing demand and available funds. In this model, doctors became ‘proletarianised’, that is, they became ‘workers’ subject to the managerial control of company or government rules, and no longer independent professionals.¹⁵ In the predominantly privately funded US, doctors in both secondary and primary care experienced similar early loss of autonomy through corporate managerialism.¹⁵ In countries with stronger public funding, this control occurred later and in New Zealand, for example, general practitioners maintained

relative autonomy through private practice until the late 1990s.²⁵

At the same time, as described by Marie Haug, 'de-professionalisation' of the medical profession occurred.²⁶ Technological advances, such as the Internet, which made previously privileged information freely available to the public, were accompanied by the socioeconomic changes that led to the establishment of an increasingly consumer-oriented medical marketplace. Furthermore, the rise of 'Rights' organisations (in women's health, psychiatric care, disability services, etc) increased public awareness that medical self-regulation was failing.²⁷ The combined effects of proletarianisation and de-professionalisation were failure of the social contract, loss of public trust, loss of state support, reduced autonomy and power, and public debate about the role of the medical profession in society.²⁸

The medical profession proved remarkably resilient to these challenges.²⁸ In a series of seminal publications, Eliot Freidson described how the profession strengthened itself with respect to governments by a process of restratification, becoming more hierarchical and bureaucratic.²⁸ He described two emergent groups that he called controlling elites, which exercised control over their much larger rank-and-file membership. 'Knowledge elites' came from the universities and other academic institutions, and controlled the content of medical knowledge. 'Administrative elites' came from the medical colleges, societies and associations, and ensured, as carefully as any professional or craft-based guild ever did, that the activities of their organisations reflected and protected the interests of their rank-and-file members.²⁹

In order to reassure governments that the medical profession was capable of self-regulation, the medical colleges and other controlling elites started programmes of continuing education and professional development, and supported initiatives such as evidence-based medicine and the development of clinical guidelines. In these ways they retained some degree of professional autonomy but at a cost to the clinical autonomy of their rank-and-file.^{30,31} They had only limited success, however, in countering

state and corporate dominance of healthcare, and maintaining control over their own areas of work.³⁰ Furthermore, they rarely used open PA and avoided comment on social justice issues, thereby failing to strengthen their relationship with the community or increase public support.³² By the 21st century, the controlling elites had become increasingly specialised, stratified and dispersed throughout the medical workforce, occupying professional-managerial hybrid positions in both public and private healthcare organisations.³³ They have therefore become unlikely to engage in open PA because their blurred employment boundaries lead to divided loyalties, and they are often gagged by employment contracts, commercial sensitivity issues and concerns about personal professional consequences.

Prospects for physician advocacy

It was expected that recent developments such as new professionalism and clinical leadership might enhance professional autonomy and even PA. So far, published evidence shows some increased influence on low-level control over work activities but no increase in PA.³⁴ Many health systems remain in a continual state of change, with persistent unmet healthcare need.^{35,36} Doctors are well-qualified to advocate on these issues, and there are examples where they have done so with some degree of success.^{37,38} In New Zealand, the 1997 report entitled "Patients are Dying" (unpublished but often cited) is an example of effective PA.³⁹ A recent example of publicised PA occurred when local orthopaedic surgeons complained Waikato Hospital was an unsafe place for elective surgery.⁴⁰

As noted above, there is still a lack of consensus in the medical profession on what type of PA is appropriate and who should do it. There are two schools of thought, as demonstrated by the reactions to the 9/11 attacks on the Twin Towers in New York. At that time, an editorial in the *New England Journal of Medicine* implored doctors not to react to terrorism but to get on with their jobs.⁴¹ In contrast, an editorial in the *Lancet* argued that health professionals should be concerned with prevention as well as healing, and medicine cannot escape politics.⁴²

One school of thought on PA has been well espoused by Thomas Huddle of the University of Alabama.⁴³ He claimed that ‘traditional medical ethics’ requires competent and ethical performance but that doctors should only provide advice when asked. He argued that PA is not a component of professionalism and that it should not be fostered or taught in academic institutions. Another supporter of this ‘traditional medical ethics’ position was the late, well-known ethicist Edmund Pellegrino who said “...the assertion of some progressives that activism and public policy can, or should, displace professional ethics is mistaken.”³ Other commentators have pointed out that doctors are too busy to engage in PA or are likely to engage policy makers to advance their own self-interests.¹ Research from the US⁴⁴ and Australia⁴⁵ showed ambivalence to the concept of social justice among some of the medical rank-and-file.

In opposition to the traditional medical ethics approach is the open advocacy school. This has been espoused by Russell Gruen and colleagues who suggested that PA is a professional responsibility and includes political advocacy not only for public health but also for social justice issues, including health resourcing and inequalities.⁴⁶ They claimed that individual action is laudable, but collective action is a hallmark of professionalism and an opportunity to regain public trust. There is support for the open advocacy school from some senior medical academics. For example, Mark Earnest from the University of Colorado and colleagues produced a widely-quoted definition for PA as: “Physicians promoting those social, economic and political changes that ameliorate the suffering and threats to human health and wellbeing that doctors identify through their professional work and expertise”.¹ The debate on PA continues, with a recent exchange between Jon Tilburt⁵ who argued that doctors must uphold the best interests of patients while ensuring the just use of health resources, and Huddle⁴ who argued that these cannot be reconciled. Despite this tension, PA is now a successful part of the undergraduate and postgraduate curricula in many universities, particularly in the US, and under some university charters it is both a right and responsibility of staff to speak out in the public interest

in their areas of expertise.⁴⁷ Martin McKee and colleagues agreed, noting that raising so-called ‘political’ issues, such as the consequences of conflict and the precursors of Type 2 diabetes, in fact appropriately draws attention to available scientific evidence.⁴⁸

In recent times, individual or small groups of healthcare professionals have raised concerns over serious health issues that were well known to, but largely ignored by, the wider medical community.^{49,50} These people, sometimes referred to as whistle-blowers, have often paid a high price for their actions; they have even occasionally been censured by their own disciplinary bodies.⁵⁰ In future it is likely such debates and disclosures will increasingly occur through blogs and other social media,⁵¹ particularly if such issues are not addressed by the relevant controlling elites.

It is noteworthy that some of the medical colleges in the UK have recently become exponents of open PA, in response to current threats to the future of the UK National Health Service. For example, the Royal College of Surgeons of England now issues a weekly political update, which records when they have managed to get their concerns mentioned in the regional or national public media.⁵² Furthermore, the Royal College of Physicians of London, considered by some to be a quite conservative organisation, has joined in the public fight. They have realised that contemporary politics is less about ‘leadership’ and more about ‘followship’ (ie, pursuing the vagaries of shifting popular public opinion),⁵³ and so open PA is becoming increasingly influential with governments. They have therefore recently released a public document “Underfunded, Underdoctored, Overstretched: The NHS in 2016”.⁵⁴ It will be interesting to see whether the controlling elites in other countries act in this way when their healthcare systems are similarly overtly threatened.

The history of PA has some important lessons for us on the relationships among doctors, patients, society, governments and health authorities. The role of PA has evolved over the last two centuries along with the political, technological and socio-economic landscape of Western society. The challenge for doctors, individually and collectively, is to debate the vital question of whether or not PA is a desirable or even

obligatory component of medical professionalism. If it is, then PA should be a core topic in undergraduate and postgraduate medical curricula. Despite the extensive literature and discussion there is, as yet, no

consensus on PA. A positive response to this challenge from doctors and their controlling elites could have many beneficial effects for Western healthcare.

Competing interests:

Nil.

Author information:

Philip Bagshaw, Clinical Associate Professor, University of Otago, Christchurch;
Pauline Barnett, Health Sciences Centre, University of Canterbury, Christchurch.

Corresponding author:

Philip Bagshaw, Canterbury Charity Hospital Trust, PO Box 20409, 349–353 Harewood Road, Bishopdale, Christchurch 8054.
philipfbagshaw@gmail.com

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7432>

REFERENCES:

- Earnest MA, Wong SL, Federico SG. Physician advocacy: what is it and how do we do it? *Acad Med*. 2010; 85:63–7.
- Watson J. Should doctors enter the political fray? *Medscape*. 11 April 2017. Accessed at www.medscape.com/viewarticle/878374 on 26 August 2017.
- Pellegrino ED. Medical ethics in an era of bioethics: Resetting the medical profession's compass. *Theor Med Bioeth*. 2012; 33:21–4.
- Huddle TS. Political activism is not mandated by the medical profession. *Am J Bioeth*. 2014; 14:51–2.
- Tilburt JC. Addressing dual agency: Getting specific about the expectations of professionalism. *Am J Bioeth*. 2014; 14:29–36.
- Gruen RL. Evidence-based advocacy: The public roles of health care professionals. *MJA*. 2008; 188:684–5.
- Pellegrino ED. Altruism, self-interest and medical ethics. *JAMA*. 1987; 258:1939–40.
- Komrad MS. A defence of medical paternalism; maximizing patients' autonomy. *J Med Ethics*. 1983; 9:38–44.
- Pearson SD. Caring and cost: The challenge for physician advocacy. *Ann Intern Med*. 2000; 133:148–53.
- Sethi MK, Sathiyakumar V, Mather RC. The evolution of advocacy and orthopaedic surgery. *Clin Orthop Relat Res*. 2013; 471:1873–8.
- Geraghty KE, Wynia MK. Advocacy and community: The social roles of physicians in the last 1000 years. Parts I, II & III. *Medscape General Medicine*. 2000; 2(4).
- Brandt AM, Gardner M. Antagonism and accommodation: Interpreting the relationship between public health and medicine in the United States during the 20th century. *Am J Public Health*. 2000; 90:707–15.
- Ackerknecht EA. *Rudolph Virchow: doctor, statesman, anthropologist*. Madison: University of Wisconsin Press, 1953.
- Mackenbach JP. Politics is nothing but medicine at a larger scale: Reflections on public health's biggest idea. *J Epidemiol Community Health*. 2009; 63:181–4.
- McKinlay JB, Marceau LD. The end of the golden age of doctoring. *Int J Health Serv*. 2002; 32:379–416.
- Starr P. *The social transformation of American medicine*. New York: Basic Books, 1984.
- Freidson E. The Changing Nature of Professional Control. *Ann Rev Sociol*. 1984; 10:1–20.
- Cruess RL, Cruess SR. Expectations and obligations: Professionalism and medicine's social contract

- with society. *Perspect Biol Med.* 2008; 51:579–98.
19. Hiepler M, Dunn B. Irreconcilable differences: Why the doctor-patient relationship is disintegrating in the hands of health maintenance organizations and Wall Street. *Pepperdine Law Review.* 1998; 25:597–616.
 20. Bura V. Health professions and the state. In: Cockerham W, Dingwall R and Quah S. eds. *Wiley Blackwell Encyclopedia of health, illness, behavior and society.* 2014:1054–9.
 21. Forrow L, Sidel VW. Medicine and nuclear war: From Hiroshima to mutually assured destruction to abolition 2000. *JAMA.* 1998; 280:456–61.
 22. Key dates in the history of anti-tobacco campaigning. ASH (Action on Smoking and Health). 21 February 2017. Access at http://www.ash.org.uk/files/documents/ASH_741.pdf on 26 August 2017.
 23. Block JP. A substantial tax on sugar sweetened drinks could help reduce obesity. *BMJ.* 2013; 347:f5947.
 24. Watts N, Adojer WN, Agnolucci P, et al. Health and climate change: Policy responses to protect public health. *Lancet.* 2015; 386:1861–947.
 25. Barnett JR, Barnett P, Kearns RA. Declining professional dominance? Trends in the proletarianisation of primary care in New Zealand. *Soc Sci Med.* 1998; 46:193–207.
 26. Haug MR. Deprofessionalization: An alternative hypothesis for the future. *Sociol Rev Monogr.* 1973; 20(S1):195–211.
 27. Rodwin MA. Patient accountability and quality of care: Lessons from medical consumerism and the Patient Rights, Women's Health and Disability Rights movements. *Am J Law Med.* 1994; 20:147–61.
 28. Light D, Levine S. The changing character of the medical profession: A theoretical overview. *Milbank Q.* 1988; 66(Suppl. 2):10–32.
 29. Freidson E. *Professionalism Reborn: Theory, Prophecy and Policy.* Chicago: University of Chicago Press. 1994.
 30. Annandale E. Proletarianization or restratification of the medical profession? The case of obstetrics. *Int J Health Serv.* 1989; 19:611–34.
 31. Coburn D, Rappolt S, Bourgeault I. Decline vs. retention of medical power through restratification: An examination of the Ontario case. *Sociol Health Illness.* 1997; 19:1–22.
 32. Peek ME, Wilson SC, Bussey-Jones J, et al. A study of national physician organizations' efforts to reduce racial and ethnic health disparities in the United States. *Acad Med.* 2012; 87:694–700.
 33. Waring J. Restratisation, hybridity and professional elites: Questions for power, identity and relational contingency at the points of professional-organisational intersection. *Sociol Compass.* 2014; 8/5:688–704.
 34. Evetts J. A new professionalism? Challenges and opportunities. *Curr Sociol.* 2011; 59:406–22.
 35. Obama BH. Repealing the ACA without a replacement: The risks to American healthcare. 2017. *N Engl J Med.* 2017; 376:297–9.
 36. McIntosh B, West S. Brexit: The consequences and impact of the NHS. *Br J Healthcare Man.* 2017; 23:154–7.
 37. Warmington R, Hum B, Lin D. Healthcare is political: Case example of physician advocacy in response to cuts in refugees' and claimants' healthcare coverage under the Interim Federal Program. *U Ottawa J Med.* 2014; 14:45–47.
 38. American Medical Association. How Physician Advocacy Shaped Health Care. 14 December 2016. Accessed at <http://wire.ama-assn.org/ama-news/how-physician-advocacy-shaped-health-care-2016> on 26 August 2017.
 39. Coney S. Report blames business plan for unnecessary deaths in New Zealand. *Lancet.* 1997; 351:1188.
 40. NZ Herald. Letter from surgeons lambasts hospital managers over patient care. 24 November 2016. Accessed at http://www.nzherald.co.nz/nz/news/article.cfm?c_id=1&objectid=11753565 on 26 August 2017.
 41. September 11, 2001 [editorial]. *N Engl J Med.* 2001; 345:1126.
 42. Horton R. Violence and medicine: the necessary politics of public health. *Lancet.* 2001; 358:1472–3.
 43. Huddle TS. Perspective: Medical professionalism and medical education should not involve commitments to political advocacy. *Acad Med.* 2011; 86:378–83.
 44. Campbell EG, Regan S, Gruen RL, et al. Professionalism in medicine: Results of a national survey of physicians. *Ann Intern Med.* 2007; 147:795–802.
 45. Furler J, Harris E, Harris M, et al. Health inequalities, physician citizens and professional medical associations: An Australian case study. *BMC Med.* 2007; 5:23.
 46. Gruen RL, Pearson SD, Brennan TA. Physician-citizens – public roles and

- professional obligations. *JAMA*. 2004; 291:94–8.
47. Dharamsi S, Ho A, Spadafora SM, Woollard R. The physician as health advocate: Translating the quest for social responsibility into medical education and practice. *Acad Med*. 2011; 86:1108–13.
 48. McKee M, Mackenbach JP, Allebeck P. Should a medical journal ever publish a political paper? *Eur J Pub Health* 2015; 25:1–2.
 49. Dixon-Woods M, Yeung K, Bosk CL. Why is UK medicine no longer a self-regulated profession? The role of scandals involving “bad apple” doctors. *Soc Sci Med*. 2011; 73:1452–9.
 50. Bolsin S, Pal R, Wilmshurst P, Pena M. Whistleblowing and patient safety: the patient’s or the profession’s interests at stake? *J R Soc Med*. 2011; 104:278–82.
 51. Sidhom E. Medical blogging, a new horizon or a dangerous cliff. 17 Sept 2014. Accessed at http://www.frontiersin.org/blog/Medical_blogging_a_new_horizon_or_a_dangerous_cliff/16622 on 26 August 2017.
 52. Royal College of Surgeons of England. Political Update. 12 August 2017. Accessed at <http://updates-rseng.co.uk/4D4N-2MW7-B8117TP971/cr.aspx> on 26 August 2017.
 53. Clem B, Jeff M. Why welfare states persist: The importance of public opinion in democracies. The University of Chicago Press, Chicago 60637. 2007.
 54. Royal College of Physicians. Underfunded, underdoctored, overstretched. Royal College of Physicians, London. 2016. Accessed at www.rcplondon.ac.uk/guidelines-policy/underfunded-underdoctored-overstretched-nhs-2016 on 26 August 2017.

Asbestos—worker exposure, family disease

William Ivan Glass, Helen Clayson

ABSTRACT

Family members, mostly female, can be at risk of asbestos-related disease as a result of the transfer of asbestos from the workplace to the home on the hair, boots and clothes of the worker. It is argued that in these cases the home should be recognised as an extension of the workplace and that the employer has a duty of care to contain and control the asbestos. Given these circumstances, the family member with the disease should be entitled to cover under the Accident Compensation Legislation.

Recent newspaper reports^{1,2} of mesothelioma occurring to two women have highlighted a particularly tragic outcome of the asbestos disease epidemic in New Zealand. An epidemic currently generating six or more cases of mesothelioma, lung cancer, asbestosis and pleural disease each week. An epidemic which has yet to plateau.

The unique feature of both these cases was that their mesothelioma was a consequence of exposure to asbestos in the home following transfer of the carcinogenic fibres from the workplace on the hair and clothes of family members who worked with asbestos.

Such 'secondary' cases usually occur unexpectedly in old age but not infrequently in middle age and predominantly affect women. A latency of 40 or more years after the initial exposure means that not only is the disease unexpected but often—at least initially—inexplicable both to the patient and to the doctor.

Asbestos disease occurring to family members in this manner was first reported in the medical literature in South Africa in 1960,³ in the UK, 1965,⁴ and later in the US,⁵⁻⁸ Italy^{9,10} and Denmark.¹¹

In New Zealand, the first case was notified to the National Asbestos Disease Register in 1994. This occurred to a 43 year-old woman whose father and older brother were both employed for eight years at an asbestos cement manufacturing company from the

time she was seven years old, thus illustrating a latency of 36 years.

As pointed out in the newspaper articles, these types of exposure do not comply with the New Zealand Accident Compensation Corporation law that requires exposure to have resulted from paid employment in New Zealand within the acceptable latency range. The consequence is that there is no entitlement for cover, lump sum payment, weekly compensation or funding for the most effective treatment. This illustrates both employment and gender discrimination affecting women who do unpaid homework.

While the primary focus of this viewpoint is to highlight the circumstances of transfer of a workplace hazard, asbestos, to the home, it does raise other questions such as "where is the workplace?" and "where are its boundaries?"

If workplace hazards are transferred from work to home on the person (hair, clothes and boots) of the worker, so that the home becomes contaminated and family members suffer, does not that make the home a physical extension of the workplace? If that is the case, it places on the employer a duty of care to control and contain workplace hazards within the workplace, as well as ensuring the worker's contaminated work clothes are retained and laundered on site.

Asbestos-related disease is of worldwide concern¹² and was first brought to the attention of the medical profession, industry

and government over 100 years ago.¹³ Its history is one of scientific conflict, industry denial, government inaction and inadequate recognition and care of the victims.

However, the immediate question is one of fairness for family members who contract asbestos-related diseases in the manner described. Is this too much to ask of a 'no fault' compensation system?

Competing interests:

Nil.

Acknowledgements:

To Grace Chen, researching the National Asbestos Disease Register.

Author information:

William Ivan Glass, Centre for Public Health Research, Massey University, Wellington, Principle Advisor—Occupational Medicine, Technical Programmes and Support, WorkSafe, New Zealand Government, Wellington; Helen Clayson, General Practitioner, Masterton Medical, Masterton.

Corresponding author:

William Ivan Glass, Centre for Public Health Research, Massey University, Wallace Street, Wellington.

biloffice@xtra.co.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7433>

REFERENCES:

1. Cann G. Dad's deadly embrace - second-hand asbestos case could lead to more claims, in *The Dominion Post*. 2017, FairFax Media: Wellington.
2. Cann G. Cancer from husband's washing - Years of exposure to toxic dust on clothes, in *The Dominion Post Weekend*. 2017, FairFax Media: Wellington.
3. Wagner JC, Sleggs CA, Marchand P. Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province. *British Journal of Industrial Medicine*. 1960; 17(4):260–271.
4. NewHouse ML, Thompson H. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *Br J Ind Med*. 1965; 22:261–269.
5. Kilburn KH, Lillis R, Anderson HA, et al. Asbestos disease in family contacts of shipyard workers. *Am J Public Health*. 1985; 75(6):615–7.
6. Kilburn KH, Warshaw R, Thornton JC. Asbestos diseases and pulmonary symptoms and signs in shipyard workers and their families in Los Angeles. *Arch Intern Med*. 1986; 146(11):2213–20.
7. Joubert L, Seidman H, Selikoff IJ. Mortality experience of family contacts of asbestos factory workers. *Ann N Y Acad Sci*. 1991; 643:416–8.
8. Anderson HA, Lillis R, Daum SM, et al. Household-contact asbestos neoplastic risk. *Annals of the New York Academy of Sciences*. 1976; 271(1):311–323.
9. Magnani C, Terracini B, Ivaldi C, et al. A cohort study on mortality among wives of workers in the asbestos cement industry in Casale Monferato, Italy. *Br J Ind Med*. 1993; 50(9):779–84.
10. Ferrante D, Bertolotti M, Todesco A, et al. Cancer Mortality and Incidence of Mesothelioma in a Cohort of Wives of Asbestos Workers in Casale Monferato, Italy. *Environmental Health Perspectives*. 2007; 115(10):1401–1405.
11. Langhoff MD, Kragh-Thomsen MB, Stanislaus S, Weinreich UM. Almost half of women with malignant mesothelioma were exposed to asbestos at home through their husbands or sons. *Dan Med J*. 2014; 61(9): A4902.
12. Takahashi K, Landrigan PJ. The Global Health Dimensions of Asbestos and Asbestos-Related Diseases. *Annals of Global Health*. 2016; 82(1):209–213.
13. Anderson AM. Observations published in H.M. Chief Inspector of Factories and Workshops. 1898.

Fanconi anaemia and oral squamous cell carcinoma: management considerations

Thasvir Singh, Kavin Andi

ABSTRACT

Fanconi anaemia (FA) is a rare multi-system genetic disorder where patients are susceptible to the development of oral malignancies. Clinicians involved in their management should be vigilant in detecting lesions early, and an individualised treatment plan should then be formulated. Although surgery forms the mainstay of oncological treatment, adjuvant therapy can be instituted with care. Unfortunately, prognosis is poor, and close long-term follow-up is required. This short report describes pertinent management considerations in relation to a case of oral squamous cell carcinoma.

Fanconi anaemia (FA) is a rare hereditary genetic disorder with an incidence of approximately 1–5 per million births, although it is more common among Ashkenazi Jews and black South Africans.¹ It has an autosomal recessive inheritance pattern, and multiple genes (*FANC*) are involved in its pathogenesis.² FA is characterised by a range of clinical, haematological and endocrinological abnormalities, with a higher rate of malignancy compared to the general population. We present a case of a patient with FA who developed a squamous cell carcinoma (SCC) of the tongue, along with a discussion of pertinent management considerations relevant to the head and neck clinician.

Case

A 39 year-old female was referred in 2014 regarding a non-healing tongue ulcer (Figure 1). The patient had been diagnosed with FA at the age of six and had undergone a bone marrow transplant at the age of 17, and in 2012 she was treated for a pT2 (13mm) invasive ductal carcinoma of the left breast. At the time of presentation to the head and neck clinic, the left sided tongue lesion had been present for approximately 3–4 months and caused pain with eating, but was otherwise asymptomatic. She experienced no weight loss, otalgia or dysphagia, and upon examination no neck masses were palpable. The 1.5cm ulcer was centred on

Figure 1: Clinical photo of left sided tongue squamous cell carcinoma prior to surgical resection.



Figure 2: Clinical photo of bilateral thenar-hypoplasia, a feature often found with FA.



the ventral aspect of the tongue's lateral border with underlying induration and pain on palpation. She was short in stature and had bilateral thenar-hypoplasia (Figure 2), however no other physical features of FA were found on clinical examination. An incisional biopsy of the oral lesion revealed a well differentiated, keratinising SCC arising from dysplastic surface epithelium, with invasion into the underlying stroma.

Staging investigations did not reveal any metastatic spread to the neck or chest (cT1N0M0 SCC), and following multi-disciplinary team discussion she was planned for a left partial glossectomy and selective neck dissection (I–IV). Pre-operative work-up and consultation with her haematologist did not reveal any signs of bone marrow dysfunction, and an anaesthetic team review cleared her for a general anaesthetic procedure. Surgery progressed uneventfully and the patient was discharged from hospital two weeks post-operatively. Her final staging was confirmed as pT1N1M0 with no adverse features seen on histopathological examination. She is currently being followed up on a regular basis, and there are no signs of loco-regional recurrence after three years of follow-up.

Discussion

FA is the result of a genetic defect in a cluster of proteins responsible for DNA repair, with 16 distinct *FANC* genes reported

in the literature.^{1,2} Genetic testing can be complicated due to the number of associated mutated genes, and large deletions, duplications or sequence variations are frequently found.³ Genetic counselling should be carried out for those families affected by, or are carriers of, FA as the implications of these genetic changes are important. Despite many FA patients developing a malignancy, their pathogenesis is not well understood. Kaplan et al suggested that there are two major defects that play a role in the development of malignancies in patients with FA: defective chromosomal stability and immunodeficiencies.⁴ This not only results in FA patients developing cancer at a relatively young age compared to the general population (median age of 31 vs 45 years old), but their risk compounds as they become older.^{5,6,7} The human papilloma virus (HPV) is now a well-known aetiological factor in the pathogenesis of oropharyngeal squamous cell carcinoma, and there is a growing body of evidence to show that FA patients have a higher rate of oral HPV than control subjects,^{8,9} thus further increasing their risk of developing head and neck SCC (HNSCC). Vaccinating all FA individuals against HPV has been suggested in the UK standards of care guidelines.¹⁰

In a literature review of 1,300 patients diagnosed with FA between 1927 and 2001, 9% developed leukaemia (primarily acute myeloid leukaemia), 5% developed solid

tumours and 3% had liver tumours.¹¹ Of the solid tumours, more than 40% occurred in the aerodigestive tract, including SCC of the oral, pharyngeal and oesophageal regions. Kutler et al showed that in 754 patients with FA, 3% developed HNSCC, resulting in an approximate 500 times higher risk compared to the general population.⁶ Of these patients, 68% developed cancer of the oral cavity with the most common subsite being the tongue.

Pancytopenia and bone marrow impairment is a common feature in patients with FA, and initial management is supportive through transfusions, growth factors and hormonal replacement therapy. Bone marrow transplant is often required for those with features of severe bone marrow failure,⁷ however there is an increased risk of developing HNSCC following haematopoietic stem cell transplantation.¹⁰ Congenital defects are seen in 60–75% of FA patients, including short stature, abnormalities of the skin, arms, head, eyes, kidneys and ears, along with developmental disabilities. Furthermore, approximately 75% of FA patients also have endocrinological abnormalities with varying degrees of severity.¹²

Due to this wide and complex range of clinical features, individualised management plans will have to be formulated when a patient requires treatment for their head and neck malignancy. Congenital defects (especially in the upper limb) and short stature may alter anatomy during resective and reconstructive procedures, and additional pre-operative investigations (eg, skeletal imaging prior to osseocutaneous free-flap planning) and multi-disciplinary consultations (eg, endocrine and haematology units) may be required to ensure an appropriate treatment plan is formulated. In particular, bone marrow dysfunction can lead to an elevated risk of haemorrhage and infection with potentially life-threatening consequences.²

Like most oral cavity malignancies, surgical management is the mainstay of primary treatment. In a review of 19 patients with FA and head and neck cancer, the majority (89%) underwent primary surgical resection, with approximately

one-third of these patients successfully undergoing reconstructive procedures.⁶ A low threshold for elective neck dissection should be considered in those with early oral cavity SCC due to an overall higher risk of oncogenesis. Defective cellular processes (such as altered DNA repair mechanisms) may increase the sensitivity and complication rates associated with conventional cytotoxic chemotherapy and radiotherapy treatment regimes. Early treatment-related complications can be more frequent, and more severe, including cytopenias, skin ulceration, infections and mucositis.^{2,6} Alternative treatments, including biological therapeutics, have been recently used in the literature, which may reduce the issues surrounding the use of non-surgical therapies in this patient cohort.²

Prognosis following the treatment of head and neck malignancy in FA is generally poor,⁷ however definitive figures are not possible given the small patient numbers. Reasons are multi-factorial, including more advanced disease at presentation,⁶ a reduced tolerance and effectiveness of radiotherapy and chemotherapy, and the possibility of further tumours development. In the largest published series, 63% of patients developed multiple malignancies with some developing more than two.¹³ As suggested in a review by Schethenbach et al,¹⁴ improved collaboration between international FA units and clinics is now needed to allow the exchange of medical and genetic information. This will help form a unified treatment approach for all FA patients, in particular for those affected by HNSCC.

In conclusion, FA is a rare genetic disorder that has a range of clinical features and potential management difficulties. Patients should have regular screening for HNSCC with a low threshold for biopsying suspicious lesions. The treatment of oral SCC in FA should be conducted in a multi-disciplinary setting to ensure a safe and effective treatment plan is completed. Surgery should be the primary method of management where possible, and adjuvant therapy should be approached cautiously. Close post-operative follow-up is required due to the life-long risk of recurrent disease and multiple malignancies.

Competing interests:

Nil.

Acknowledgements:

The authors would like to acknowledge the contributions of Dr P Henry (Oral and Maxillofacial Surgery Department) and Dr P Wilson (Pathology Department). There were no commercial associations or external financial funding used in the production of this case report.

Author information:

Thasvir Singh, Oral and Maxillofacial Surgery, Waikato District Health Board, Hamilton; Kavin Andi, Department of Maxillofacial Surgery, St George's Hospital, London, United Kingdom.

Corresponding author:

Thasvir Singh, Oral and Maxillofacial Surgery, Waikato District Health Board, Pembroke Street, Hamilton.

thasvir.singh@waikatodhb.health.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7434>

REFERENCES:

1. Joenje H, Patel KJ. The emerging genetic and molecular basis of Fanconi anaemia. *Nat Rev Genet* 2001; 2:446–57.
2. Wong WM, Parvathaneni U, Jewell PD, Martins RG, Futran ND, Laramore GE, Liao JJ. Squamous cell carcinoma of the oral tongue in a patient with Fanconi anemia treated with radiotherapy and concurrent cetuximab: A case report and review of the literature. *Head Neck* 2013; 35:E292-8. DOI 10.1002/hed.23155
3. Lee HJ, Park S, Kang HJ, Jun JK, Lee JA, Lee DS, Park SS, Seong MW. A case report of Fanconi anemia diagnosed by genetic testing followed by prenatal diagnosis. *Ann Lab Med* 2012; 32:380–84.
4. Kaplan MJ, Sabio H, Wanebo HJ, Cantrell RW. Squamous cell carcinoma in the immunosuppressed patient: Fanconi's anemia. *Laryngoscope* 1985; 95:771–75.
5. Rosenberg PS, Socie G, Alter BP, Gluckman E. Risk of head and neck squamous cell cancer and death in patients with Fanconi anemia who did and did not receive transplants. *Blood* 2005; 105:67–73.
6. Kutler DI, Auerbach AD, Satagopan J, Giampietro PF, Batish SD, Huvos AG, Goberdhan A, Shah JP, Singh B. High incidence of head and neck squamous cell carcinoma in patients with Fanconi anemia. *Arch Otolaryngol Head Neck Surg* 2003; 129:106–12.
7. Masserot C, Peffault de Latour R, Rocha V, Leblanc T, Rigolet A, Pascal F, Janin A, Soulier J, Gluckman E, Socié G. Head and neck squamous cell carcinoma in 13 patients With Fanconi anemia after hematopoietic stem cell transplantation. *Cancer* 2008; 113:3315–22.
8. Kutler DI, Wreesmann VB, Goberdhan A, et al. Human Papillomavirus DNA and p53 polymorphisms in squamous cell carcinomas from Fanconi anaemia patients. *J Natl Cancer Inst* 2003; 95:1718–21.
9. Sauter SL, Wells SI, Zhang X, et al. Oral human papillomavirus is common in individuals with Fanconia anaemia. *Cancer Epidemiol Biomarkers Prev* 2015; 24:864–72.
10. Carroll T, Vora A, McMahon J. Standards of Care for Fanconi Anaemia affected Individuals and their Families. UK & Ireland Fanconi Anaemia Clinical Network, 2008. *Clin Oncol* 2010; 22:898.
11. Alter BP. Cancer in Fanconi anemia, 1927–2001. *Cancer* 2003; 97:425–40.
12. D'Andrea AD. Susceptibility pathways in Fanconi's anemia and breast cancer. *N Engl J Med* 2010; 362:1909–19.
13. Kutler DI, Singh B, Satagopan J, Batish SD, Berwick M, Giampietro PF, Hanenberg H, Auerbach AD. A 20-year perspective on the International Fanconi Anemia Registry (IFAR). *Blood* 2003; 101:1249–56.
14. Schethenbach K, Wagenmann M, Freund M, Schipper J, Hanenberg H. Squamous Cell Carcinomas of the Head and Neck in Fanconi Anemia: Risk, Prevention, Therapy, and the Need for Guidelines. *Klin Padiatr* 2012; 224:132–138.

Aggressive prostate cancer incidence in New Zealand— “united we fall, divided we stand”

Venkatesh Vaidyanathan, Nishi Karunasinghe, Vetrivhel Krishnamurthy, Chi Hsiu-Juei Kao, Vijay Naidu, Radha Pallati, Alice Wang, Khanh Tran, Prasanna Kallingappa, Anower Javed, Syed M Shahid, Jonathan Masters, Clare Wall, Ajit Narayanan, Lynnette R Ferguson

ABSTRACT

Prostate cancer is an important health burden to the healthcare system of any country. However, with the current prostate-specific antigen biomarker having low predictive value even for diagnostic purposes, the challenge is still open to tackle this chronic disease. There have been a number of studies which have indicated and encouraged a multi-directional approach to combat this disease. We have been carrying out a multi-directional approach in order to identify certain New Zealand-specific factors which may be drivers for this cancer and its aggressive forms. These will be explained in further detail in this research letter.

Prostate cancer (PCa) is one of the most significant non-skin cancer male health concerns worldwide,¹ with at least one in six PCa patients estimated at being at risk of developing aggressive PCa.² This makes the identification of a strong predictive biomarker and/or treatment of this disease a priority, especially from the New Zealand perspective. The Australia/New Zealand region records the highest rates for age-standardised men with PCa, relative to the population of men worldwide.^{2,3}

Although age, ethnicity and family history are among the most widely accepted risk factors for PCa, nothing concrete has yet been achieved to clinically alter the outcome.⁴ Other basic underlying components that connect these three factors remain lifestyle and nutrition. With progressing age, lifestyle changes; different individuals across various ethnicities enjoy different kinds of lifestyle; and certain families also have very personalised lifestyle factors, such as the amount and kind of meat eaten. Environmental factors play a major role in the expression of genes and the encoded proteins. Hence, work was

started in identifying the most relevant external conditions in New Zealand for their potential effects on the high incidence rate of aggressive PCa.^{1,4-7}

There are certain environmental, nutritional and lifestyle conditions prevalent in New Zealand, such as low levels of selenium in soil,⁸ deficiency of Vitamin D,⁹ high intake of fatty foods¹⁰ and rate of obesity,¹¹ high percentage of tobacco smokers¹¹ and ageing population¹² that may combine in as yet unknown ways to increase the risk of aggressive PCa locally. We have been undertaking a holistic approach to understand the gene by environment interaction(s) and the risk of aggressive PCa in a cohort including New Zealand men of self-declared European ethnicity with different clinically diagnosed grades/stages of PCa, and gender matched healthy controls within a similar age range (Ethics reference NTY05/06/037 by Northern B Ethics Committee, New Zealand, previously, Northern Y Ethics Committee, New Zealand).

Our results have identified a number of single nucleotide polymorphisms (SNPs) statistically significantly associated with a risk of PCa and aggressive PCa. SNPs are

increasingly becoming strong biomarker candidates to identify susceptibility of PCa (among other cancers).⁴ Very interestingly, a number of these genes are related directly and/or indirectly to selenium metabolism, Vitamin D metabolism, obesity and fat metabolism, inflammation and inflammatory pathways, metabolism of tobacco constituents as well as being involved with androgen metabolism, mismatch repair and oncogenesis. PCa is a common but complex disease, involving a number of aspects of genetics such as failure of mismatch repair genes and over-expression of oncogenes, but it will be naïve to forget about the impact of external factors. Current research focus is on the identification of potential and universal biomarkers for aggressive PCa. But it is also well established that we are what we eat, and local external factors such as consumption of red meat, duration of exercise and consumption of dietary supplements will need to be examined. This will aid us in understanding how the progression of PCa can be checked; especially bearing in mind the prevalent health and lifestyle factors in New Zealand.

Although genome-wide association studies are used for the identification of the direct role SNP association plays as risk for aggressive PCa,¹³ and the various environmental conditions mentioned above have also been related to various non-communicable health diseases,^{14–16} our results indicate that SNP interactions with demographic and lifestyle factors could also add to the allelic effect of producing a modified risk of a disease.^{1,5,17,18} Those SNPs that have come up statistically significantly associated with

the risk of aggressive PCa in our studies could be indicating a unique situation for New Zealand men with PCa.⁵ Our belief now is that a uniform multifactor approach will add value towards current clinical practices in improving diagnosis and along with detailed patient history is vital for combatting certain cases of aggressive PCa, which may be influenced by region-specific lifestyle factors as well as of universal genetic factors. In other words, some SNPs important for the progression of PCa may be triggered by local conditions. It is possible that local conditions also play a part for other chronic diseases as well.

The nature of PCa onset is being unraveled with further development of techniques for genomic analysis, with greater access through affordability and accuracy being key drivers of this trend. We propose that the model of patient health should unite the nature and nurture of pathologies in patients equally, and thus the risk of cancers, including PCa, should be region-specific rather than global to take into account local external factors. By identifying such local factors, preventative education programs can also be started to help reduce the risk of PCa as well as encourage early diagnosis of PCa before it becomes aggressive. Such programs may vary from population to population, taking into account lifestyle and nutritional differences. To conclude, we believe that physicians, nutritionists and dieticians, researchers, geneticists and statisticians should be “united” in their approach to tackling PCa, which is to discuss and follow preventative measures on a local, “divided” basis.

Competing interests:

Nil.

Author information:

Venkatesh Vaidyanathan, Teaching Assistant, Department of Molecular Medicine and Pathology, FM & HS, University of Auckland, Auckland;
 Nishi Karunasinghe, Research Fellow, Auckland Cancer Society Research Centre, Auckland;
 Vetrivhel Krishnamurthy, Post-Graduate student, University of Auckland, Auckland;
 Chi Hsiu-Juei Kao, PhD student, Discipline of Nutrition and Dietetics, FM & HS, University of Auckland, Auckland; Vijay Naidu, PhD student, School of Engineering, Computer and Mathematical Sciences, Auckland University of Technology, Auckland;
 Radha Pallati, Laboratory Technician, Auckland Clinical Studies, Auckland;
 Alice Wang, FM & HS, University of Auckland, Auckland; Khanh Tran, PhD student, Department of Molecular Medicine and Pathology, FM & HS, University of Auckland, Auckland; Prasanna Kallingappa, Research Fellow, Vernon Jenson Unit, FM & HS, University of Auckland, Auckland; Anower Javed, Research Fellow, Department of Molecular Medicine and Pathology, FM & HS, University of Auckland, Auckland;
 Syed M Shahid, Postdoctoral Academic Visitor, FM & HS, University of Auckland, Auckland; Jonathan Masters, Urologist, Urology Department, Auckland District Health Board, Auckland; Clare Wall, Head of Department, Discipline of Nutrition and Dietetics, FM & HS, University of Auckland, Auckland; Ajit Narayanan, Head of the Research, School of Engineering, Computer and Mathematical Sciences, Auckland University of Technology, Auckland;
 Lynnette R Ferguson, Emeritus Professor, University of Auckland, Auckland.

Corresponding author:

Dr Venkatesh Vaidyanathan, Teaching Assistant, Department of Molecular Medicine and Pathology, FM & HS, University of Auckland, Auckland 1023.
 v.vaidyanathan@auckland.ac.nz

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7435>

REFERENCES:

- Vaidyanathan V, Naidu V, Kao CH-J, et al. Environmental factors and risk of aggressive prostate cancer among a population of New Zealand men - a genotypic approach. *Mol Biosyst* 2017; 13:681–98.
- Prostate Cancer-Estimated Incidence, Mortality and Prevalence Worldwide in 2012. International Agency for Research on Cancer, 2012. (Accessed 19/09/2017, at <http://globocan.iarc.fr/old/FactSheets/cancers/prostate-new.asp>).
- Ministry of Health. Cancer: New registrations and deaths 2013. Wellington, New Zealand; 2016.
- Vaidyanathan V, Naidu V, Karunasinghe N, et al. Effect of ageing and single nucleotide polymorphisms associated with the risk of aggressive prostate cancer in a New Zealand population. *Mol Biosyst* 2017;7.
- Vaidyanathan V, Naidu V, Karunasinghe N, et al. SNP-SNP interactions as risk factors for aggressive prostate cancer. In: *F1000Research*; 2017.
- Karunasinghe N, Han DY, Zhu S, et al. Effects of supplementation with selenium, as selenized yeast, in a healthy male population from New Zealand. *Nutr Cancer* 2013; 65:355–66.
- Karunasinghe N, Lange K, Yeo Han D, et al. Androgen Pathway Related Gene Variants and Prostate Cancer Association in Auckland Men. *Current Pharmacogenomics and Personalized Medicine* 2013; 11:22–30.
- Hewitt A, Dymond J. Survey of New Zealand soil orders. Ecosystem services in New Zealand: conditions and trends 2013:121–31.
- Ministry of Health. Vitamin D Status of New Zealand Adults; 2012.
- University-of-Otago-and-Ministry-of-Health. A Focus on Nutrition: Key findings of the 2008/09 New Zealand Adult Nutrition

- Survey: Wellington: Ministry of Health. 2011.
11. Ministry-of-Health. Annual Update of Key Results 2014/15: New Zealand Health Survey. Wellington: Ministry of Health; 2015.
 12. Cornwall J, Davey J. Impact of population ageing in New Zealand on the demand for health and disability support services, and workforce implications. Wellington, New Zealand: Ministry of Health; 2004.
 13. Turner AR, Kader AK, Xu J. Utility of Genome-Wide Association Study findings: prostate cancer as a translational research paradigm. *J Intern Med* 2012; 271:344–52.
 14. Bhaskaran K, Douglas I, Forbes H, dos-Santos-Silva I, Leon DA, Smeeth L. Body-mass index and risk of 22 specific cancers: a population-based cohort study of 5.24 million UK adults. *Lancet* 2014; 384:755–65.
 15. Holick MF. Vitamin D deficiency. *N Engl J Med* 2007; 357:266–81.
 16. Moosmann B, Behl C. Selenoproteins, cholesterol-lowering drugs, and the consequences: revisiting of the mevalonate pathway. *Trends Cardiovasc Med* 2004; 14:273–81.
 17. Karunasinghe N, Han DY, Goudie M, et al. Prostate Disease Risk Factors among a New Zealand Cohort. *Journal of Nutrigenetics and Nutrigenomics* 2013; 5:339–51.
 18. Karunasinghe N, Han DY, Zhu S, et al. Serum selenium and single-nucleotide polymorphisms in genes for selenoproteins: relationship to markers of oxidative stress in men from Auckland, New Zealand. *Genes & Nutrition* 2012; 7:179–90.

Tiotropium in early-stage chronic obstructive pulmonary disease

Patients with mild or moderate chronic obstructive pulmonary disease (COPD) rarely receive medications, because they have few symptoms. These researchers speculate that long-term use of tiotropium would improve lung function and ameliorate the decline in lung function in such patients.

Eight hundred and forty-one patients were involved in this randomised placebo-controlled trial. Half were treated with a daily inhaled dose of 18 micrograms of tiotropium and the other half a matched placebo. The primary end point was the between-group difference in the change from baseline to 24 months in the forced expiratory volume in one second (FEV1) before bronchodilator use.

Tiotropium resulted in a higher FEV1 than placebo at 24 months and ameliorated the annual decline in the FEV1 after bronchodilator use in patients who had mild to moderate COPD.

N Engl J Med 2017; 377:923–35

Optimal timing of an invasive strategy in patients with non-ST-elevation acute coronary syndrome

A routine invasive strategy is recommended for patients with non-ST-elevation acute coronary syndromes (NSTEMI-ACS). However, optimal timing of invasive strategy is less clearly defined.

As individual clinical trials were underpowered to detect benefit, this group undertook a meta-analysis of eight relevant trials. Over 5,000 patients with a median follow-up of 180 days were involved. Overall there was no significant mortality reduction in the early invasive group compared with the delayed invasive group. However, a lower mortality rate was associated with the early invasive strategy in those with elevated cardiac biomarkers at baseline and in those with diabetes or who were 75 years of age or older.

An early invasive strategy does not reduce mortality compared with a delayed invasive strategy in all patients with NSTEMI-ACS. However, an early invasive strategy might reduce mortality in high-risk patients.

Lancet 2017; 390:737–46

Risk of acute myocardial infarction with NSAIDs in real world use

What are the risks of acute myocardial infarction associated with use of common non-steroidal anti-inflammatory drugs (NSAIDs) under real life practice circumstances?

That is the question addressed in this meta-analysis. A cohort of over 400,000 individuals was acquired from healthcare databases. The onset of risk and effects of duration of use and daily dose were characterised for celecoxib, diclofenac, ibuprofen, naproxen and rofecoxib.

All NSAIDs were associated with an increased risk of acute myocardial infarction. The odds ratio of risk was 1.24 for celecoxib, 1.48 for ibuprofen, 1.50 for diclofenac, 1.53 for naproxen and 1.58 for rofecoxib. The risk was greatest with higher doses and during the first month of NSAID use without obvious further increase with continued use.

BMJ 2017; 357:j1909

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7436>

Examination of the Heart, and Life Insurance

December 1917

The medical referee of one of the largest life assurance corporations doing business in New Zealand comments as follows on Sir James Mackenzie's book on "Principles of Diagnosis and Treatment in Heart Affections":—"I have read it through twice with great interest. It is most suggestive. I do not see, however, how the principles can be safely acted upon in life assurance. Apparently he would have us ignore physical defects such as valvular lesions unless they are accompanied by impaired 'response to effort.' Now, this weak response to effort can be estimated by a physician who sees a good deal of the case and can follow it through. He also need not in any way doubt a patient's history of his case. But how could a life assurance examiner, who only sees a proponent during his single examination, form any estimate whatever of the

capability of a heart to respond to effort? Moreover, it is the interest of a proponent to belittle any adverse experiences of that sort. It seems to me that we must still be guided by the rough and ready, even if partially incorrect, principle of rejecting or heavily loading cases with valvular lesions or much enlarged hearts. It does not seem to me possible for us to see enough of the case to assess, with safety from risk, except on our present plan. We have all known cases with valvular murmurs who have lived to good ages with no inconvenience; but we do not know what percentage of deaths from other diseases, e.g., pneumonia and typhoid, are accelerated because of old-standing cardiac lesions. Surely a man with such an affection is less capable to resist a severe physical strain caused by illness. His first line of resistance is gone."

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7437>

Proceedings of the Waikato Clinical Campus Biannual Research Seminar

Wednesday 20 September 2017

Mortality from cardiac arrest after cardiac surgery—what can be done?

Mr David J McCormack,
Mr Cheyaanthan Haran,
Mr Paul Conaglen, Mr Nand Kejriwal,
Mr Zaw Lin, Mr Nick Odom,
Mr Grant Parkinson,
Mr Adrian Levine, Dr Tom O'Rourke,
Assoc Prof Adam El-Gamel
Waikato Cardiothoracic Unit.

Background

Internationally, mortality following cardiac arrest after cardiac surgery is high. The Virginia State (USA) registry of 79,582 cardiac operations reported the mortality rate of 49–69% in those suffering cardiac arrest after surgery. Factors such as education, teamwork and communication are crucial in improving outcomes. Recent EACTS (2009) and STS (2016) guidelines address the resuscitative management of such patients. The Australasian Cardiac Surgery Advanced Life Support (CAL S) Course is taught in Sydney, Melbourne and Adelaide. We report the impact of the inaugural New Zealand on resuscitation team confidence.

Objectives

To assess what can be done to reduce mortality from cardiac arrest after cardiac surgery.

Methods

Multidisciplinary staff from seven New Zealand units participated in a one-day CAL S course at Waikato Hospital. Twelve delegates were included. All were ALS trained; none had attended a previous CAL S course. Anonymised self-assessment of confidence

was documented pre- and post-course using a Likert Scale focused on six domains (overall confidence, managing cardiovascular emergencies, managing respiratory emergencies, managing the airway, assisting in re-sternotomy, perform re-sternotomy). Data was analysed with a Wilcoxon signed-rank test.

Findings

Confidence to assist in a re-sternotomy had the greatest increase after the course ($p < 0.01$), followed by confidence to perform a re-sternotomy ($p < 0.01$), managing emergencies involving cardiovascular problems ($p < 0.05$), managing emergencies involving respiratory problems ($p < 0.05$), managing the airway ($p < 0.05$).

The overall confidence with resuscitation after cardiac surgery increased ($p < 0.05$).

Conclusions

Significant improvements in confidence in resuscitation after cardiac surgery are achieved following the CAL S course. Team dynamics are ALSO enhanced with clearly defined roles.

Rates of unsuspected thyroid cancer in multinodular thyroid disease in Aotearoa

Miriam Karalus, Jade Au Tamatea,
Helen M Conaglen,
Goswin Y Meyer-Rochow,
John V Conaglen, Marianne S Elston
Waikato Clinical Campus,
University of Auckland, Hamilton 3240, New Zealand.

Background

The association of concomitant thyroid cancer in multinodular goitre (MNG) has

been reported to be about 4%. Cancer risk in toxic MNG was considered to be lower than for non-toxic MNG and attributed to TSH suppression. However, recent international studies suggest an approximate 18% risk of occult malignancy in both toxic and non-toxic MNG.

Objectives

To ascertain the risk of thyroid cancer New Zealand population undergoing thyroidectomy for MNG.

Methods

Single-centre study of patients undergoing thyroidectomy for multinodular disease 1 December 2006 to 30 November 2016.

Findings

Six hundred and two patients underwent surgery for multinodular disease (448 non-toxic and 154 toxic MNG). Of these, 95/602 (16%) had thyroid cancer. After excluding patients with a preoperative suspicion of cancer, 30/401 (8%) patients with non-toxic MNG and 15/151 (10%) with toxic MNG had unsuspected or occult thyroid cancer ($p = 0.358$). Patients with toxic MNG were less likely to undergo preoperative fine needle aspiration than those with non-toxic MNG (34% vs 52%, respectively $p = 0.0001$). Two-thirds of unsuspected thyroid cancers were incidental micropapillary carcinomas, which were unlikely to alter survival irrespective of therapy.

Conclusion

Malignancy rates in MNG are higher than historically reported, although most unsuspected cancers are unlikely to alter patient outcome even if diagnosis is delayed.

Is the medium the message? Format matters in medicines information

Megan Veail, Andre Mutavdzic, Rajan Ragupathy, Jan Goddard
Pharmacy Services, Waikato District Health Board.

Background

Providing tailored information about prescribed medicines to patients is an important part of clinical pharmacy services. A good understanding of the purpose of the medicines and possible side effects may contribute to better compliance as well as reducing risks from inappropriate use of medicines. However, the patients' preferences and the form in which patients prefer to receive this information has only sparsely been examined.¹ This study aimed to examine the patients' preferences and whether these preferences were affected by demographic factors (self-identified ethnicity, education, age or gender).

Objectives

To ascertain the patients' preferences and the form in which patients prefer to receive prescribed medicine information.

Methods

A questionnaire requesting demographic data and patient preferences was developed. The choices offered were personalised medication cards, pamphlets, smart-phone app, face-to-face conversation, e-mail and video. Patients were asked to rank their preferences. Choices were then ordered by the percentage of patients who ranked each among their top three preferences. The questionnaire was tested and refined after a pilot study. Patients in Waikato, Thames and Tokoroa hospitals were selected on a random basis. Patients under 18 years old were excluded from the study. The questionnaire was distributed to the selected patients by clinical pharmacists of the wards involved. Patients were left to fill out the questions on their own, and the questionnaires were collected by the pharmacists on the same day.

Findings

Overall the preferred media were face-to-face conversations (81%, 95% CI 71–88%) and personalised medication cards (78%, 95% CI 69–86%), followed by pamphlets (58%, 95% CI 48–68%). Smartphone apps, e-mail and videos were not popular choices in the overall population. Patients under 50 years old showed an increased preference for smartphone apps (67%, 95% CI 41–85%), compared to patients 50 years and older (17%, 95% CI 11–28%). This preference was also shown by Māori patients, but could reflect the younger mean age of hospitalised Māori patients (52 years versus 68 years for non-Māori). Preferences were not influenced by gender or education.

Conclusions

Age and ethnicity may affect the preferred medium for receiving information about medicines, but the patient's personal preference should be considered when delivering medicines information.

References

1. Street C. Medicines information: What Matters Most to Patients. Paper presented at the New Zealand Hospital Pharmacists' Association Conference; Napier (New Zealand); 2015 Aug 28–30.

Findings from the Midland Region Lung Cancer registry

Ross Lawrenson,^{1,2} Chunhuan Lao,¹ Leonie Brown,¹ Janice Wong,² Karen Middleton²

¹University of Waikato;

²Waikato District Health Board.

Background

The completeness and accuracy of the New Zealand Cancer Registry (NZCR) are vital for cancer control in New Zealand. This study aims to report the characteristics of newly diagnosed lung cancer cases and compare the data accuracy of registrations in the NZCR with the Midland Region Lung Cancer register (MLCR).

Objectives

To compare the data accuracy of registrations in the NZCR Midland Region Lung Cancer register.

Methods

Lung cancer (ICD code: C33, C34) and mesothelioma cases (ICD code: C45) diagnosed in 2011–2015 were extracted from both the NZCR and the MLCR. The two datasets were linked by the National Health Index (NHI) number. The cancer extent/stage, date of diagnosis, gender, ethnicity, DHB, date of birth, date of death and date of diagnosis were compared for cancer cases identified in both datasets. For cancer cases diagnosed in the Waikato DHB and identified in the NZCR only, clinical records of these patients were examined to verify the lung cancer or mesothelioma diagnosis.

Findings

In total, 2,126 lung cancer registrations and 81 mesothelioma registrations were identified in the NZCR, including four duplicate lung cancer registrations. Of the 1,570 lung cancer registrations and 59 mesothelioma registrations recorded in the MLCR, 1,483 (94.5%) lung cancer cases and 54 (91.5%) mesothelioma were identified in the NZCR. Of the cancer cases identified in both datasets, 51.3% of the cancer extent in the NZCR was correct for lung cancer registrations and only 17.0% for mesothelioma registrations. The consistency of the two datasets was 99.0% for gender, 96.2% for ethnicity, 98.4% for DHB, 99.7% for date of birth, 94.4% for date of death and 89.9% for date of cancer diagnosis (difference ≤30 days). There are 639 lung cancer registrations and 27 mesothelioma registrations not identified in the MLCR, including 190 lung cancer registrations and 10 mesothelioma registrations in the Waikato DHB. After examining the clinical records of the 200 Waikato patients, 110 (57.9%) were confirmed to be diagnosed with lung cancer or mesothelioma in 2011–2015, 10 (5.3%) were diagnosed with lung cancer or mesothelioma before 2011 or after 2015, 34 (17.9%) did not have lung cancer nor mesothelioma, and 36 (18.9%) could not be verified.

Conclusion

The MLCR provides excellent clinical data on newly diagnosed lung cancer cases. However, there is some under-reporting compared with the NZCR. Combining the two sources of data gives a more complete picture of the incidence of lung cancer in our region.

Improving coronary graft patency with postoperative aspirin and clopidogrel versus aspirin and ticagrelor

Pavan Thanneru,¹ Melissa Kirk,¹ Sean Galvin,² Adam El-Gamell, Scott Harding,² Madhav Menon,¹ Gerard Devlin¹

¹Waikato Hospital, Hamilton; ²Wellington Hospital, Wellington.

Background

Dual anti-platelet therapy (DAPT) reduces events post-cor-

onary artery bypass graft (CABG).¹ In PLATO CABG study aspirin and ticagrelor (AT) was superior to aspirin and clopidogrel (AC).² The mechanism remains unclear. We hypothesise this may relate to superior graft patency with AT.

Objectives

The primary objective is to compare the effect of dual antiplatelet therapy on the incidence of graft occlusion at 12 months. As assessed by multi-slice computed tomography coronary angiography (CTCA) in patients randomised to AT or AC.

Methods

Randomised, open label design of patients undergoing isolated CABG following an acute coronary syndrome (ACS).

Findings

As of 1 June 2017, a total of 85 patients have been randomised.

(43 AT v 42 AC) with 83% male and mean age 63 years. Demographics were similar for both groups. Follow-up results of CTCA were available in 58 patients at 12 months.

Conclusion

Preliminary results of IMPACT study show that DAPT is safe in post-ACS patients undergoing CABG. No difference is apparent in clinical outcomes or graft patency at 12 months. Significant ticagrelor discontinuation due to dyspnoea (P<0.01).

References

1. Gao G, et al. Aspirin plus clopidogrel therapy increases early venous graft patency after coronary artery bypass surgery a single-center, randomized, controlled trial, *J Am Coll Cardiol*, 2010; 56:1639–43.
2. Held C, Asenblad N, et al. PLATO-CABG. *J Am Coll Cardiol* 2011; 57:672–84.

CTCA Outcomes at 12 months intention to treat	Aspirin and ticagrelor (n=28)	Aspirin and clopidogrel (n=30)	P
Grafts assessed	94	93	
Arterial grafts assessed	23	33	
Any grafts occluded	9 (9.6%)	14 (15.1)	0.36
Arterial grafts occluded	3 (13%)	3 (9%)	0.98
Vein grafts occluded	6 (8.5%)	11 (18%)	0.16

Clinical outcomes	Aspirin and ticagrelor (n=32)	Aspirin and clopidogrel (n=31)	P
Death	0	1 (3.2%)	NS
Revascularisation	2 (6.3%)	2 (6.4%)	NS
Symptomatic graft failure	3 (9.4%)	3 (9.7%)	NS
CABG related bleeding events	1 (3.1%)	0	NS
Non-CABG related bleeding events	0	1 (3.1%)	NS

Reason for study drug discontinuation	Aspirin and ticagrelor (n=32)	Aspirin and clopidogrel (n=31)	P
Need for anticoagulation	2 (6.4%)	1 (3.2%)	NS
Side effect—dyspnoea	8 (25%)	0	<0.01
Clinical event	0	1 (3.2%)	NS

Clinical outcomes of overweight or obese patients with stage III colon cancer treated with adjuvant oxaliplatin-based chemotherapy in the Waikato Region

Jayden Wong, Alvin Tan, Sagun Banjade, Michael Jameson
Waikato District Health Board, Hamilton, New Zealand.

Background

According to Ministry of Health statistics, as of 2015/16 data, 35% of New Zealand adults were overweight and 32% were obese. A large retrospective study by Dignam et al demonstrated poorer disease-free and overall survival in obese patients with Duke B and C colon cancer.¹ A meta-analysis by Sinicrope et al concluded that obesity is an independent prognostic variable in colon cancer patients.² We present our local data in the Waikato region, comparing the clinical outcomes of patients with normal weight (NW) and patients who were overweight or obese (OO).

Objectives

To assess the clinical outcomes of overweight or obese patients with Stage III colon cancer treated with adjuvant oxaliplatin-based chemotherapy in the Waikato Region.

Methods

This was a retrospective cohort study of all patients with completely resected Stage III colon cancer who received adjuvant oxaliplatin-based chemotherapy in the Waikato region from 1 January 2008 to 31 December 2013. Patient baseline characteristics, treatment records and cancer-specific outcomes [three-year disease-free survival (3yr DFS) and three-year overall survival (3yr OS)] were recorded. Patient body mass index (BMI) was documented prior to chemotherapy commencement.

Findings

Total of 86 patients with Stage III colon cancer were treated with oxaliplatin-based chemotherapy over this six-year period; 68 patients received FOLFOX6, 16 patients received FLOX and two patients received CAPOX. Among these patients, three patients were underweight (BMI <18.5kg/m²), 25 were of normal weight (BMI 18.5–24.9 kg/m²), 42 were overweight (BMI 25–29.9 kg/m²) and 16 were obese (BMI ≥30kg/m²). Baseline characteristics were fairly balanced, apart from a higher proportion of males (60% vs 28%), Māori descent (14% vs 4%) and left-sided primary site (57% vs 32%) in the OO group, compared to the NW group. 3yr DFS was worse in the OO group than the NW group (70.9% vs 77.3%, p=0.57). 3yr OS was similar in both groups (87.9% vs 88.0%). Chemotherapy dosing was not capped based on body surface area at our institution. A greater amount of oxaliplatin was received in the OO group as a mean percentage of their planned total oxaliplatin dose when compared to the NW group (81% vs 68%), with a lower rate of early cessation of oxaliplatin (41% vs 60%). Rate of any-grade peripheral neuropathy was higher in the OO group (95% vs 76%).

Conclusion

Among patients with Stage III colon cancer, overweight or obese patients demonstrated a poorer three-year disease-free survival when compared to normal weight patients, despite receiving a greater amount of oxaliplatin-based chemotherapy. This difference was not statistically significant; but is in keeping with contemporary literature.

References

1. Dignam JJ, Polite NB, et al. Body Mass Index and Outcomes in Patients Who Receive Adjuvant Chemotherapy for Colon Cancer. *Journal of the National Cancer Institute*, November 2006; 98(22):1647–54.
2. Sinicrope FA, Foster NR, et al. Obesity is an Independent Prognostic Variable in Colon Cancer Survivors. *Clinical Cancer Research*, March 2010; 16(6):1884–93.

The influence of comorbidity on guideline-concordant surgical treatment for primary breast cancer

Melissa J Edwards,¹ Ian D Campbell,¹ Ross A Lawrenson²

¹Department of Surgery, Waikato Clinical School, University of Auckland; ²National Institute of Demographic and Economic Analysis, University of Waikato.

Background

Patients with breast cancer and concomitant comorbidity have poorer disease prognosis, which may, in part, be related to a reduction in the receipt of guideline-concordant curative treatment.¹ Excision of the breast tumour and surgical staging/treatment of the axilla are key components of treatment for non-metastatic breast cancer. In this study, we sought to determine the impact of comorbidity on the receipt, quality and timeliness of surgical treatment for primary breast cancer.

Objectives

To assess the influence of comorbidity on guideline-concordant surgical treatment for primary breast cancer.

Methods

Incident cases of unilateral, stage I–III breast cancer, diagnosed between June 2000 and June 2015 were identified from the prospectively collected Auckland and Waikato Breast Cancer Registers. Comorbidity information was obtained via National Health Index number linkage with administrative hospital discharge data (the National Minimum Dataset), limited to five years preceding the date of breast cancer diagnosis. Comorbidity severity was measured by C3 index score.² Receipt of surgical treatment, as well as surgical quality and timeliness indicators, were examined with respect to guideline-concordance by C3 score and individual important comorbidities. Guideline-concordance was assigned in relation to the Standards of Service Provision for Breast Cancer Patients in New Zealand³

and St Gallen International Expert Consensus Statements from relevant years. Multi-variable logistic regression analyses were performed, adjusted for patient demographic and healthcare access factors, as well as tumour stage. Age and C3 score were modelled using cubic splines due to non-linear relationships.

Findings

Application of the inclusion criteria resulted in the identification of 12,652 patients, with 2,609 (20.6%) possessing at least one major comorbidity. Increasing levels of comorbidity severity were associated with reducing likelihood of receiving surgical excision of the primary breast tumour and operative staging/treatment of the axilla. For patients who received surgical treatment, comorbidity had no impact upon the receipt of definitive quality surgery, defined as mastectomy or breast conserving surgery with a 2mm resection margin negative for invasive/in situ disease. Similarly, comorbidity had no association with receipt of appropriate surgical axillary management. High levels of comorbidity were associated with a reduction in the receipt of timely primary breast surgery (within 31 days of diagnosis).

Conclusion

Compared with their non-comorbid counterparts, comorbid patients with primary breast cancer receive less guideline-concordant cancer surgery. If surgery is performed, it is of equivalent quality but received at greater delay. The impact of inferior surgical treatment on survival in the context of comorbidity is yet to be determined.

Grant support

This work was supported by the New Zealand Health Research Council (Clinical Research Training Scholarship; 17/016), the Cancer Society of New Zealand (Training Scholarship in Cancer Research), the University of Auckland (Research Fellowship in Surgery) and the Waikato Breast Cancer Research Trust.

References

1. Hong C-C, Ambrosome C, Goodwin P. Comorbidities and their management: potential impact on breast cancer outcomes. In: Ganz P, editor. Improving outcomes for breast cancer survivors: perspectives on research challenges and opportunities. Cham: Springer International Publishing AG; 2015. p. 155–75.
2. Sarfati D, Gurney J, Stanley J, Salmond C, Crampton P, Dennett E, et al. Cancer-specific administrative data-based comorbidity indices provided valid alternative to Charlson and National Cancer Institute indices. *J Clin Epidemiol.* 2014; 67(5):586–95.
3. National Breast Cancer Tumour Standards Working Group. Standards of service provision for breast cancer patients in New Zealand - provisional. Wellington (NZ): Ministry of Health; 2013.

Post-operative cardiothoracic x-ray protocols deliver low clinical yield and results that are not cost effective

Damian Gimpel, EJ O'Malley, Paul Conaglen, Zaw Lin, Nand Kejriwal, Nick Odom, David McCormack, A Prof Adam El-Gamel
The Waikato Hospital, Hamilton.

Background

New literature suggests that routine post-operative x-rays are no longer necessary and should be determined by clinical assessment.

Objectives

To assess if post-operative cardiothoracic x-ray protocols deliver low clinical yield and results that are not cost effective.

Method

A retrospective analysis of the quantity, indications, new radiological findings and medical intervention post x-ray in cardiothoracic postoperative patients. Positive findings were determined from radiological reports and patient notes utilised for management post x-ray.

Findings

Patient cohort n=49 consisted of average age of 61.7±8.41 and

an average number of chest x-rays 4.46±2.58. M:F ratio = 5:1. Total number of x-rays performed was n=219 with those undertaken days 0–2 days postoperatively n=169 (60%). Patients requiring change in management post positive finding was n=16 (17%).

The most common indication for imaging was positioning of lines, tubes and drains n=95 (43%) followed by screening for pneumothorax post drain removal in n=55 (20%). Of those 55, chest tube reinsertion occurred in n=6 (11%). New findings found in n=121 images (55%). Most common new finding was postoperative atelectasis, n=63 (52%) followed by pleural effusions, n=40 (33%) of which n=25 (63%) were graded small.

Conclusion

A small number of post-operative chest x-rays had meaningful positive findings and intervention. Each chest x-ray costs \$106, potentially saving \$69,960 per year by abolishing routine imaging post chest drain removal. Positive findings demonstrated a diagnosis that can be ascertained clinically rather than requiring imaging. A collective effort between cardiothoracic teams and those responsible for postoperative care should aim to reduce unnecessary imaging, decreasing exposure, decreasing money expenditure and improving clinical astuteness.

Microsurgical dexterity tuition of students and house surgeons: a necessary and worthwhile investment

Cheyaanthan Haran,¹ Nicholas Brunger,² Oliver Pumphrey,² Kelsey Simpson,² Jacque Roberts,² Nand Kejriwal,² Grant Parkinson,² David J McCormack,² Adam El-Gamel²

¹School of Medicine, University of Auckland; ²Cardiothoracic Surgical Unit, Waikato Hospital, Hamilton.

Background

The undergraduate curriculum does not include surgical dexterity teaching.

Trainees may not attempt microsurgical dexterity tasks until their mid-late 20's. Waikato Cardiothoracic Surgery Unit introduced a dry-labs course aiming to equip aspiring surgeons with skills to practice microsurgical dexterity.

Objectives

To assess the impact of early surgical skills teaching on a junior cohort of aspiring trainees.

Method

Invited medical students and house surgeons at Waikato Hospital to participate in a four-hour microsurgical dexterity course. The course was created and delivered by a multidisciplinary cardiothoracic faculty. Self-assessment of skill was documented pre- and post-course using a Likert Scale focused on five domains (knot tying, bi-manual microsurgical dexterity, suturing fluency, needle passing skills and tissue handling skills). Quality control aspects of the course were assessed (faculty approachability, course delivery, facilities and catering).

Findings

Twenty questionnaires were returned (nine medical students, 11 house surgeons). Forty percent (8/20) of respondents never practiced their surgical skills in theatre. Knot tying had the greatest increase in self-assessment ($p < 0.001$), followed by tissue handling skills ($p < 0.001$), bi-manual microsurgical dexterity ($p < 0.001$), suturing fluency ($p < 0.005$) and needle passing skills ($p < 0.01$). All quality control aspects of the course rated 4.69–4.75.

Conclusion

Dramatic improvements in self-assessed microsurgical dexterity and self-confidence can be achieved. Further work is required to objectively measure learning and performance curves. It is crucial that students are exposed to a broad surgical skillset early in their career. We propose early training courses as a mechanism to enhance motivation and surgical ability in the future generation.

Association between performance status and tumour response to immunotherapy in patients with advanced melanoma: a single regional experience

Joanne Deacon, Osama SM Salih
Medical Oncology Department,
Waikato Hospital, New Zealand.

Background

On 1 July 2017, PHARMAC amended the funding criteria for PD-L1 inhibitors, pembrolizumab and nivolumab, for patients with advanced melanoma by adding restrictions on ECOG performance status of 0–2. There is no consensus around offering immunotherapy to patients with poor performance status and most of the published trials only include patients with ECOG performance status of 0–1.

Objectives

This study examines the association between performance status and tumour response to immunotherapy in patients with advanced melanoma.

Methods

A study was performed of patients with advanced melanoma treated with immunotherapy at Waikato Oncology Service between 1 July 2016 and 30 June 2017. The electronic chemotherapy prescribing system was used to identify all patients treated during this period. Records were searched to extract patient demographics and treatment-related factors such as ECOG performance, site of metastases, number of doses received, adverse events and radiological response.

Result

Forty-four patients were commenced on, or received treatment prior to 1 July 2017. Thirty-eight patients received pembrolizumab, while only six patients had nivolumab. The median age at the time of immunotherapy commencing was 67.5 years. Approximately 2/3 of patients had more than two medical comorbidities prior to commencing immunotherapy. 18/44 patients had multiple

liver metastases, and five had brain metastases. The majority of patients had ECOG performance status 0–1; six patients had ECOG performance status of 2 and none had performance status >2.

Out of 35 patients who have completed their first radiological tumour assessment, 23 patients had tumour response or stable disease compared to 12 patients with disease progression. Of the 23 patients with tumour response, 22 had ECOG Ps of 0–1, and one had ECOG Ps = 2. Four patients died from complications of metastatic disease shortly after commencing treatment; two of these had performance status of 2.

Conclusion

This study has shown that all patients treated with immunotherapy at Waikato Hospital since funding was first approved had a performance status between 0–2. It is unlikely that the new changes to funding criteria will significantly impact upon clinical practice. The study also showed only a small proportion of the patients had ECOG performance of 2 (6/44), however seemed to have worse outcomes.

Development of a qPCR method to measure mitochondrial and genomic DNA damage with application to chemotherapy-induced DNA damage and cryopreserved cells

Stephen Evans,¹ Michael Jameson,²
Ray Cursons,¹ Linda Peters,¹
Steve Bird,¹ Gregory Jacobson¹

¹Biomedical Research Unit, School of Science, University of Waikato Private Bag 3105, Hamilton 3240, Waikato, New Zealand; ²Department of Oncology, Regional Cancer Centre, Waikato Hospital, Hamilton West 3204, New Zealand.

Background

DNA damage quantitation assays such as the comet assay have focused on the measurement of total nuclear damage per cell. The adoption

of PCR-based techniques to quantify DNA damage has enabled sequence- and organ-elle-specific assessment of DNA lesions.

Objectives

We look at an adaptation of a real-time qPCR technique, to assess DNA damage in nuclear and mitochondrial targets relative to control.

Methods

Novel aspects of this assay include: application of the assay to the Rotor-Gene platform with optimised DNA polymerase/fluorophore/primer set combination in a touchdown PCR protocol. Assay validation was performed using ultraviolet C radiation in A549 and THP1 cancer cell lines. A comparison was made to the comet assay applied to peripheral blood mononuclear cells and an estimation of the effects of cryopreservation on ultraviolet C induced DNA damage was carried out. Finally, dose responses for DNA damage were measured in peripheral blood mononuclear cells following exposure to the cytotoxic agents bleomycin and cisplatin.

Findings

We show reproducible experimental outputs across the tested conditions and concordance with published findings with respect to mitochondrial and nuclear genotoxic susceptibilities.

Conclusions

The application of this DNA damage assay to a wide range of clinical and laboratory-derived samples is both feasible and resource-efficient.

Kaumātua mana motuhake: kaumātua managing life-transitions through tuakana-teina/peer-education

Brendan Hokowhitu, John Oetzel, Rangimahora Reddy, Linda Smith, Mary Simpson, Sophie Nock, Hineiti Greensill, Michael Cameron, Pare Meha, Kirsten Johnston
University of Waikato.

Background

People face significant transition points as they age, such as loss of independent living, loss of a spouse and changing health conditions. Successfully navigating these transitions depends on being able to manage emotional and socio-economic factors, as well as service systems, while often being reliant on family or whānau. Historically however, kaumātua have faced a dominant society that has failed to realise their full potential as they age. Yet, for Māori, kaumātua are “carriers of culture, anchors for families, models for lifestyle, bridges to the future, guardians of heritage and role models for younger generations.” Kaumātua mana motuhake is invested in upholding kaumātua tino rangatiratanga (independence and autonomy) via high-quality Māori research that will lead to better life outcomes for kaumātua and their whānau.

Objectives

This seeks to address the mana motuhake of kaumātua (older Māori aged 55 or older), through a ‘tuakana-teina’ peer-educator model where kaumātua work with other kaumātua in relation to signif-

icant life-transitions. The project investigates the health outcomes of a ‘tuakana-teina’ peer-educator model in relation to wellness, social connectedness, life enhancement, independence and significant life-transitions.

Methods

The research comprises two stages: training of kaumātua who will then serve as tuakana (peer educators) for other kaumātua (teina/peers). The research design is a pre- and post-test, clustered randomised staggered design with Tuatahi (intervention) and Tuarua (control) groups. Tuatahi participate in the training programme initially, while Tuarua participate in subsequent training. The capacity of tuakana is assessed at three stages: pre-test, post-intervention for the Tuatahi group and post-intervention of the Tuarua group. After training, each tuakana will talk with each teina at least three times to address relevant life-transitions of their teina. Teina will also complete three evaluations at the same stages as the tuakana. The research design enables a rigorous comparison of the training while ensuring that all teina receive the intervention.

Findings

The outcome of the research is a manualised intervention bringing a strength-based, holistic and cultural approach to meet social and health needs of kaumātua and their whānau.

Conclusion

We engage stakeholders throughout the research process with the aim of scaling up the intervention, provided it demonstrates efficacy and cost-effectiveness.

URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1466-1-december-2017/7438>