Access to joint replacement: have we got it right?

Rebalancing health service use for older people: simulating policy-relevant scenarios under demographic ageing

Parental smoking during pregnancy: findings from the Growing Up in New Zealand cohort

Publication rates and characteristics of undergraduate medical theses in New Zealand

How effective is our current Orthopaedic Prioritisation Tool for scoring patients for arthroplasty surgery?

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 2016

New Zealand subscription rates		Overseas subscription rates	
Individuals*	$298	Individual	$415
Institutions	$517	Institutions	$557
Individual article	$25	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

CONTENTS

Equity of publicly-funded hip and knee joint replacement surgery in New Zealand: results from a national observational study

Helen Harcombe, Gabrielle Davie, Sarah Derrett, Haxby Abbott, David Gwynne-Jones

The provision of publicly-funded hip and knee total joint replacement (TJR) procedures varies between District Health Boards (DHBs) and national rates have not increased since 2007, new University of Otago research has found. The Dunedin School of Medicine study, which appears in the latest edition of the New Zealand Medical Journal, examined rates of elective TJR procedures between 2006 and 2013. Study principal investigator Dr Helen Harcombe says that geographically-based inequities emerged from the research. "Even taking into account age and ethnicity, rates of TJR procedures varied between DHBs, with DHBs covering larger populations tending to have lower rates than smaller ones" Dr Harcombe says. The study also showed that while there has been an increase in the number of TJR procedures carried out in the public system between 2007 and 2013, rates are barely keeping pace with a growing population. The number of publicly-funded TJR procedures increased by six percent over this period, but New Zealand's growing population meant the surgery rate per head of population (aged over 20 years) actually decreased by 0.6 percent.

Access to orthopaedic spinal specialists in the Canterbury public health system: quantifying the unmet need

Tom Inglis, Rowan Schouten, Kristian Dalzell, Jeremy Evison, Grahame Inglis

This study quantifies the unmet need for both Spinal Orthopaedic Specialist assessment and if warranted, surgical management of elective spine conditions within the Canterbury public health system. It highlights the degree of rationing within the public health system and its failure to adequately provide for the Canterbury Public.

Rebalancing health service use for older people: simulating policy-relevant scenarios under demographic ageing

Roy Lay-Yee, Janet Pearson, Peter Davis, Martin von Randow, Ngaire Kerse, Laurie Brown

The ageing of society has major implications for providing health services. We use a simulation model of a representative sample of older New Zealand people to study their use of health services. Our model shows that, in the near future, there will be a moderate increase in long-term illness and health service use. When we simulate a reduction in long-term illness, the effect is a moderate reduction in overall health service use. When we simulate an increase in people visiting the practice nurse at least once a year, the effect is a substantial reduction in use of other health services particularly public hospital admissions.

Epidemiology of intussusception in New Zealand pre-rotavirus vaccination

Bronwyn Rosie, Stuart R Dalziel, Elizabeth Wilson, Emma J Best

Intussusception is a rare form of gut obstruction seen in infants typically aged between six to nine months. It rarely can occur in the first week after a baby receives his/her first or second rotavirus vaccination. Since 2014 rotavirus vaccination has been given in New Zealand to all babies at age six weeks, three months and five months—this is expected to prevent thousands of hospitalisations with rotavirus diarrhoea. We set out to describe intussusception rates in New Zealand babies to compare with other parts of the world, and understanding background rates of intussusception is an important way to monitor rotavirus vaccination safety.

Publication rates and characteristics of undergraduate medical theses in New Zealand

Ibrahim Saleh Al-Busaidi, Yassar Alamri

In New Zealand, the fate and publication rates of theses produced by medical students is unknown. Adding to the existing literature on New Zealand medical student research and publishing, this study sheds light on their contribution to international scientific literature. During the period from January 1995 to December 2014, almost one-third of BMedSc(Hons) theses resulted in a publication in a peer-reviewed journal. Although higher than reported figures from previous studies, publication rates of BMedSc(Hons) theses remain lower than expected. To improve our understanding of medical student publishing in New Zealand, formal examination of the factors hindering medical students from publishing their theses is imperative.

How effective is our current Orthopaedic Prioritisation Tool for scoring patients for arthroplasty surgery?

Neal Singleton, Lewis Agius, Sudhindra Rao

The aim of this study was to compare those patients being accepted onto the waiting list for total hip or knee replacement in Hawke's Bay with those being declined surgery using the Oxford score which is a validated questionnaire for assessing patient function. Patients are currently prioritised for surgery using a non-validated tool which scores patients according to their symptoms and likely benefit from surgery. We found that there was no difference between those patients being accepted for surgery and those being declined surgery. In other words, patients were equally disabled. Patients being seen in Hawke's Bay Hospital for consideration of arthroplasty surgery are severely disabled and yet nearly half are declined surgery. This paper has highlighted the issue of unmet need for arthroplasty surgery which is becoming an increasing issue with New Zealand's ageing population.

Parental smoking during pregnancy: findings from the Growing Up in New Zealand cohort

Gayl Humphrey, Fiona Rossen, Natalie Walker, Chris Bullen

We used the Growing Up in New Zealand cohort study, which follows a group of people over a number of years, to explore smoking behaviour in cycle one of the study which was when all the women in the study were pregnant. This paper looked at factors that may contribute to women who continued to smoke during pregnancy and also the exposure to second-hand smoke. We used analyses to show the importance of these factors in reducing or stopping smoking as well as what factors influenced continued smoking.

Low FODMAP diet efficacy in IBS patients—what is the evidence and what else do we need to know?

Tim Kortlever, Clarice Hebblethwaite, Julie Leeper, Leigh O'Brien, Chris Mulder, Richard B Gearry

Irritable Bowel Syndrome (IBS) is a common gastrointestinal disorder characterised by intermittent abdominal pain with altered bowel habit. Low FODMAP diet has been shown to reduce gastrointestinal symptoms in people with IBS. Low FODMAP diet should be taught by an experienced dietitian.

Access to joint replacement: have we got it right?

Gary Hooper

Much has been talked about the ageing population and its impact on current and future health delivery. Within New Zealand we can expect a similar pattern to other developed countries, with a rapidly increasing aged population who have improved health and functional requirements compared to previous generations. The 65+ age group is likely to make up over one quarter of our population in the late 2030s.[1] Musculoskeletal problems comprise well over 50% of chronic conditions affecting those over 50 years of age[2] and are the second leading cause for disability worldwide.[3] Although governments have been alerted to this for some time with such initiatives as the New Zealand joint initiative (2006), few changes have occurred to create strategies to deal with this burden of disease. Osteoarthritis is one of the most common age-dependent diseases, which has resulted in the recent and predicted marked increase in requirement for joint replacement as a remedy for the debilitating pain associated with this condition.[4] Harcombe et al in *"Equity of publically-funded hip and knee replacement surgery in New Zealand: Results from a national observational study"* criticise the current funding strategies, highlighting significant differences between District Health Boards (DHBs) in New Zealand and the fact that there has been no real increase in provision of these procedures between 2006 and 2013 within public hospitals. They conclude that females, patients over 75 years, Māori, poorer socioeconomic groups and smaller DHBs were all associated with higher rates of publically funded surgery, which may indicate that the most vulnerable groups are being targeted, however, as pointed out by the authors, this "fails to meet the Ministry's key objective of equity across the country".

The introduction of a four-month waiting list, although well intentioned, has created further problems with patients reaching the surgical threshold but being dismissed due to inability to perform the surgical procedure in the mandated time-frame. One of the advantages of having a longer waiting list has been the ability of patients to get a surgical assessment and confirmation of diagnosis as well as surety that surgery will help. Currently patients with clinical need are being denied access to Waiting lists. Singleton et al in *"How effective is our current Orthopaedic Prioritisation Tool for scoring patients for arthroplasty surgery?"* have confirmed that patients not accepted on to waiting lists have similar functional needs to those that have been accepted. This suggests that severely impaired patients are being denied this life changing surgery. This is a common theme in a number of recent studies[5,6] and highlights that there is a significant unmet need within our community. The recent introduction of a national prioritisation tool (CPAC), which has been specially developed and validated to prioritise all patients accepted onto the waiting list, may enable direct comparison between subspecialties and different DHBs. This may result in better distribution of resources and improve equity of access. However, prioritising patients on a waiting list is irrelevant if they still fail to access surgery.

Not only is there a problem accessing waiting lists, there is also a significant problem in obtaining the first specialist assessment (FSA) required to even be considered for surgery, further compounding this unmet need. Triage of patients (rationing) is required to ensure that DHBs remain compliant with the Ministry's mandate that all patient accepted for a FSA must be seen within

four months. Recent data from Canterbury show that 74% of spinal referrals requesting a FSA were declined due to lack of clinical resource[7] and a similar pattern was observed for hip and knee referrals with approximately 50% declined. This is just the tip of the problem as targeted areas of need, such as hip and knee arthroplasty, have been relatively privileged compared to other musculoskeletal conditions.

Obviously there is a resource problem. Both of the above studies have documented the current failings in orthopaedic waiting times and shown that the current model of care is not working. Attracting increased health expenditure in this area has been difficult for a specialty which largely deals with healthy patients whose disability does not have the emotive overtones of specialties involved with "cancer, cardiac or kids". Increasing health expenditure alone, without increasing the overall "surgical resource" is not the solution. This must be recognised by the Ministry and a co-ordinated approach, with DHBs, Orthopaedic departments and the New Zealand Orthopaedic Association in conjunction with community engagement to change the model of health delivery, is urgently required to avoid the unnecessary suffering of patients with musculoskeletal disorders.

Author information:
Gary Hooper, Department of Orthopaedic Surgery and Musculoskeletal Medicine, University of Otago, Christchurch.
Corresponding author:
Gary Hooper, Professor and Head of Department, Department of Orthopaedic Surgery and Musculoskeletal Medicine, University of Otago, Christchurch.
gary.hooper@otago.ac.nz
URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7012

REFERENCES:

1. Statistics New Zealand 2006 & 2009 census. http://www.stats.govt.nz/Census.aspx

2. S Weinstein. The burden of musculoskeletal disease. JBJS, 98 (16) August 2016

3. Global Burden of Diseases, Injuries and Risk Factors Study 2013. Lancet. 2014;22(July). http://www.thelancet.com/themed/global-burden-of-disease.

4. Hooper G, Lee A, Rothwell A, Frampton C. Current trends and predictions in the utilisation rates of hip and knee replacement in New Zealand from 2001 to 2026. NZMJ 09/2014;127(1401):82–93

5. Gwynne-Jones D, Iosua E. Rationing of hip and knee replacement: effect of severity of patient reported symptoms and the demand for surgery in Otago. NZ Med J 2016; 129(1432):59–66

6. Blackett J, Carslaw Z, Lees D, et al. The impact of the six-month waiting target for elective surgery: a patient record study. NZ Med J 2014; 127 (1405):45–53

7. Inglis T, Schouten R, Dalzell K et al. Access to orthopaedic spinal specialists in the Canterbury public health system: quantifying the unmet need. NZ Med J 2016; 129(1442):20–25

Equity of publicly-funded hip and knee joint replacement surgery in New Zealand: results from a national observational study

Helen Harcombe, Gabrielle Davie, Sarah Derrett, Haxby Abbott,
David Gwynne-Jones

ABSTRACT

AIM: This study examines equity in the provision of publicly-funded hip and knee total joint replacement (TJR) surgery in New Zealand between 2006 and 2013 to: 1) investigate national rates by demographic characteristics; 2) describe changes in national rates over time; and 3) compare rates of provision between District Health Boards (DHBs).

METHODS: Hospital discharge data for people aged 20 years or over who had at least one hip or knee TJR between 2006 and 2013 was obtained from the Ministry of Health's National Minimum Dataset.

RESULTS: Higher TJR rates were observed among those aged 75–84 years, females, those of Māori ethnicity, those not living in rural or main urban areas and those in the most deprived socio-economic groups. TJRs increased from 7,053 in 2006 to 8,429 in 2013, however the rate was highest in 2007. In 2012–13, age-ethnicity-standardised rates varied between DHBs from 196 to 419/100,000 person years, with larger DHBs having lower rates than smaller DHBs.

CONCLUSION: There was evidence of geographic inequity in TJR provision across New Zealand. Despite increased numbers of procedures, rates of publicly-funded TJR surgery are barely keeping up with population increases. Reasons behind differences in provision should be examined.

Healthcare budgets are constrained and there are perennial concerns about the potential mismatch between health-care need and the provision of publicly-funded services. Ageing populations[1] and technological advances[2] are increasing pressures on healthcare budgets and the prioritisation of healthcare services can be required.[3] One area that is likely to be affected by these pressures is total joint replacement (TJR) surgery. The most common reason for TJR surgery in New Zealand is osteoarthritis (OA)[4] that is not responding adequately to conservative treatment. OA has a high prevalence among older adults[5,6] with 29% of New Zealanders aged over 65 years diagnosed with this condition.[7] This is important to consider as, currently in New Zealand, those aged over 65 years comprise 14% of the population but this is predicted to increase to 27% in 2063.[8] Therefore, demand for TJR will likely increase substantially. In New Zealand, hip TJR surgery (including privately-funded procedures) has already increased between 1999 and 2013 by 75%; there was a corresponding 158% increase for knee TJR surgery.[6] However, despite these increases, concerns have been raised that the provision of these procedures may not be expanding sufficiently to keep up with increases in clinical need or population changes.[9]

Publicly-funded healthcare in New Zealand is provided by 20 District Health Boards (DHBs) "...responsible for providing or funding the provision of health services in their district."[10] In New Zealand, prioritisation scoring tools are used to determine access to publicly-funded TJR surgery. This should ensure equitable access across the

country. However, Derrett et al (2009)[11] found a lack of equity between DHBs in the provision of elective hip and knee TJR (2000 to 2005), and an analysis of New Zealand newspaper articles and Parliamentary questions from 2000–2006 found that "...access inequities remained a persistent theme..." (p.57).[12] Although there has been an increase in funding for TJR surgery in New Zealand in recent years it is not clear whether that has translated into increased rates of surgical provision. Additionally, any increases in provision of TJR should be equitable with regard to geographic and demographic determinants such as place of residence, age, sex, ethnicity and socioeconomic deprivation.[13] This paper examines publicly-funded elective hip and knee TJR surgery provision among DHBs in New Zealand from 2006–2013. The aims of this study are to:

1. describe changes in rates of publicly-funded hip and knee TJR surgery nationally between 2006 and 2013,

2. investigate whether national rates vary according to age, sex, ethnicity, small-area deprivation and rurality, and

3. determine whether the provision of publicly-funded hip and knee TJR surgery is equitable across DHBs in New Zealand.

Methods

This study examined New Zealand hospital discharge data for publicly-funded hip and knee TJR surgery from 2006–2013. Ethical approval for the study was received from the University of Otago Human Ethics Committee (Reference number D13/253). Relevant hospital discharge data was obtained from the Ministry of Health's National Minimum Dataset (NMDS).[14] The NMDS is a national collection containing publicly-funded hospital discharges and some privately-funded hospital discharges. Data was obtained for patients with at least one publicly-funded hip or knee TJR procedure who were discharged between 1 January 2006 and 31 December 2013. This time period was chosen as similar work on this topic[11] analysed data up until the end of 2005, and 2013 data was the latest available at the time this study commenced. The

variables obtained from the NMDS included the International Classification of Diseases version 10 (ICD10) clinical code, age at discharge, sex, domicile code, ethnicity, type of admission, diagnosis type, event dates and the principal health service purchaser. As well as waiting list admissions, arranged admissions defined as "a planned admission where: the admission date is less than seven days after the date the decision was made by the specialist that the admission was necessary..."[14] were also included as these were likely to capture urgent sub-acute OA patients. Acute admissions and injury admissions (primary diagnosis code within ICD10 S00-T98)[15] were excluded as were those under 20 years of age at time of surgery and overseas residents. Hip and knee TJR surgeries were identified using the clinical codes in the 3rd edition of the Australian Modification of ICD10.[15] The specific procedures included were: total arthroplasty of hip, total arthroplasty of knee, total arthroplasty of knee with bone graft to femur or to tibia, total arthroplasty of knee with bone graft to femur and tibia and total replacement arthroplasty of patellofemoral joint of knee. Hemiarthroplasty of the knee was also included because indications for this are similar to TJR and their popularity may vary across the country. Revisions of hip and knee joint replacements were not included as the aim was to focus on primary procedures. Records with missing or historic domicile codes that could not be forward-mapped were excluded as these could not be analysed by DHB, area-level deprivation or rurality. Self-identified ethnicity data collected at the patient's health event was obtained from the NMDS. The recording of at least one ethnicity is mandatory, and two additional ethnic group codes may be recorded.[14] As the DHB-level denominator data was only available by 'prioritised ethnicity', this approach was used in our analyses with estimates obtained for Māori, Pacific, Asian and Other ethnicity groupings. Prioritisation follows a Statistics New Zealand (SNZ) algorithm with the end result being each person associated with only one ethnic group.[16] Māori ethnicity has the highest priority, meaning that people who identified as both Māori and any other ethnicities are classified as Māori. For example, those who identify as both Māori

and Pacific are classified as Māori. Pacific ethnicity is given the next highest priority with those who identify as Pacific and any other ethnicity (apart from Māori) being classified as Pacific.

The New Zealand Deprivation Index (NZDep2006) is a "...small-area index of relative socio-economic deprivation..."[17] (p.S7) derived from 2006 Census data. The NZDep scale runs from one (an area in the least deprived 10% of small areas) to 10 (in the 10% most deprived small areas). The 1:1 mapping between domicile codes available in the NMDS and Census area units used by SNZ enabled NZDep to be assigned to each TJR discharge record. Rurality was also derived from domicile codes by 1:1 mapping with SNZ's Census area units and SNZ's Urban/Rural Profile Classification.[18] The seven categories of the Urban/Rural profile were categorised for analysis as:

1. 'Main Urban' (described as being "...very large and centred on a city or main urban centre... minimum population of 30,000"),[18]

2. 'Other Urban' which consisted of 'Satellite Urban' ("defined as urban areas (other than main urban areas) where 20 percent or more of the usually resident employed population's workplace address is in a main urban area"[18] and 'Independent Urban' (defined as for Satellite Urban but <20 percent with a main urban area workplace), and

3. 'Rural' comprising the four rural profiles ('Rural Areas with a High Urban Influence,' 'Rural Areas with a Moderate Urban Influence,' 'Rural Areas with a Low Urban Influence' and 'Highly Remote Areas').

Denominator data were sourced from SNZ, and restricted to those aged 20 years and above. Annual resident population estimates by year, ethnicity, sex, age group and DHB region for 2006–2013 were calculated by SNZ. Usually resident population counts from the 2006 Census were used for calculations involving rurality and deprivation. In 2010 the Southern DHB was created from a merger of two DHBs (Otago and Southland); for this study we combined data from those DHBs and considered them as the Southern DHB throughout the period analysed. Crude rates per 100,000 person years (py) were calculated and presented alongside exact Poisson 95% Confidence Intervals (CIs). Age-standardised rates (ASRs) were calculated using direct standardisation and five-year age groups. Ten five-year age groups (<45, 45–49...80–84, 85+) were used for sex and ethnicity ASRs. Denominator data for deprivation and rurality ASRs was not available disaggregated by age for those over 65 years so these ASRs were calculated using age groups <45, 45–49, 50–54, 55–59, 60–64, 65+ years. Age- and ethnicity-standardised rates (AESRs) by DHBs were calculated in a similar way using four prioritised ethnic groups: Māori, Pacific, Asian and Other. Linear trends in rates were analysed using Poisson regression. Pitman's variance ratio test was used to compare the distribution of AESRs by DHB over time. Analyses were carried out using Stata/SE (version 13.1).[19]

Results

Of the 74,784 procedures obtained from the NMDS for people with at least one publicly-funded hip or knee TJR and a date of discharge between 2006 and 2013, 62,907 (84.1%) met the inclusion criteria. Figure 1 details the exclusions. Of these 62,907 publicly-funded primary hip or knee TJR procedures, 2% were bilateral joint replacements giving a total of 64,222 primary hip or knee joints replaced (Table 1).

Figure 1:

New Zealand hospital discharge records (2006–2013) for people with at least one publicly-funded hip or knee total joint replacement (n=74,784)

Ineligible (n=11,775) comprised of:

Overseas residents (n=88)

Under 20 years old (n=40)

Discharges with primary diagnosis of injury (S00–T98):

Fractured femur (S72) (n=2,916)

Other primary diagnosis injury (S00–S71, S73–T78) (n=280)

Complications or sequelae (T79-T98) (eg T84 complications of internal orthopaedic prosthetic devices, implants and grafts) (n=477)

Acute admissions (n=1,678)

Psychiatric patients returned from more than 10 days leave (n=3)

Joint replacement revision procedures (n=6,293)

Data not analysable (n=102) comprised of:

Missing domicile codes (n=39)

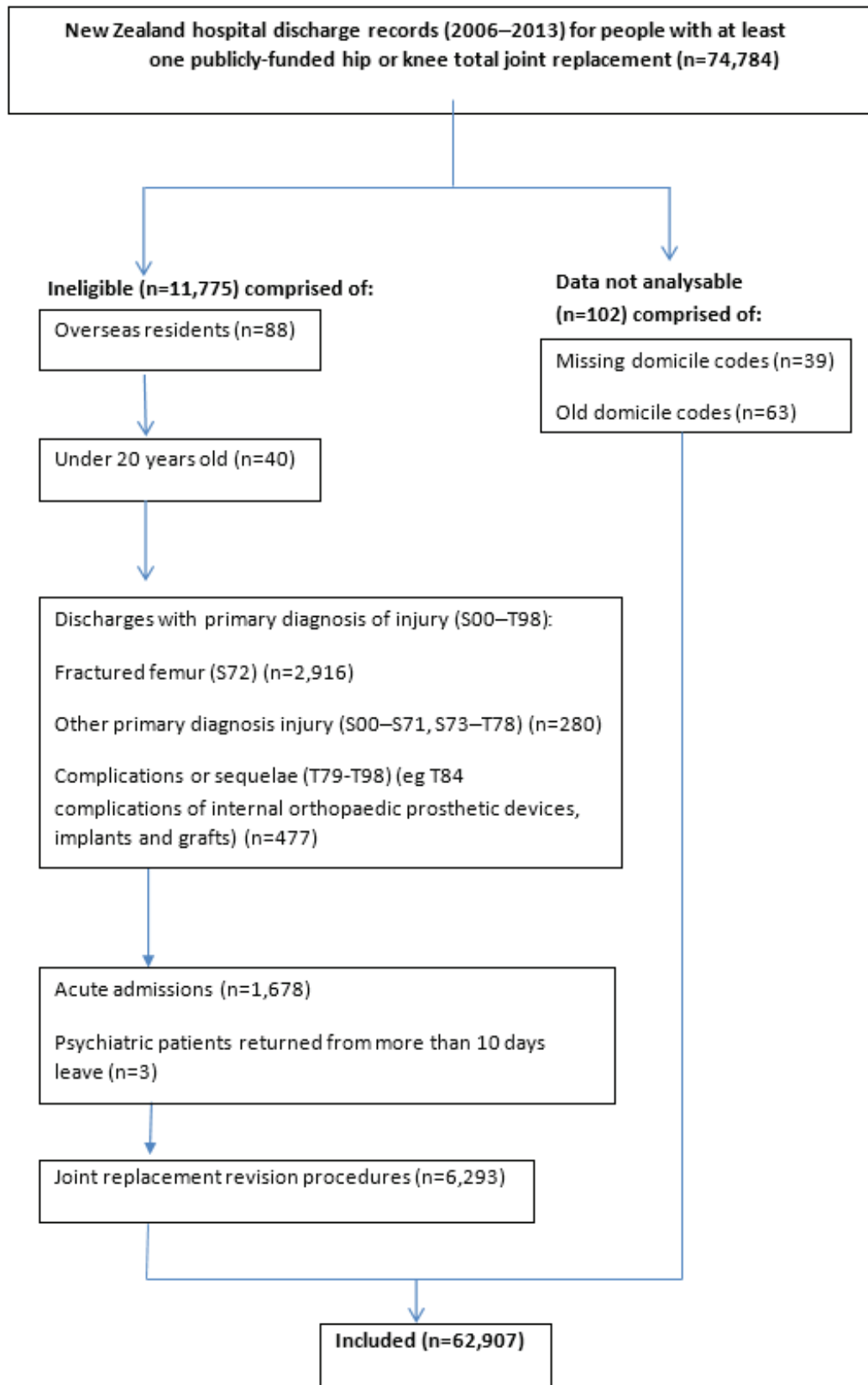Old domicile codes (n=63)

Included (n=62,907)

Table 1: Publicly-funded primary total hip and knee joint replacement procedures in those aged 20 years and over, 2006–2013 by District Health Board (DHB).

District Health Board	Overall N	Bilateral %	Population*	Overall Crude Rate** (95% CI)	Ranking/20***
Auckland	3,472	1.8	330,660	131.3 (126.9, 135.7)	20
Bay of Plenty	4,373	1.9	149,663	365.2 (354.5, 376.2)	5
Canterbury	6,781	2.5	367,993	230.3 (224.9, 235.9)	16
Capital and Coast	3,001	5.4	211,259	177.6 (171.3, 184.0)	19
Counties Manukau	5,636	2.8	318,674	221.1 (215.3, 226.9)	17
Hawke's Bay	2,703	0.2	109,900	307.4 (296.0, 319.3)	11
Hutt Valley	1,918	5.3	100,735	238.0 (227.5, 248.9)	15
Lakes	1,827	1.4	71,043	321.5 (306.9, 336.6)	9
Mid Central	2,719	1.2	118,968	285.7 (275.1, 296.6)	12
Nelson Marlborough	3,015	2.4	102,653	367.1 (354.2, 380.5)	4
Northland	2,987	1.8	112,131	333.0 (321.2, 345.1)	8
South Canterbury	1,460	0.3	41,890	435.7 (413.6, 458.6)	3
Southern	4,734	2.7	222,008	266.5 (259.0, 274,2)	14
Tairawhiti	889	0.1	31,134	356.9 (333.8, 381.2)	7
Taranaki	2,013	1.3	79,248	317.5 (303.8, 331.7)	10
Waikato	5,757	1.4	256,493	280.6 (273.4, 287.9)	13
Wairarapa	866	0.9	29,839	362.8 (339.0, 387.8)	6
Waitemata	6,274	2.0	378,591	207.1 (202.1, 212.3)	18
West Coast	871	0.6	24,241	449.1 (419.8, 480.0)	1
Whanganui	1,611	0.8	45,012	447.4 (425.8, 469.8)	2
Total	**62,907**	**2.1**	**3,102,133**	**253.5 (251.5, 255.5)**	

*Population = Average DHB population for 2006–2013 of those aged 20 years and over.
** Rate/100,000 person years.
***Ranking is from highest to lowest overall crude rate for the 20 DHBs.

Table 2: Publicly-funded hip and knee total joint replacement procedures in New Zealand for those aged 20 years and over from 2006–2013 by year.

Discharge Year	Denominator	Number	Rate*	95% CI
2006	2982345	7,053	236.5	(231.0, 242.1)
2007	3015800	7,943	263.4	(257.6, 269.2)
2008	3046505	7,535	247.3	(241.8, 253.0)
2009	3083845	7,934	257.3	(251.7, 263.0)
2010	3124770	7,745	247.9	(242.4, 253.4)
2011	3158140	7,950	251.7	(246.2, 257.3)
2012	3185125	8,318	261.2	(255.6, 266.8)
2013	3220535	8,429	261.7	(256.2, 267.4)

*Rate/100,000 person years of those aged 20 years and over.

Nationally, the number of publicly-funded hip and knee TJR procedures increased by 19.5% from 7,053 in 2006 to 8,429 in 2013 (Table 2) while the rate increased by only 10.7%. The rate peaked in 2007 at 263/100,000 py before decreasing (2008–2011) and returning to 261 and 262/100,000 py in 2012 and 2013 respectively. Although there was a statistically significant increase in the rates from 2006 onwards (p-value <0.001), there is no evidence to suggest a linear change in the rates from 2007 onwards (p-value=0.2).

From 2006 to 2013 inclusive, the highest rate of publicly-funded hip and knee TJR procedures was for those aged 75–84 years at the time of surgery (1,063/100,000 py) followed by those aged 65–74 (907/100,000 py), with the lowest rate among those aged less than 55 years (45/100,000 py) (Table 3). ASRs were significantly higher for females (260/100,000 py) than for males (246/100,000 py).

The crude TJR rate of 300/100,000 py was highest among those categorised as 'Other' ethnicity (ie those not identifying

Table 3: Publicly-funded primary hip and knee total joint replacement procedures for those aged 20 years and over for 2006–2013 by socio-demographic characteristics.

	Denominator*	N	Annual crude rate	(95% CI)**	ASR***	(95% CI)
Overall	**3,102,133**	**62,907**	**253.5**	**(251.5, 255.5)**	**--**	**-- --**
Age group (years)						
<55	2,066,311	7,405	44.8	(43.8, 45.8)	--	-- --
55–64	474,338	14,939	393.7	(387.4, 400.0)	--	-- --
65–74	311,123	22,581	907.2	(895.4, 919.2)	--	-- --
75–84	183,611	15,611	1062.8	(1046.2, 1080.0)	--	-- --
85+	66,751	2,371	444.0	(426.3, 462.2)	--	-- --
Sex						
Female	1,612,114	34,075	264.2	(261.4, 267.0)	259.7	(257.0, 262.5)
Male	1,490,019	28,832	241.9	(239.1, 244.7)	246.2	(243.4, 249.1)
Prioritised Ethnicity						
Māori	366,255	5,793	197.7	(192.7, 202.9)	303.1	(294.8, 311.5)
Pacific	158,319	1,809	142.8	(136.3, 149.6)	224.2	(213.4, 235.0)
Asian	327,923	1,259	48.0	(45.4, 50.7)	93.8	(88.2, 99.3)
Other****	2,249,636	54,046	300.3	(297.8, 302.9)	258.3	(256.1, 260.4)
Rurality						
Main Urban Area	2,037,012	40,003	245.5	(243.1, 247.9)	258.6	(256.1, 261.1)
Other Urban Area	399,417	14,672	459.2	(451.8, 466.7)	361.6	(355.7, 367.5)
Rural	362,802	8,230	283.6	(277.5, 289.8)	295.9	(289.4, 302.3)
NZDep						
1–3 (least deprived)	785,292	13,314	211.9	(208.3, 215.6)	219.6	(215.9, 223.4)
4–7	1,136,757	26,780	294.5	(291.0, 298.0)	279.9	(276.6, 283.3)
8–10 (most deprived)	877,113	22,806	325.0	(320.8, 329.3)	342.0	(337.6, 346.5)

*Uses 2006–2013 resident population estimates for all except Rurality and NZDep comparisons which use 2006 usually resident Census counts.
**Rate/100,000 person years (≥20 year-olds).
***Age-standardised rate.
****The numerator of those classified as 'other' ethnicity includes those with ethnicity recorded as 'Don't Know,' 'Refused to answer,' 'Response unidentifiable' or 'Not Stated' to align with denominator.

Figure 2: Age- and ethnicity-standardised rates of publicly-funded hip and knee total joint replacements per 100,000 person-years by District Health Board from 2006–2013.
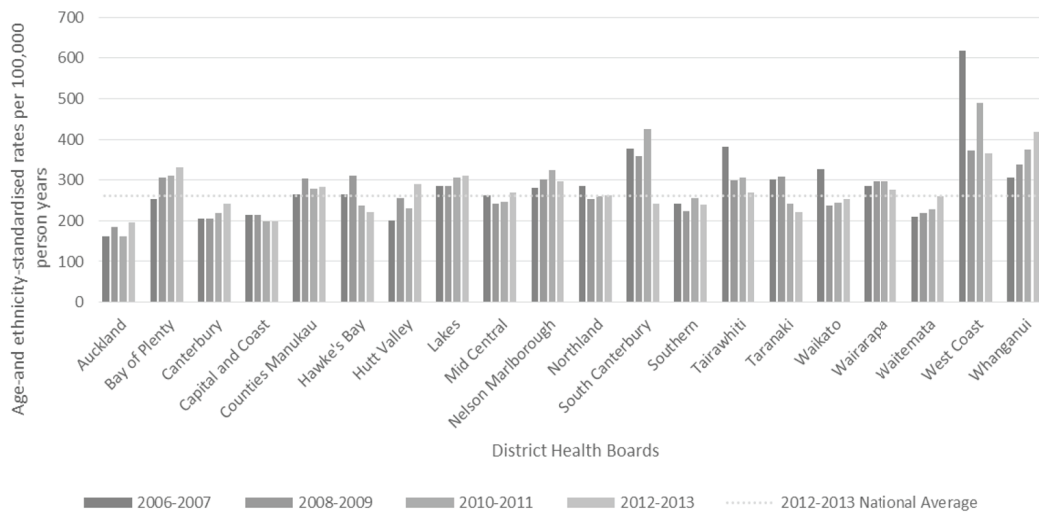
as Māori, Pacific or Asian). Māori had the second highest crude rate (198/100,000 py). However, Māori had the highest ASR of procedures (303/100,000 py) followed by those of 'Other' and Pacific ethnicities (258 and 224/100,000 py respectively). Those of Asian ethnicity had a substantially lower ASR (94/100,000 py). Differences in crude and ASRs between prioritised ethnic groups were all statistically significant.

Rates were highest for people living in 'Other Urban Areas' (ie urban areas other than those classified as centred on a city or main urban centre) with a crude rate of 459/100,000 py and an ASR of 362/100,000 py). This ASR was significantly higher than the ASR for those in 'Rural' (296/100,000 py) and 'Main Urban Areas' (259/100,000 py).

There was a clear linear relationship between TJR procedure rates and socio-economic deprivation, with people that lived in the most deprived three deciles (deciles 8–10) having a significantly higher ASR (342/100,000 py) than those in deciles 4–7 (280/100,000 py) and similarly those who lived in the least deprived deciles (deciles 1–3) had a substantially lower ASR again (220/100,000 py).

Of the 20 DHBs, 10 had increases in their age- and ethnicity-standardised rate (AESR) of TJR procedures between the periods 2006–07 to 2012–13, one was unchanged and nine had a reduced rate (Figure 2). Of the eight largest DHBs by population, five had an increase in AESR: Bay of Plenty (31%), Auckland (22%), Waitemata (25%), Canterbury (19%) and Counties Manukau

(7%). Southern's AESR remained unchanged and Capital Coast's and Waikato's fell by 7% and 22% respectively. In contrast, AESRs fell between the periods 2006–07 to 2012–13 in seven of the 12 smaller DHBs: West Coast, Wairarapa, Tairawhiti, South Canterbury, Taranaki, Hawke's Bay and Northland. However, in 2012–13, the six smallest DHBs by population (with the exception of South Canterbury) had AESRs higher than five of the six DHBs with the largest populations. Five of the eight largest DHBs (Auckland, Canterbury, Capital and Coast, Southern and Waikato) were below the New Zealand average of 261/100,000 py in 2012–13 as were three of the smallest DHBs (Hawkes Bay, Taranaki and South Canterbury). To assess whether the variation in AESRs by DHB had changed over time, the standard deviation of DHB's AESRs for 2006–07 was compared with that from the rates for 2012–13. Excluding one outlier (West Coast), there was no statistically significant difference over time (ratio of standard deviations 1.13, (95% CI 0.70, 1.82), p=0.6).

There were also variations by DHB for those in the most deprived deciles. For those in the most deprived three deciles, considering the eight years of the study combined, the ASRs varied from 236/100,000 py (Auckland) to 514/100,000 py (South Canterbury) (results not shown). Again, the smaller DHBs had greater provision within this group of the population, with the five smallest DHBs by population having ASRs of at least 400/100,000 pys, a rate which was not reached for the most deprived deciles in any of the other larger DHBs.

Discussion

This study demonstrates that national rates of publicly-funded elective hip and knee TJR procedures have not increased beyond their 2007 peak. Higher rates were observed in older adults, females, those not living in 'Rural' or 'Main Urban Areas' and those living in areas of greater social deprivation. Rates varied between DHBs, even when age- and ethnicity-standardised. In general, there were higher rates of the provision of publicly-funded hip and knee TJR procedures among the smallest DHBs in New Zealand compared with the largest population DHBs in 2012–13.

A strength of this study was the use of consistently collected data for the entire New Zealand population. A limitation is that domicile was obtained from the National Health Index database which is updated when patients present at their DHB and therefore may no longer reflect the domicile as it was at the time of surgery for all participants. This study is restricted to publicly-funded procedures, therefore it does not consider the overall provision of TJR surgery, some of which are privately-funded. Comparing NMDS discharge data of publicly-funded hip and knee TJR with National Joint Registry data which includes both privately- and publicly-funded procedures,[4] it appears that approximately 65% of TJR were publicly-funded in New Zealand between 2006–2012. The provision of privately-funded procedures may vary by DHB and may influence the provision of publicly-funded procedures. Derrett et al[11] previously reported that DHBs with low rates of publicly-funded hip and knee TJR procedures had high rates of privately-funded procedures. A further limitation of our analyses is that we have reported on the provision of TJR; provision does not necessarily reflect demand for procedures or the severity of disease. Previous research has suggested that there is unmet need for these procedures in New Zealand.[9,13] Demand may vary across the country[9] and in some DHBs, 33–41% of patients listed for TJR are being returned to their General Practitioner without surgery due to waiting time targets.[20] There was an increase in the number of publicly-funded hip and knee TJR procedures carried out nationally between 2006 and 2013 in those aged 20 years and over. However, the bulk of the increase in both numbers and rate occurred between 2006 and 2007 as the Orthopaedic Joint Initiative ("...a programme of increased funding specifically targeting major joint replacement...") (p.15)[21] finished. During the period of this study, 2006–2013, the rate was highest in 2007. Between 2007 and 2013 the number of publicly-funded TJR procedures increased by 486 (6%) but the rate decreased by 0.6% suggesting that the increased number of publicly-funded TJR procedures is barely keeping up with population increases. Hooper et al[22] have predicted that numbers of hip and knee replacements will increase significantly by 2026. Such a predicted increase has clear implications for public funding of TJRs. The highest rate of hip and knee TJR procedures was for those people aged 75–84 years followed by 65–74 year olds. This is not surprising and aligns with the higher prevalence of OA among older age groups.[5] As life expectancy increases, it is likely that demand in the over 85 year-olds will increase.

Although the rate of procedures was higher among females, the difference between males and females was relatively small and probably reflects the higher prevalence of OA among women.[5]

Nationally, people in the least deprived deciles had the lowest rate of publicly-funded TJR procedures while those in the most deprived deciles had the highest rate. This is open to a number of different interpretations. Poorer access to medical care in the lower deciles might be expected to lead to a decreased rate of TJR rather than the increased rate seen. It is likely that there is greater use of private surgery by those of higher socio-economic status either through insurance or self-funding. However the findings may also reflect greater need for TJR among people of lower socio-economic status (for example, if need is related to type of occupation). However, no direct link between socio-economic deprivation and joint replacement has yet been identified other than possibly obesity; people in the most deprived areas of New Zealand having higher rates of obesity compared with those in the least deprived areas.[23]

Nationally, by prioritised ethnicity, Māori had the highest ASR of publicly-funded TJR

procedures. In a series of patients from a regional registry, Singleton et al[24] found that Māori were younger, had poorer pre-operative function than non-Māori patients and comprised 13.7% of their TJR procedures but only 11.2% of their population. Hooper et al[22] reported a relative rate in Māori of 0.72 for hip and 0.76 for knee TJR compared with those of 'European' ethnicity using data from the New Zealand Joint Registry. The main differences between that study and the current study are that privately-funded and acute procedures are included in the Joint Registry figures and that their analysis was based on joints not procedures. Their use of total response rather than prioritised ethnicity will not affect the rate for Māori as Māori are given top priority in our analysis by prioritised ethnicity. It is possible that lower rates of private utilisation among Māori may explain the difference in findings. It is not clear whether the higher rate of TJR in the current study is a reflection of an additional need among Māori or whether it is due to greater demand in the public sector due to lower private provision. It has been recognised previously that ethnicity data collected in the NMDS may undercount people of Māori ethnicity[25] which may have influenced the findings of this study. However, if Māori undergoing TJR surgery were less likely to be classified as Māori in the NMDS, the rate reported for Māori would be an under-estimate. It is unclear why rates were substantially lower among those of Asian ethnicity compared with those of Māori, Pacific or 'Other' ethnicities. It is possible that this may relate to more privately-funded procedures among this ethnic group. However Hooper et al[22] found similar results while including privately-funded procedures and suggested that older Asians living in New Zealand may return to their home country for joint surgery.[22] The ASR of TJR for people of Pacific ethnicity was over twice the rate for those of Asian ethnicity but was still significantly lower than the rate for those of Māori and 'Other' ethnicities. It is unclear why this is the case given Pacific people are highly represented in the most deprived areas of New Zealand and have higher rates of obesity compared with other ethnicities.[23] As the DHB-level denominator data was only available by 'prioritised

ethnicity,' estimates for Pacific people do not include those who identified with both Māori and Pacific ethnic groups. Similarly, those who responded as being of both Pacific and Asian ethnicity are only included as Pacific.

There were differences in procedure rates by rurality with the highest rate for those living in 'Other Urban Areas' and the lowest rate for those in 'Main Urban Areas.' The lower rate for those living in 'Main Urban Areas' may have been influenced by a greater availability of private procedures in these areas but we cannot determine that in this study. While there may be some relationship between rurality and DHB-specific rates, the denominator data available for this analysis precluded examining this.

In the current study, AESRs varied by DHB with a 3.8 fold rate variation between lowest and highest in 2006–07 and a two-fold rate variation in 2012–13. However, if one outlier was excluded, there was no statistically significant change in the variation between DHBs from 2006–07 to 2012–13. In other words, there has been no apparent improvement in the equity of provision of publicly-funded TJR across DHBs over the eight years of the study period. Derrett et al,[11] although not standardising for ethnicity and also including revision procedures, reported nearly a five-fold variation of ASRs for publicly-funded TJR between DHBs in 2001–2002. They also reported geographic inequity for those in the poorest three deciles and found that rates of publicly-funded procedures were lowest for this group of people in DHBs that had the highest rates of privately-funded procedures.[11] Examining the ASR of publicly-funded TJR procedures by DHB for those in the most deprived deciles in the current study also found that rates varied considerably.

The larger DHBs typically had lower rates of publicly-funded TJR compared with the smaller DHBs and five of the eight largest DHBs had rates that were below the New Zealand average in 2012–13. These findings indicate that those living within the largest DHBs (by population) may be disadvantaged in terms of access to publicly-funded hip and knee TJR surgery. We cannot determine the reasons behind these findings. There may be greater access to private surgery in the larger DHB regions which may reduce

the demand for public surgery. It has also been suggested that higher rates of private surgery could lead to lower rates of publicly-funded surgery due to surgeons not being available for public work.[11] Other factors such as high acute loads and complex tertiary referrals, which are likely to be more common in larger DHBs, may also influence access to publicly-funded procedures.

Conclusion

Despite an increase in the number of publicly-funded hip and knee TJR procedures between 2006 and 2013, the national increase in rate has been negligible since 2007 suggesting that the increased number of procedures may be only just keeping up with increases in the population. While the data demonstrated higher rates in older adults, females, people of Māori ethnicity, and those living in areas of greater social deprivation and 'other urban areas,' there was no systematic evidence of inequities disadvantaging vulnerable, higher needs or isolated groups, although this study did not include privately-funded procedures. In general, there were higher rates of the provision of publicly-funded hip and knee TJR procedures among the smallest DHBs in New Zealand, by population, compared with the largest population DHBs. The finding that rates vary between DHBs, even when age- and ethnicity- standardised, suggest equity among DHBs is not being achieved nationally. This indicates that further work is required to meet one of the key objectives in the New Zealand Ministry of Health's programme for elective surgery which is to "Work towards everyone having equal access to elective surgery no matter where they live".[26] Further research, using validated scoring tools, is needed to compare access to TJR according to need and to examine reasons behind differences in provision.

Author information:

Helen Harcombe, Lecturer, Preventive and Social Medicine, University of Otago, Dunedin; Gabrielle Davie, Senior Research Fellow, Preventive and Social Medicine, University of Otago, Dunedin; Sarah Derrett, Associate Professor, Preventive and Social Medicine, University of Otago, Dunedin; Haxby Abbott, Research Associate Professor, Department of Surgical Sciences, University of Otago, Dunedin; David Gwynne-Jones, Associate Professor, Department of Surgical Sciences, University of Otago, Dunedin.

Corresponding author:

David Gwynne-Jones, Department of Surgical Sciences, University of Otago, Great King Street, Dunedin.
david.gwynne-jones@otago.ac.nz

URL:

https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7017

REFERENCES:

1. Rice DP, Fineman N. Economic implications of increased longevity in the United States. Annu Rev Public Health. 2004;25:457–73.

2. Okunade AA, Murthy VNR. Technology as a 'major driver' of health care costs: a cointegration analysis of the Newhouse conjecture. J Health Econ. 2002 Jan;21:147–59.

3. Ministry of Health. Targeting more elective operations. Improved access to elective surgery. Ministry of Health; 2011; Available from: http://www.health.govt.nz/system/files/documents/publications/targeting-electives-health-target.pdf.

4. The New Zealand Joint Registry Fifteen Year Report January 1999 to December 2013. 2014 [30 January 2014]; Available from: http://www.nzoa.org.nz/system/files/NZJR2014Report.pdf.

5. Buckwalter JA, Saltzman C, Brown T. The impact of osteoarthritis: implications for research. Clin Orthop Relat Research. 2004 Oct:S6–15.

6. Hooper G. The aging population and the increasing

demand for joint replacement. NZMJ. 2013;126.

7. Ministry of Health. Regional results from the 2011-2014 New Zealand Health Survey. 2015 [30 October 2015]; Available from: http://www.health.govt.nz/publication/regional-results-2011-2014-new-zealand-health-survey.

8. Statistics New Zealand. 2013 Census QuickStats about people aged 65 and over. 2015; Available from: www.stats.govt.nz.

9. Gwynne-Jones D. Quantifying the demand for hip and knee replacement in Otago, New Zealand. NZMJ. 2013 Jun 28;126:7–17.

10. Ministry of Health. District health boards. [Accessed 30 January 2015]; Available from: http://www.health.govt.nz/new-zealand-health-system/key-health-sector-organisations-and-people/district-health-boards.

11. Derrett S, Bevin TH, Herbison P, Paul C. Access to elective surgery in New Zealand: considering equity and the private and public mix. Int J Health Plann Manage. 2009 Apr-Jun;24:147–60.

12. Derrett S, Cousins K, Gauld R. A messy reality: an analysis of New Zealand's elective surgery scoring system via media sources, 2000-2006. Int J Health Plann Manage. 2013 Jan-Mar;28:48–62.

13. Derrett S, Paul C, Herbison P, Williams H. Evaluation of explicit prioritisation for elective surgery: a prospective study. J Health Serv Res Policy. 2002 Jul;7 Suppl 1:S14–22.

14. National Health Board. National Minimum Dataset (Hospital Events) Data Dictionary. Wellington: Ministry of Health; 2014.

15. NCCH (National Centre for Classification in Health). The International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification (3rd edn). Sydney: NCCH, Faculty of Health Sciences, The University of Sydney; 2002; Available from: http://meteor.aihw.gov.au/content/index.phtml/itemId/270546.

16. Allan J. Review of the measurement of ethnicity. Statistics New Zealand; 2001 [2 September 2015]; Available from: http://www.stats.govt.nz/browse_for_stats/population/census_counts/review-measurement-of-ethnicity/classifications-and-issues.aspx.

17. Salmond CE, Crampton P. Development of New Zealand's Deprivation Index (NZDep) and its uptake as a national policy tool. Can J Public Health. 2012;103(Suppl 2):S7–S11 page S7.

18. Statistics New Zealand. Defining urban and rural New Zealand. [Accessed 14 August 2015]; Available from: http://www.stats.govt.nz/browse_for_stats/Maps_and_geography/Geographic-areas/urban-rural-profile/defining-urban-rural-nz.aspx.

19. StataCorp. Stata Statistical Software: Release 13: College Station, TX: StataCorp LP; 2013.

20. Blackett J, Carslaw A, Lees D, et al. The impact of the 6-month waiting target for elective surgery: a patient record study. NZMJ. 2014 Nov 7;127:45–53.

21. Synergia Limited. 2008. A review of the elective services orthopaedic major joint and ophthalmology cataract initiatives: A report prepared for the Ministry of Health. Wellington: Ministry of Health.

22. Hooper G, Lee AJ, Rothwell A, Frampton C. Current trends and projections in the utilization rates of hip and knee replacement in New Zealand from 2001 to 2026. NZMJ. 2014 Aug 29;127:82–93.

23. Ministry of Health. Annual update of key results 2013/14: New Zealand Health Survey. Wellington: Ministry of Health; 2014.

24. Singleton N, Buddicom E, Vane A, Poutawera V. Are there differences between Maori and non-Maori patients undergoing primary total hip and knee arthroplasty surgery in New Zealand? A registry-based cohort study. NZMJ. 2013 Aug 2;126:23–30.

25. Cormack D, Harris R. Issues in monitoring Māori health and ethnic disparities: an update. Te Rōpū Rangahau Hauora a Eru Pōmare: Wellington. 2009.

26. Ministry of Health. About the electives programme. [Accessed 2 Sept 2015]; Available from: http://www.health.govt.nz/our-work/hospitals-and-specialist-care/elective-services/about-electives-programme

Access to orthopaedic spinal specialists in the Canterbury public health system: quantifying the unmet need

Tom Inglis, Rowan Schouten, Kristian Dalzell, Jeremy Evison, Grahame Inglis

ABSTRACT

AIM: The aim of this project was to determine the unmet need within the public health system for patients referred for elective Orthopaedic Specialist Spinal assessment and treatment in the Canterbury District Health Board (CDHB) region.[1]

METHODS: Between January 2014 and January 2015 data was collected from all elective referrals to the CDHB Orthopaedic Spinal Service. During this period, the number of available outpatient appointments was set by the CDHB. Within this clinical capacity, patients were triaged by the four consultant surgeons into those of most need based on the referral letter and available radiological imaging. Those unable to be provided with a clinical appointment were discharged back to their GP for ongoing conservative care. Of those patients that received specialist assessment and were considered in need of elective surgical intervention, a proportion were denied treatment if the surgery was unable to be performed within the government determined four-month waiting time threshold.

RESULTS: During the study period, 707 patients were referred to the CDHB orthopaedic spinal team for elective specialist assessment. Of these, 522 (74%) were declined an outpatient appointment due to a lack of available clinical time. Of the 185 patients given a specialist assessment, 158 (85%) were recommended for elective surgery. Ninety-one (58%) were denied surgery and referred back for ongoing GP care due to unavailable operating capacity within the four-month waiting list threshold. Within this group of 91 patients, 16 patients were declined on multiple occasions (14 patients twice and two patients on three occasions).

CONCLUSIONS: This study quantifies the unmet need for both Spinal Orthopaedic Specialist assessment and, if warranted, surgical management of elective spine conditions within the Canterbury public health system. It highlights the degree of rationing within the public health system and its failure to adequately provide for the Canterbury Public.

The CDHB is the second largest district health board in New Zealand. It provides public health care to a population of over 500,000 people and provides orthopaedic services to the West Coast region through a clinical partnership. It is also the tertiary referral centre for elective spinal surgery in the South Island and manages acute spinal cord injuries occurring throughout the South Island and the lower half of the North Island.

Locally, elective orthopaedic spinal referrals are largely dominated by degenerative conditions such as lumbar spinal stenosis. Spinal stenosis involves narrowing of the spinal canal by bone and soft tissue with subsequent pressure on the neural structures. This leads to symptoms characterised by debilitating pain in the buttocks and legs when walking or standing.[2,3] In this setting, surgical decompression with or without stabilisation ('fusion') has been shown to be clinically effective and superior to non-operative management. [3]

The population within Canterbury is aging. By 2026 one in every five people in Canterbury will be over 65 and the number of people aged over 85 will have doubled.[1]

Spinal stenosis is the most common reason for both cervical and lumbar spine surgery in adults over 65.[4,5] As the elderly proportion of our patient population grows we can expect demand on our service to increase accordingly.[6]

In recent years a key priority of the New Zealand Government has been to reduce the maximum waiting time to four months for patients requiring an initial consultation with a surgical hospital specialist (first specialist assessment (FSA)) or surgical treatment.[7]

Since its inception in January 2015, the introduction of a four-month maximum waiting time for elective FSA and surgery has raised concerns among medical providers. With the introduction of maximum waiting times, patients are required to be triaged so that they will receive their surgery within the expected time frame. Those patients unable to have their procedure within this time are returned to their GP for ongoing care, missing out on specialist care altogether. These patients represent the 'unmet need' within the medical system and currently this cohort has not been measured or monitored. In the past when the public demand for surgery exceeded supply, this was recorded via the hospital surgical waiting list with the length of waiting list reflecting the needs of the service.

The aim of this study was to quantify the "unmet need" within the CDHB in terms of patients referred to the Orthopaedic Spinal Service.

Methods

From the period of January 2014 to January 2015 the fate of every patient referred to the elective CDHB Orthopaedic Spinal Service was recorded.

Referrals were received from hospital specialists (frequently orthopaedic surgical colleagues) and general practitioners within the CDHB catchment region as well as tertiary referrals from around the South Island. All referrals were seeking advice on the management of patients with non-acute spinal pathology. Those patients with traumatic or oncologic pathology were managed within the acute service and were not included in this triaging process.

Within the CDHB the number of new spinal patients able to be seen by an Orthopaedic Spine Specialists in each clinic are limited. According to the new government imposed guidelines, only those people able to receive their surgery within a four-month time are given a place on the surgical waiting list.

While all referrals from GPs and other hospital specialists are deserving of clinical review, only a minority could be accommodated within the current resource allocation. At each triaging session the CDHB managers stated the number of FSA clinical appointments available. In order to identify those patients in most need of this limited resource, all referral letters to the Orthopaedic elective spinal service were reviewed by one of four fellowship trained Orthopaedic spine surgeons. The description of their presenting symptoms and clinical signs in the referral letter were critically assessed. All relevant radiological studies (plain x-rays and occasionally Computed-Tomography (CT) scans) were also reviewed. Almost universally an MRI scan was organised to assist with the triaging process. No specific clinical prioritisation scoring tool was utilised. Those considered by the spinal surgeons to have the most severe symptoms and pathology, and who were considered amenable to surgical treatment, were offered a first specialist assessment (FSA). The remainder are referred back to their GP. If indicated, a dictated letter is provided containing advice for further management or investigations prior to re-referral.

Information on the outcome of each referral was recorded including those referred patients who were denied a specialist assessment. For those patients who received a FSA their clinical records were analysed to determine if they were recommended for surgery or conservative management.

For those patients for whom surgical intervention was recommended, a second triaging process occurs based on the available operating capacity. Surgeons are required to determine which of those patients put forward for surgery will actually be able to have surgery performed within the now permitted four-month period as mandated by the CDHB. This is

done according to need; those who are the most incapacitated and would receive the most benefit from surgery are prioritised. Those patients who qualify are then effectively booked to receive their operative intervention within four months while those who are unable to be offered surgery within the time frame are returned to the care of their GP for ongoing management and re-referral to the service as required.

The number of patients who were operated on in the corresponding time period was determined from the clerical records.

Results

In the year from January 2014 to January 2015 a total of 707 patients were referred to the CDHB orthopaedic spinal team for specialist assessment from their general practitioner or other specialist services. Of these 707 patients, 522 (74%) were unable to be offered an outpatient appointment due to resource constraints. Of the 185 (26%) patients seen in clinic, 158 patients were recommended to have surgery (85%). Of these 158 surgically suitable patients, 91 (58%) were denied surgery and sent back to their general practitioner for ongoing care due to a lack of surgical resources. Within this cohort, 14 patients had been declined twice and two patients on three occasions.

Almost exclusively only patients with symptoms and radiological findings consistent with severe spinal stenosis could be offered specialist assessment and surgical treatment. Other spinal pathology was unable to be accommodated within the constrained system.

On review of the surgical booking records, we were able to determine that the Canterbury Spinal Team performed 68 spinal procedures over this interval. Therefore, the database missed one patient who received surgery during the study period.

Discussion

This study has quantified the unmet need for Spinal Orthopaedic Specialist services in the Canterbury Public Health System. During a 12-month period beginning in January 2014, 522 (74%) patients referred to this service were declined a first specialist assessment. Of those patients who were assessed and surgical intervention recommended, 91 (58%) were denied access to this procedure and sent back to the care of their GP. Our figures likely underestimate the clinical need as it remains unknown what proportion of patients unable to be offered a FSA would have also benefited from spinal surgery.

Figure 1: Flowchart of the referral system.
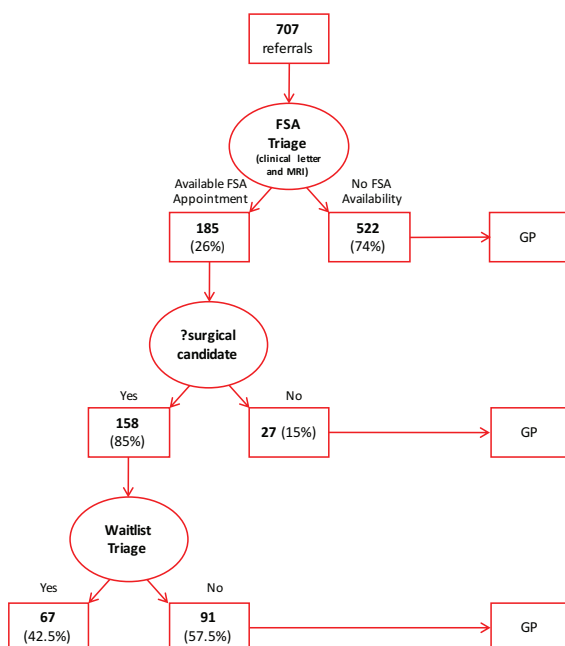
Despite access to public health care being a critical issue to many New Zealanders, currently there is no accurate measure of the number of people unable to access the health care their doctors consider they require. As a consequence, a true assessment of how well the New Zealand public health system is functioning remains incomplete. Both the Ministry of Health and independent projects are underway to address this information void.

The health burden of spinal pathology is significant and on par or greater than other chronic conditions including diabetes, heart disease, stroke and arthritis.[8] There exists abundant literature that demonstrates spinal surgery is beneficial in terms of clinical outcomes and cost-effectiveness compared with non-operative treatment for common degenerative spinal conditions.[4,6,9,10,11] Currently, hip and knee arthroplasty surgery is considered a priority surgical service in most DHB due to its well-proven clinical and cost-effectiveness. The degree of post-operative health related quality of life (HRQoL) improvement seen in patients with spinal stenosis following surgery compares favourably to the benefits received following hip and knee arthroplasty[13–16] and has a similar incremental cost-utility ratio.

This study demonstrates the degree of rationing occurring at two levels within our DHB. The first involves access to a FSA, a service afforded to one in four referrals. The second bottleneck involved patients considered to benefit from surgery by a specialist surgeon. Within this study period 57% of patients were denied an operation, as they were unable to be accommodated within the government specified four-month waiting period. Instead patients were referred back to their GP's care to continue less effective conservative treatments.

A high-level review performed in 2013 by an expert panel convened by the Director General of Health to determine if there were any unintended consequences for patient care[17] concluded that there was "no evidence that the pursuit of elective waiting time goals has resulted in unintended consequences for patient care." Our data demonstrates the contrary.

To address the goal of improving access to specialist spine services firstly requires a recognition that a significant unmet need exists. The Government and DHBs have been lacking in their willingness to quantify this and monitor the health consequences of those patients denied care.

Returning patients to the care of their GPs is unlikely to provide a clinical solution to these patients as most referrals come from the GPs themselves, citing failure of all non-operative strategies. This only promotes the repeated use of non-operative resources that have already proven to be ineffective (and therefore have negligible cost-utility) or the acceptance of persistent disability with significant impact on patients HRQoL.

Further development of GP referral pathways is similarly unlikely to significantly address this unmet need as inappropriate referrals were seldom noted. The referrers knowledge of the available non-operative treatments was of a high standard and the relevant clinical question asked were centred on diagnosis, prognosis and the suitability of surgery for these conditions, questions best addressed by specialists.

The public are currently unaware of the degree of rationing occurring and are thus not fully informed when they consider personal decisions about their future health. It is beyond the scope of this paper to determine the optimal way of improving access to specialist's spinal services. However, these options will not be explored until the current reality is recognised.

One criticism of the triaging system utilised during this study period was a lack of a prioritisation tool. However, the degree of pathology and limited number of resources meant that only severe spinal stenosis could be offered treatment. One guide to the effectiveness of our triaging process was that 85% of patients who were given an FSA were deemed appropriate surgical candidates. Also, the focus of this study remains the unmet need—those that missed out. While a different method of triaging may have selected a different cohort of patients for a FSA it would not have changed the proportion missing out on specialist assessment altogether. One

further concern is that occasionally patients with serious pathology, such as tumours, can present with relatively benign clinical features that may not reach the threshold for a FSA under the current conditions.

Subsequent to this study period, a prioritisation tool has been rolled out across New Zealand to help stratify patients already considered to benefit from orthopaedic surgery. This is expected to provide some quantifiable measure to assist with resource allocation of surgical services nationwide and within DHBs. However this tool has not been independently validated or its reliability tested. It also does not provide a framework for prioritising referrals. Those

patients unable to access a FSA remain unrecognised and unquantified. It is our hope that this study will prompt further analysis of the health consequences of this forgotten cohort.

Conclusion

This study quantifies the unmet need for both spinal orthopaedic specialist assessment and, if warranted, surgical management of spine conditions within the Canterbury public health system. It highlights the degree of rationing within the public health system and reveals the impact of the introduction of maximum waiting times for surgery within the CDHB.

Author information:
Tom Inglis, Orthopaedic Trainee, Orthopaedic Department, Christchurch and Burwood Hospitals, Canterbury District Health Board, Christchurch; Rowan Schouten, Consultant Orthopaedic Surgeon, Orthopaedic Department, Christchurch and Burwood Hospitals, Canterbury District Health Board, Christchurch; Kristian Dalzell, Consultant Orthopaedic Surgeon, Orthopaedic Department, Christchurch and Burwood Hospitals, Canterbury District Health Board, Christchurch; Jeremy Evison, Consultant Orthopaedic Surgeon, Orthopaedic Department, Christchurch and Burwood Hospitals, Canterbury District Health Board, Christchurch; Grahame Inglis, Consultant Orthopaedic Surgeon, Orthopaedic Department, Christchurch and Burwood Hospitals, Canterbury District Health Board, Christchurch.
Corresponding author:
Tom Inglis, Orthopaedic Department, Christchurch Hospital, 2 Riccarton Avenue, Christchurch Central, Christchurch.
inglistom@gmail.com
URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7013

REFERENCES:

1. Our Region [Internet] http://www.cdhb.health.nz/About-CDHB/Pages/Our-Region.aspx, Canterbury District Health Board. Published: Jun 2013, Last updates: May 2014

2. Amundsen T, Weber H, Lilleas F, et al. Lumbar Spinal Stenosis: Clinical and Radiologic Features. Spine. 1995; 20(10): 1178–1186

3. North American Spine Society. Evidence-Based Guidelines for Multidisciplinary Spine Care: Diagnosis and Treatment of Degenerative Lumbar Spinal Stenosis. Revised 2011 https://www.spine.org/Portals/0/Documents/Research-ClinicalCare/Guidelines/LumbarStenosis.pdf

4. Weinstein JN, Tosteson TD, Lurie JD. Surgical versus Nonsurgical Therapy for Lumbar Spinal Stenosis. N Engl J Med. 2008; 358(8): 794–810

5. Deyo RA, Ciol MA, Cherkin DC, et al. Lumbar spinal fusion: a cohort study of complications, reoperations and resource use in the Medicare population. Spine. 1993; 18(11):1463–1470.

6. Rihn JA, Hilibrand AS, Zhao W et al. Effectiveness of surgery for lumbar stenosis and degenerative spondylolisthesis in the octogenarian population: analysis of the Spine Patient Outcomes

Research Trial (SPORT) data. J Bone Joint Surg Am. 2015; 97(3):177–85

7. Government strategy paper. Reduced waiting times for public hospital elective services. March 2000 and Addendum August 2012

8. Battié MC, Jones CA, Schopflocher DP, Hu RW. Health-related quality of life and comorbidities associated with lumbar spinal stenosis. Spine J. 2012; 12(3): 189—195

9. Weinstein JN, Lurie JD, Tosteson TD, et al. Surgical compared with non-operative treatment for lumbar degenerative spondylolisthesis: Four-Year results in the spine patient outcomes research trial (SPORT) randomized and observational cohorts. JBJS (Am). 2009; 91(6):1295–304

10. Amundsen T, Weber H, Nordal HJ, et al. Lumbar spinal stenosis: conservative or surgical management? A prospective 10-year study. Spine. 2000; 25(11): 1424–143

11. Malmivaara A, Slatis P, Heliovaara M, et al. Surgical or non-operative treatment for lumbar spinal stenosis. Spine. 2007; 32(1): 1–8

12. Fernandes A, Sundararajan K, Rampersaud R. Changes in self-reported clinical status and health care utilization during wait time for surgical spine consultation: a prospective observational study. Can J Surg. 2014 Jun; 57(3 Suppl 2): S18–S46

13. Rampersaud YR, Lewis SJ, Davey JR, et al. Comparative outcomes and cost-utility after surgical treatment of focal lumbar spinal stenosis compared with osteoarthritis of the hip or knee – part 1: long-term change in health related quality of life. Spine J. 2014 Feb; 14(2): 234–243

14. Rampersaud YR, Tso P, Walker KR, et al. Comparative outcomes and cost-utility following surgical treatment of focal lumbar spinal stenosis compared with osteoarthritis of the hip or knee: part 2 – estimated lifetime incremental cost-utility ratios. Spine J. 2014 Feb; 14(2): 244–254

15. Hansson T, Hansson E, Malchau H. Utility of spine surgery: a comparison of common elective orthopaedic surgical procedures. Spine. 2008; 33(25): 2819–2830

16. Mokhtar SA, McCombe PF, Williamson OD, et al. Health-related quality of life: a comparison of outcomes after lumbar fusion for degenerative spondylolisthesis with large joint replacement surgery and population norms. Spine J. 2010; 10(4): 306–312

17. Quality Dimensions of the Elective Waiting Time Goals – High Level Review, May 2013

Rebalancing health service use for older people: simulating policy-relevant scenarios under demographic ageing

Roy Lay-Yee, Janet Pearson, Peter Davis, Martin von Randow, Ngaire Kerse, Laurie Brown

ABSTRACT

AIMS: The demographic ageing of New Zealand society has greatly increased the proportion of older people (aged 65 years and over), with major policy implications. We tested the effects on health service use of alterations to morbidity profile and the balance of care.

METHODS: We developed a microsimulation model using data from an official national health survey series to generate a synthetic replicate for scenario testing.

RESULTS: Projections on current settings from 2001 to 2021 showed increases in morbidity—long-term illness (2%)—and in health service use—doctor visits (21%), public hospital admissions (16%). Scenarios with decreasing morbidity levels showed moderate reductions in health service use. By contrast, rebalancing towards the use of practice nurses showed a large decrease in public hospital admissions for people aged 85 years and over.

CONCLUSION: Demographic ageing may not have a major negative effect on system resources in New Zealand and other developed countries. Rebalancing between modalities of care may soften the impact of increasing health service use required by a larger older population.

The demographic structure of New Zealand, as in other developed countries, is changing. The proportion of older people in the population has greatly increased—along with their experience of multi-morbidity—with major implications for the provision of health services.[1,2] Forecasts of future compression or expansion of morbidity hinge on whether extended life-expectancy will be spent largely in good or ill health.[3] Nevertheless, there is pressure on available resources to keep pace with the sheer increase in volume of health care required for larger numbers of older people. The recent World Health Organization's 'World Report on Ageing and Health' proposes a public-health framework for healthy ageing—defined as "the process of developing and maintaining the functional ability that enables well-being in older age"—in which the first of four priority areas is "aligning health systems to the needs of older populations" (p 13).[4] The policy quandaries posed by demographic ageing apply no less to New Zealand,[5] with the proportion of people aged 65 years and over projected to increase by nearly two-fifths from 12.1% in 2001 to 16.8% in 2021.[6]

Aims

We aimed to model a range of policy scenarios on the future shape of the New Zealand health-care system under conditions of demographic ageing. To do this, we constructed and applied a discrete-time dynamic microsimulation model to health service use in older people. Here, we define health services as a balance of three modalities: practice nurse visit, family doctor visit and public hospital admission. We report on the construction of the model and the results of projections and scenario testing.

Research questions

After establishing a baseline for our model, we aimed to address two key policy initiatives proffered internationally: promoting healthier ageing to reduce the need for health care[7] and changing the balance of care[8] towards more effective configurations.[9] We focussed on testing scenarios where the burden on the health system might be lessened. Our research questions can be formalised as follows:

1. What will be future levels of health service use for older people under the status quo? This is our 'base projection'.

2. What is the impact of reducing morbidity levels—proxy for healthier ageing (and the compression of morbidity)—on health service use of older people? This is our 'reduced morbidity' scenario.

3. What is the impact of changing the balance among providers on health service use of older people? This is our 'balance of care' scenario.

The model (Figure 1) was: (1) hierarchically structured—with long-term illness (morbidity) driving health service use, with practice nurse use affecting family doctor use (via potential prevention or substitution) and with practice nurse and family doctor use affecting public hospital admission (via potential prevention or delay)—and (2) dynamic—incorporating demographic and morbidity changes over time.[10]

Microsimulation

Microsimulation—first proposed by Orcutt[11] in 1957—has been used, for example, to assess the impact of demographic aging on population health.[12] Microsimulation relies on data from the real world to create an artificial version like the original. It operates at the level of individual units (here older people), each assigned attributes as a starting point—eg age and health state, to which quantitative rules (eg statistical equations) are applied to simulate changes in state or behaviour. Thus a synthetic set of typical life histories can be generated. The model can then be used to test scenarios—essentially thought experiments—by modifying key factors and assessing impact on outcomes of policy interest.[13,14]

Figure 1: Conceptual model of late-life ageing and health care trajectory.
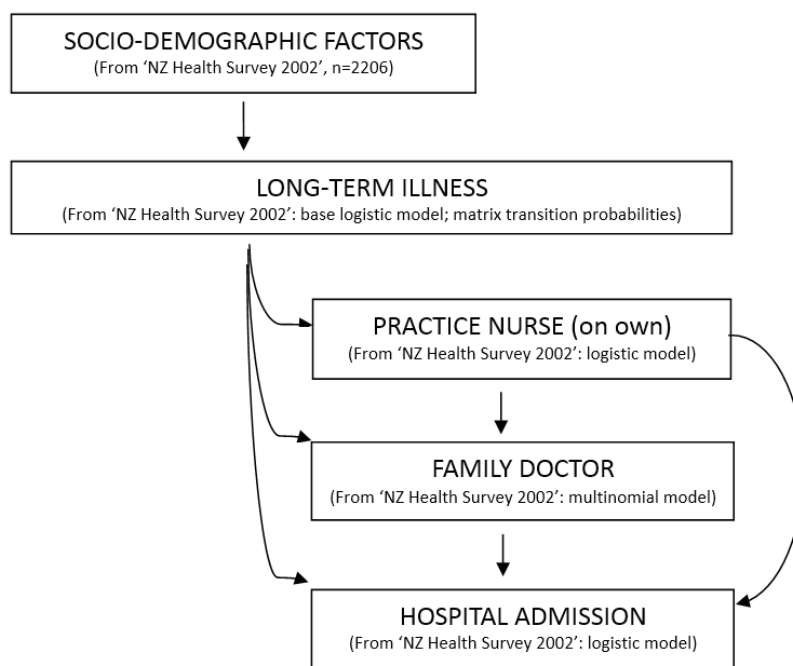
Long-term illness drives health service use, with practice nurse use affecting family doctor use and public hospital admission, and with family doctor use affecting public hospital admission.

Methods

Our methods are outlined briefly in this section with a detailed report published online.[15] Microsimulation was adopted as a technical approach well-suited to modelling the dynamics of a complex system such as health care, and for testing policy scenarios related to utilisation.

Data sources

We used individual-level data on older people aged 65 years and over from the New Zealand Health Survey undertaken in 2002 and 2006 (NZHS 2002 and 2006).[16] As well as the person's demographic characteristics, there was information on whether they had long-term illness, and on their use of health care; NZHS was the only national data source available with these features. These survey data had the advantage of being nationally representative and relatively recent with adequate sample sizes. NZHS sample weights were taken into account in analyses and simulations. The NZHS 2002 contributed data on 2,206 individuals to form a starting sample at the base year, set to 2001, providing initial conditions representative of older people living in the community. A description of characteristics of the starting sample can be found in Table 1. Thus 9.3% were aged 85 years and over while 85.6% were experiencing long-term illness.

Table 1: Description of starting sample. Characteristics of older people (aged 65+ years) living in the community, 2001.

Characteristic	Percentage of weighted sample† (n=2206)
Age group	
65–74	54.2
75–84	36.5
85+	9.3
Gender	
Female	55.3
Ethnicity	
European	91.8
Māori (the indigenous people)	4.0
Pacific	1.7
Asian	2.2
Other	0.3
Marital status	
Partnered	56.5
Deprivation decile	
1 (low deprivation)	6.5
2	7.6
3	9.3
4	10.4
5	10.1
6	13.5
7	10.6
8	13.8
9	11.1
10 (high deprivation)	7.3
Long-term illness	
Present	85.6

† The starting sample was taken from the New Zealand Health Survey 2002/3 (Ministry of Health 2004), with weighting calibrated to the New Zealand Census 2001.

Definition of variables

The following individual characteristics incorporated in the model can be categorised into three types:

1. Socio-demographic
- Age: 65+ years.
- Gender: male, female.
- Self-reported ethnicity (in prioritised sequence): Māori, Pacific, Other, European. A single ethnicity variable was constructed to account for individuals who reported multiple ethnic affiliations.[17]
- Socio-economic deprivation: 'NZDep' (decile)—a census-based small-area measure.[18]
- Self-reported partnership: married, or partnered and not legally married (yes/no).

2. Morbidity
- Self-reported long-term illness (yes/no): any medical condition lasting six months or more.

3. Health service use (outcomes)
- Self-reported health service use (in the last 12 months): any practice nurse visit—formal consultation with nurse on their own, ie without seeing a doctor (yes/no); family doctor visit categories 0, 1–2, 3–4, 5–6, or 7+ visits (yes/no, in each category); public hospital admission for any reason, comprising inpatient and day patient (yes/no). For family doctor visits, simulated results are reported for the combined '5+ visits' category signifying a high user group.

Analysis

Firstly, transition probabilities for long-term illness were estimated from matrices using repeated cross-sectional data (NZHS 2002 and 2006), depending on age and gender (use of other characteristics was constrained by small numbers). This estimation was based on known long-term illness levels in 2002 and 2006, and assumed that an individual could remain in the same state or progress to the next state but not revert to a former state. These results imparted dynamic change to the cross-sectional models of health service use (as below).

Secondly, we used cross-sectional data (NZHS 2002) to predict health service use from long-term illness (as above) in a series of regression models: practice nurse visit—logistic, family doctor visit (as categories)—multinomial and public hospital admission—logistic. Earlier events or states could exert an influence over later ones (Figure 1). Thus, practice nurse visit was a function of long-term illness; while family doctor visit was a function of both long-term illness and practice nurse visit; and finally public hospital admission was a function of long-term illness, practice nurse visit and family doctor visit. Age, gender, ethnicity, deprivation level and partnership status were also accounted for as potential socio-demographic control variables while, for each model, only statistically significant ones were retained.

Simulation

From 2001, we applied parameters (derived from statistical analysis of NZHS data) to update time-variant attributes of 2,206 individuals (in the starting sample) at five-year intervals using Monte Carlo simulation. The simulation process for each subsequent time interval followed a sequence of steps from demographic characteristics, through health status, to final health care outcomes. To reduce the effect of random error, a simulated estimate was taken as the average result of 20 runs, sufficient to generate a stable value. Thus a set of typical though varied individual life histories was created. To maintain a representative sample over time, at each five-yearly interval, we allowed individuals to enter (being randomly drawn from 65–69 year-olds from NZHS 2002) and to die (according to probabilities from official period life tables), as well as re-weighting (according to official population statistics) to account for demographic changes in composition (eg due to migration).

Validation

Validation of simulated results was carried out by comparison to actual NZHS 2006 data (the latest available). The test was whether the simulation model could reproduce benchmark averages and distributions. Where necessary and possible, simulated results were calibrated to population parameters (from NZHS, censuses and official

projections) so that findings could be generalised to the national population.

Scenario testing

Key factors influencing health service use may be considered as potential levers for policy intervention. These can be tested via simulating scenarios. We used the simulated results—with no changes made—as the base case. For each scenario, we changed factors of interest in the starting sample, while holding other initial factors constant, and observed impact on downstream outcomes (compared to the base case). At an individual level, changes were made to those in or at high risk of being in a particular state, eg having long-term illness. Note that the settings for the scenarios were heuristic: we started with small changes in morbidity or care levels and gradually increased or decreased them, over a reasonable range of proportions to an upper limit of possibility (5 to 20 percent).

1. *Base projection* of status quo to 2021

We simulated from the starting sample in 2001 forward to 2021 with *no changes* to inputs or parameters. We considered 20 years as a reasonable projection period that would be useful without over-stretching the data.

2. *'Reduced morbidity' scenario* (2021)

We artificially *reduced*, by varying proportions (5%, 10% and 20% respectively), the prevalence of and transition probabilities for long-term illness to assess impact on levels of health service use.

3. *'Balance of care'* scenario (2021)

We artificially *increased*, by varying proportions (5%, 10% and 20% respectively), the level of practice nurse visits to assess impact, in turn, on levels of family doctor visits and public hospital admissions.

Results

Validation

The simulated sample followed the general pattern for the real sample (from NZHS 2006) though not uniformly so across all measures: compared to the benchmarks, long-term illness and practice nurse visit were under-estimated while family doctor visit and public hospital admission were over-estimated (Table 2). Note that, in the interpretation of simulated results, greater importance should be placed on direction and magnitude rather than specific point estimates.

Table 2: Morbidity and health service use for older people (aged 65+ years) living in the community. Comparing simulated to real data, 2006.

Age group	Morbidity		Health care modalities					
	Long-term illness (lasting at least six months) (%)		Practice nurse visit (any in last 12 months) (%)		Family doctor 5+ visits (in last 12 months) (%)		Public hospital admission (any in last 12 months) (%)	
	Simulated	Real [†]	Simulated	Real [†]	Simulated	Real [†]	Simulated	Real [†]
65–69	78.0	86.7	42.9	43.6	36.6	31.1	18.7	14.1
70–74	89.2	89.7	43.2	46.2	38.8	32.9	20.7	17.5
75–79	89.8	89.6	43.7	45.8	52.0	36.8	25.5	19.7
80–84	93.8	94.0	44.2	48.4	52.3	42.2	26.5	25.2
85+	91.1	89.9	44.6	45.2	47.9	39.7	22.5	19.1
All (65+)	86.6	89.3	43.5	45.5	43.6	35.0	22.0	18.1
(95% CI)[‡]	(86.3–87.2)		(42.6–44.4)		(42.6–45.3)		(21.5–23.1)	

† Taken from NZ Health Survey 2006.

‡ 95% confidence intervals were calculated from 20 simulation runs.

Scenario testing

Our comparison between the base simulation (with no changes) and a scenario (with a factor change) were relative to one another within the virtual world. The two simulated results—conditioned on the same input data and parameters—are directly comparable and give a good assessment of impact of the changed factor.

1. *Base projection*

Simulation under current settings, ie projection, from 2001 to 2021 showed a moderate absolute increase overall in the level of long-term illness (Table 3) which was more marked with increasing age: 2% for the 65+ age group and 13% for the 85+ age group (results not tabled). There was a concomitant proportional increase in the use—by people aged 65+—of family doctor visits (up 21%) and public hospital admissions (up 16%) while practice nurse visits remained stable (Table 3).

Table 3: Base projection and 'reduced morbidity' scenarios. Morbidity and health service use for older people (aged 65+ years) living in the community, 2001 and 2021.

Simulations†	Morbidity	Health care modalities		
	Long-term illness (lasting at least six months) (%)	Practice nurse visit (any in last 12 months) (%)	Family doctor 5+ visits (in last 12 months) (%)	Public hospital admission (any in last 12 months) (%)
Q1. Base projection‡				
2001	85.6	42.1	36.0	18.8
2006	86.6	43.5	43.6	22.0
2011	87.2	43.3	43.4	22.1
2016	86.5	43.2	42.9	21.6
2021	87.4	43.3	43.5	21.8
Q2. 'Reduced morbidity' scenarios§				
Make 5% decrease in long-term illness		(%) [% change]¶		
2006	-	43.0	42.7	20.8
2011	-	43.1	42.9	21.1
2016	-	43.0	42.4	21.3
2021	-	43.2 [-0.2]	42.9 [-1.4]	21.4 [-1.8]
Make 10% decrease in long-term illness				
2006	-	42.6	41.8	19.7
2011	-	43.0	42.1	20.5
2016	-	43.1	41.6	20.7
2021	-	43.1 [-0.5]	42.5 [-2.3]	21.1 [-3.2]
Make 20% decrease in long-term illness				
2006	-	42.6	40.2	18.8
2011	-	42.7	41.0	19.4
2016	-	43.0	40.4	19.6
2021	-	43.1 [-0.5]	41.1 [-5.5]	20.0 [-8.3]

† Simulations are calibrated to NZ Health Survey 2006 data.
‡ Base projection to 2021 is on current settings.
§ Scenarios represent the impact of reducing base prevalence of and transition probabilities for morbidity (long-term illness) by nominated percentage of base projected level.
¶ Proportional change in outcome (due to the scenario settings) compared to the base projection for that year.

2. *'Reduced morbidity'* scenario

Scenarios projected to 2021, implemented by progressively decreasing long-term illness levels, had the effect of only moderately reducing health service use compared to the base projection (Table 3). For example, with long-term illness levels reduced by 20%, there were proportional reductions of 0.5% in practice nurse visits, 5.3% in family doctor visits and 8.3% in public hospital admissions.

3. *'Balance of care'* scenario

Scenarios projected to 2021, implemented by progressively rebalancing towards practice nurse use, had the effect of moderately decreasing family doctor visits but markedly decreasing public hospital admissions—compared to the base projection (Table 4). This effect was much more pronounced with increasing age. This is illustrated by the scenario where the proportion of older persons who visited the practice nurse at least once in a year was increased by 20%. Thus, in the 65+ age group, relative to the basic projection, the proportion of high users of family doctors visits was reduced by 0.7% and the proportion of people admitted to public hospital was reduced by 1.4%; in the 85+ age group those relative reductions were 0.8% and 25.5% respectively (Table 4).

Table 4: 'Balance of care' scenarios. Towards more practice nurse visits for older people living in the community, 2021.

Simulations†	Health care modalities					
	Practice nurse visit (any in last 12 months) (%)		Family doctor 5+ visits (in last 12 months) (%)		Public hospital admission (any in last 12 months) (%)	
	Aged 65+	Aged 85+	Aged 65+	Aged 85+	Aged 65+	Aged 85+
Q1. Base projection‡						
2001	42.1	26.6	36.0	36.2	18.8	22.7
2006	43.5	44.6	43.6	47.9	22.0	22.5
2011	43.3	43.8	43.4	48.9	22.1	22.8
2016	43.2	42.4	42.9	48.7	21.6	22.1
2021	43.3	42.4	43.5	48.8	21.8	23.1
Q3. 'Balance of care' scenarios§						
Make 5% increase in practice nurse visits			(%) [% change]¶			
2006	-	-	43.4	47.9	21.9	22.2
2011	-	-	43.5	48.9	21.9	22.8
2016	-	-	42.9	47.6	21.7	22.3
2021	-	-	43.5 [0]	50.5 [+3.5]	21.9 [+0.5]	22.4 [-3.0]
Make 10% increase in practice nurse visits						
2006	-	-	43.3	47.9	21.9	21.8
2011	-	-	43.5	48.9	21.8	22.5
2016	-	-	42.9	47.5	21.7	21.6
2021	-	-	43.4 [-0.2]	50.4 [+3.3]	21.9 [+0.5]	21.9 [-5.2]
Make 20% increase in practice nurse visits						
2006	-	-	43.3	47.5	21.8	21.0
2011	-	-	43.2	47.7	21.4	18.5
2016	-	-	42.6	47.2	21.7	20.3
2021	-	-	43.2 [-0.7]	48.4 [-0.8]	21.5 [-1.4]	17.2 [-25.5]

† Simulations are calibrated to NZ Health Survey 2006 data.
‡ Base projection to 2021 is on current settings.
§ Scenarios represent the impact of increasing the level of use of practice nurses by nominated percentage of base projected level, culminating in all individuals visiting the practice nurse at least once.
¶ Proportional change in outcome (due to the scenario settings) compared to the base projection for that year.

Discussion

The New Zealand health care system, in resource terms, is driven by a complex mix of demand and supply elements, one of which is demographic ageing.[5] We developed and tested a microsimulation model of health service use in older age that may be useful for policy decision-making. The principal findings and their implications will be discussed in the context of each of the research questions posed earlier in the paper. From the results of our simulations, we show *what would happen if* there were policy interventions in place that could increase or decrease current settings in morbidity or health care levels. Discussion of *actual* policy initiatives, their feasibility or effectiveness is beyond the scope of this paper; there are other system improvements that could be made.

Answering research question 1: Base projection

The base projection under current settings from 2001 to 2021 showed a moderate increase—more marked with age—in long-term illness and in health service use for older people (aged 65+). This assumed that while older people were living longer, they were suffering the same historical pattern of illness. The findings point to the moderate future expansion of morbidity in New Zealand.[19] Furthermore, they indicate that pure demographic ageing—the morbidity impact of a greater number of older people—may not have a major negative effect on health care resources.[20,21] This is consistent with other studies showing, for example, time to death rather than age having the greater impact on health service volumes.[22]

Answering research question 2: 'Reduced morbidity' scenario

Scenarios in which long-term illness level was decreased—a proxy for healthier ageing and the compression of morbidity—had the effect of moderately reducing health service use. Our findings indicate the limited effect of policy intervention on levels of health service use that are based solely on promoting healthier ageing (while not forgetting its general benefits). The morbidity impact of demographic ageing is complicated by other factors such as: rising living standards and consumer expectations on the demand side and developments in medical technology on the supply side[23] as well as evidence that obesity may give rise to more chronic illness in the future elderly.[24]

Answering research question 3: 'Balance of care' scenario

Scenarios in which health care was rebalanced towards the use of practice nurses had the effect of reducing family doctor visits and public hospital admissions, being more pronounced in the 85+ age group. Shifting to a modality where substitution is practicable may be arguably more effective with improved patient outcomes. We refer not to directly transferring services for *existing* patients from, for example, hospitals to primary care settings,[25] but to provision of appropriate services geared to *arising* patient need.[26] Thus, for example, visiting a practice nurse for a non-urgent reason such as for advice, screening, monitoring or maintenance activities may avert, prevent or delay the need to see a family doctor or for hospitalisation; and similarly a timely visit to the family doctor may avoid hospitalisation. Depending on life stage, providing appropriate health services may help to prevent the development of chronic illness, slow the decline in functional ability, or manage chronic illness when it occurs.[4] In short, changing the balance of care may make better use of limited system resources[10,27] while sustaining the health of older people.

In New Zealand, the family doctor has traditionally provided the majority of primary prevention and treatment services. In recent times, the role of the practice nurse[28] has become more professionalised with wider scope of responsibility in the primary care team. The practice nurse may be better placed to provide particular services for older people, notably related to care for chronic conditions. As an example, drawing on our findings, a possible policy intervention might be increasing the proportion who visit a practice nurse (at least once in a 12-month period) for the oldest old (people aged 85+) which could substantially reduce the proportions needing to be admitted to public hospital (with potential to generate cost savings).

Strengths and limitations

The microsimulation approach employed here has many combined advantages: it has an empirical basis; multiple processes are modelled together and contextualised within a system; and pathways are modelled that may be amenable to policy influence. However, it relies heavily on the availability, quality and compatibility of data. In our case, official data sources were particularly advantageous as results from modelling could be generalised to the future population. Data limitations in our case were: a small starting sample; excluding the institutionalised; self-reported information on use of care (not need nor supply); restricted definitions of both long-term illness (rather than disease-specific) and health service use (only the core trio of modalities); narrow range of explanatory variables; no longitudinal data to derive transition probabilities for long-term illness (matching only by age and gender); no accounting for amenable conditions or multi-morbidity; and no costing information.

However, the data used were the most recent and suitable, available at the time modelling was undertaken; time has passed but the underlying dynamic processes are likely still the same particularly over the period of study. The model was able to approximate benchmark data and parameter settings, and substantive results from scenario testing have been plausible and interpretable. In testing a scenario by manipulating a factor of interest, we assumed that other initial conditions and relationships between factors remained the same. Scenario testing generally showed modest impact on outcomes with a degree of stability in the model perhaps due to the interplay among both promoting and inhibiting factors. This may be an accurate reflection of a reality that is complex. Our model is a simplification—with its specific assumptions and modelling choices, and somewhat gross scenarios—but it may be considered as indicative, stimulating further research, and fitting alongside other evidence for policy.

In policy terms, the model has not been able to take into account health reforms in New Zealand since 2001. For example, there have been initiatives to increase access to primary care[29]—which may increase utilisation—and to develop integrated care (across both health and social components)[30]—which may reduce overall health service use.

Conclusions

We constructed a microsimulation model of older age using official data and applied it to a substantive health policy area. The model serves as a starting point with the potential to be improved and extended. Findings suggest that the system is robust to change. Adding to the international debate, our model indicates that demographic ageing may not have a major negative effect on system resources in developed countries. Furthermore, the sheer volume of health services required, with larger numbers of older people, may be alleviated not only by healthier ageing but also by rebalancing health care to make better use of limited resources.

Author information:
Roy Lay-Yee, Senior Research Fellow, Centre of Methods and Policy Application in the Social Sciences (COMPASS), University of Auckland, Auckland; Janet Pearson, Statistician, Centre of Methods and Policy Application in the Social Sciences (COMPASS), University of Auckland, Auckland; Martin von Randow, Analyst, Centre of Methods and Policy Application in the Social Sciences (COMPASS), University of Auckland, Auckland; Ngaire Kerse, Professor, School of Population Health, University of Auckland, Auckland; Laurie Brown, Professor, National Centre for Social and Economic Modelling (NATSEM), University of Canberra, Australia.
Corresponding author:
Roy Lay-Yee, Senior Research Fellow, Centre of Methods and Policy Application in the Social Sciences (COMPASS), University of Auckland, Auckland.
r.layyee@auckland.ac.nz
URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7014

REFERENCES:

1. Lloyd-Sherlock P, McKee M, Ebrahim S, et al. Population aging and health. Lancet. 2012;379(9823):1295–6.

2. Rechel B, Doyle Y, Grundy E, McKee M. How can health systems respond to population aging? Copenhagen: World Health Organisation; 2009.

3. Crimmins EM, Beltrán-Sánchez H. Mortality and morbidity trends: Is there compression of morbidity? J Gerontology B Psychol Sci Soc Sci. 2011; 66B(1):75–86.

4. World Health Organization. World report on aging and health. Summary. Geneva: World Health Organization; 2015.

5. Cumming J, McDonald J, Barr C, et al. New Zealand health system review. In: Gericke C, editor. Health Systems in Transition. Vol. 4, No. 2. Manila: World Health Organisation; 2014.

6. Statistics New Zealand. NZ.Stat DataHub table viewer; 2015. Retrieved from http://nzdotstat.stats.govt.nz/wbos/

7. Oxley H. Policies for healthy aging: An overview. Health Working Paper No. 42. Paris: Organisation for Economic Co-operation and Development; 2009.

8. Tucker S, Brand C, Wilberforce M, Challis D. The balance of care approach to health and social care planning: Lessons from a systematic literature review. Health Serv Manage Res. 2013;26(1):18–28.

9. Wanless D, Forder J, Fernandez JL, et al. Securing good care for older people: Taking a long-term view. London: King's Fund; 2006.

10. Layte R, Barry M, Bennett K, et al. Projecting the impact of demographic change on demand for and delivery of health care in Ireland. Research series, number 13. Dublin: The Economic and Social Research Institute; 2009.

11. Orcutt G. A new type of socio-economic system. Review of Economics and Statistics. 1957;39(2):116–23.

12. Gupta A, Harding A. Modelling our future: Population aging, health and aged care. Amsterdam: Elsevier; 2007.

13. Davis P, Lay-Yee R, Pearson J. Using microsimulation to create a synthesized data set and test policy options: The case of health service effects under demographic aging. Health Policy. 2010;97:267–74.

14. Zucchelli E, Jones AM, Rice N. The evaluation of health policies through dynamic microsimulation methods. International Journal of Microsimulation. 2012;5(1):2–20.

15. Lay-Yee R, Pearson J, von Randow M. Balance of care in an ageing society: Construction of the BCASO microsimulation model. Technical Report. Auckland: COMPASS Research Centre, University of Auckland; 2015. ISBN 978-0-473-32639-2 (online). Retrieved from URL: https://cdn.auckland.ac.nz/assets/arts/research-centres/compass/documents/bcaso-technical-report.pdf

16. Ministry of Health. A portrait of health: Key results of the 2002/03 New Zealand Health Survey. Wellington: Ministry of Health; 2004.

17. Lang K. Measuring ethnicity in the New Zealand population census. Wellington: Statistics New Zealand; 2002.

18. Salmond C, Crampton P. Development of New Zealand's Deprivation Index (NZDep) and its uptake as a national policy tool. Can J Public Health. 2012;103(Suppl. 2):S7–S11.

19. Graham P, Blakely T, Davis P, et al. Compression, expansion or dynamic equilibrium? The evolution of health expectancy in New Zealand. J Epidemiol Community Health. 2004;58:659–66.

20. Appleby J. Spending on health and social care over the next 50 years: Why think long term? London: The King's Fund; 2013.

21. Spijker J, MacInnes J. Population aging: The timebomb that isn't. BMJ. 2013;347:f6598.

22. Murphy M, Martikainen P. Use of hospital and long-term institutional care services in relation to proximity to death among older people in Finland. Soc Sci Med. 2013;88:39–47.

23. Smith S, Newhouse JP, Freeland MS. Income, insurance, and technology: Why does health spending outpace economic growth? Health Aff. 2009;28(5):1276–84.

24. Lakdawalla DN, Goldman DP, Shang B. The health and cost consequences of obesity among the future elderly. Health Aff. 2005;W5:R30–41.

25. Sibbald B, McDonald R, Roland, M. Shifting care from hospitals to the community: a review of the evidence on quality and efficiency. J Health Serv Res Policy. 2007;12:110–7.

26. SBC Delivery Group. Improving outcomes by shifting the balance of care. Scotland: National Health Service; 2009.

27. Philp I, Mills KA, Thanvi B, et al. Reducing hospital bed use by frail older people: Results from a systematic review of the literature. Int J Integr Care. 2013;13:e048.

28. McKinlay E. New Zealand practice nursing in the third millennium: Key issues in 2006. New Zealand Family Physician. 2006;33(3):162–8.

29. Hefford M, Crampton P, Foley J. Reducing health disparities through primary care reform: The New Zealand experiment. Health Policy. 2005;72:9e23.

30. Cumming J. Integrated care in New Zealand. Int J Integr Care. 2011;11:e138.

Epidemiology of intussusception in New Zealand pre-rotavirus vaccination

Bronwyn Rosie, Stuart R Dalziel, Elizabeth Wilson, Emma J Best

ABSTRACT

AIMS: To describe the epidemiology of intussusception in New Zealand children aged 0–36 months prior to the introduction of routine rotavirus vaccination.

METHODS: ICD-10 coding data from the New Zealand National Minimum Data Set (NMDS) was used to identify all cases of intussusception in children aged 0–36 months between January 1998 and December 2013. These data were linked with birth data from the New Zealand census. Population incidence rates of intussusception were calculated, and demographic characteristics described.

RESULTS: Over the 16-year study period, there were 794 cases of intussusception. The majority (56%) occurred in the first year of life (age adjusted incidence rate 56.1/100,000 child-years, 95% confidence interval (CI) 41.7–71.2). Intussusception occurred more frequently in males (36.4/100,000 (95% CI 24.6–48.2) versus 19.5/100,000 (95% CI 10.8–28.1, p<0.001)). There was no difference in intussusception incidence between ethnic groups, although cases occurred at a younger age in Māori and Pacific infants compared to Asian and other ethnicities (Pacific median 7.5 months (interquartile range 5.9–11.6), Māori 7.8 months (IQR 5.5–12.3), European 9.2 months (IQR 5.8–15.8), Other Ethnicity 10.2 months (IQR 8.2–12.3), Asian 10.5 months (IQR 7.0–17.1)). There was a weak seasonal trend with incidence troughs in January and July, and corresponding peaks in March and September.

There was wide variation in presentation rates across District Health Board (DHB) regions, with a national average of 18.0/100,000 child-years (95% CI 9.7–26.3). Most patients were admitted on a single occasion to a single hospital for treatment (81%).

CONCLUSIONS: This study updates background incidence rates of intussusception prior to the introduction of a national rotavirus vaccination programme in July 2014. It identifies a trend of earlier intussusception in Māori and Pacific infants; the relationship between earlier intussusception and the risk of vaccine-associated events is unknown.

Intussusception occurs when one segment of the intestine invaginates into the adjacent distal segment,[1] with the initiating event subclinical infection and gut adenopathy. Untreated intussusception disrupts the bowel's vascular supply and can cause ischaemia, perforation and ultimately death. Intussusception is a relatively rare event, with a worldwide incidence of 74 per 100,000 (range 9–328) in children aged less than one year of age.[2]

Rotavirus is a significant cause of infant gastroenteritis worldwide both in developed and developing countries. Annual estimates of rotavirus associated deaths in children under five range from 200,000 to 450,000, most of which occur in the developing world.[3] Rotavirus is also a significant cause of morbidity, responsible for 2.4 million hospitalisations worldwide each year.[4] In New Zealand it is estimated that rotavirus is responsible for the hospitalisation of one in 43 children by the age of five.[5]

In 1999 an oral human-rhesus rotavirus quadrivalent vaccine (RotaShield, Wyeth-Lederle) was introduced to the US infant schedule, but withdrawn later that year after reports of an association with intussusception (a risk of approximately one case in 5,000–10,000 vaccinees).[6]

Two second-generation vaccines against rotavirus are available in New

Zealand: RotaTeq (Merck Sharp Dohme), a pentavalent human-bovine reassortment vaccine, containing viral protein types G1–4 and P8; and Rotarix (Glaxo Smith Klein), a human monovalent G1 vaccine. No increased risk of intussusception was detected in the large phase III pre-licensure clinical trials of Rotarix and RotaTeq despite this being a specifically monitored adverse event.[7]

Prior to 2014, only Rotarix was marketed for private sale in New Zealand and covered <10% of the annual birth cohort.[8] From July 2014, New Zealand included RotaTeq in the National Immunisation Schedule (NIS) at six weeks, three months and five months of age.

Introduction of these vaccines has been remarkably successful in both high and middle-income countries. Brazilian and Mexican studies have shown significant declines in diarrhoeal mortality post-vaccine introduction.[9] Studies from high-income countries have identified a 74–90 percent decline in rotavirus gastroenteritis hospitalisations in children under two, and a 29–50 percent decline in 'all-cause' acute gastroenteritis hospitalisations for children under five.[10] Recent studies have demonstrated post-vaccination reductions in hospitalisations for 'all-cause' seizures in children under five, most likely attributable to decreased rotavirus associated febrile illnesses.[11,12]

However, post-licensure studies of both RotaTeq and Rotarix in a number of countries including Australia have demonstrated small increases in intussusception attributable to the vaccines, particularly following the first dose.[13] Recent Australian data estimated the excess risk attributable to rotavirus vaccination to be 5.6 additional cases of intussusception per 100,000 vaccinated infants.[7] It is not clear whether populations with a high background risk of intussusception have a proportionally elevated risk of vaccine associated intussusception.[2]

Given this complex relationship, it is important for countries including New Zealand to monitor intussusception rates before and after establishment of rotavirus vaccination programmes.

A previous New Zealand study[14] found no association between wild type rotavirus gastroenteritis hospitalisations at eight sites and national intussusception rates over a two year period. However, these data are over a decade old. We undertook the current study, incorporating 16 years of data, to determine a contemporary intussusception rate prior to the national introduction of the RotaTeq vaccine to allow monitoring for vaccine-associated change in intussusception rates and to assess for evidence of changing intussusception rates over time.
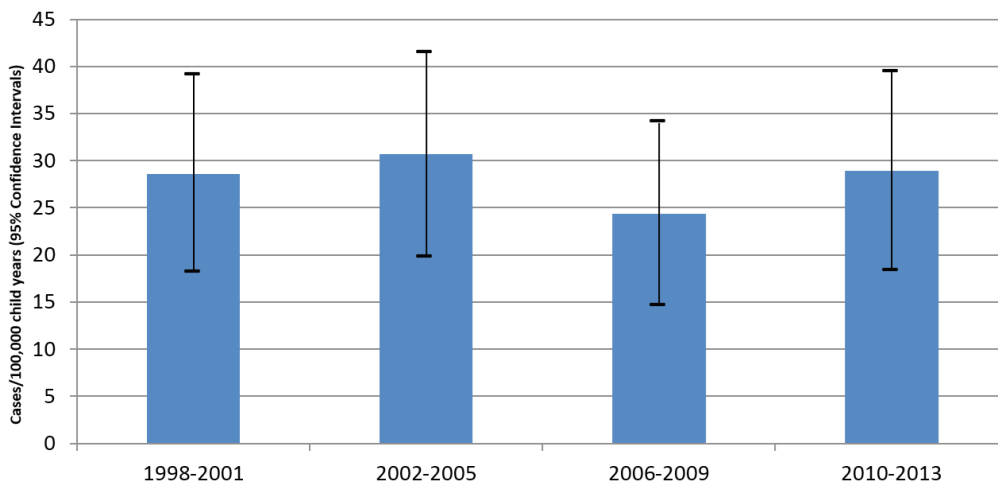
Methods

Data for all public hospitals for the period January 1998 to December 2013, collected in the National Minimum Dataset (NMDS), were reviewed. Intussusception cases in children aged 0–36 months were identified by discharge code (ICD-10 AM code K561 or equivalent ICD-9 code).[15] Additional data on patient birth date, sex, ethnicity, and admitting hospital were also extracted. Within the NMDS, ethnicity is assigned using a standard priority system where Māori, Pacific Islander and Asian ethnicity is assigned preferentially (in the order stated).[16] NHI numbers (national health index number, a unique identifier), were not extracted, ensuring that patients remained anonymous.

Intussusception cases where the patient had the same birth date, sex and ethnicity, and which occurred within a one-week period were counted as a single episode.

Population birth rates by month of birth, sex, ethnicity and DHB region were obtained from Statistics New Zealand.[17] These data were used to calculate the 'at risk' population (children age 0–36 months) for each month of the study period. These population estimates did not take into account deaths, immigration, or emigration. New Zealand census allows individuals to identify themselves in more than one ethnic group, so total population numbers by ethnicity are higher. This slightly decreased ethnicity-specific incidence rates.

Intussusception incidence rates were estimated by combining monthly cases from the NMDS data set with monthly birth rates from Statistics New Zealand. As intussusception is a rare event, confidence intervals were calculated using standard poisson distribution methods. Significance tests were completed using standard nonpara-

Figure 1: Incidence of intussusception by year (New Zealand children 0–36 months, 1998–2013).

metric methods (Kruskal Wallis and Mann Whitney-U) with Bonferroni corrections when comparing multiple groups.

The study protocol was reviewed by the New Zealand Health and Disabilities Ethics Committee who determined ethical approval was not required because of the anonymous nature of the data.

Results

Over the 16-year study period (January 1998–December 2013) 961 episodes were identified with an ICD9 or ICD10 code for intussusception in children aged 0-36 months. One hundred and sixty-seven (17%) of these episodes constituted re-presentations within a week. Thus we identified 794 patients with non-concurrent intussusception events, an average of 50 cases per year (range 39–62).

The incidence of intussusception (cases/100,000 child-years) varied from a low of 20.8/100,000 child-years in 2009, to a high of 37.3/100,000 child-years in 2002 (Figure 1). There was no increasing or decreasing trend over the study period (Kruskal Wallis, p=0.54).

The data was initially analysed in two periods, the first corresponding to the time period of the previous New Zealand study[14] (Jan 1998–Jun 2003) and the second comprising all subsequent data (July 2003–Dec 2013). There was no significant difference in incidence between the two periods and for all subsequent analyses, the data were considered as a single group (Table 1).

Intussusception rates varied significantly by season, with troughs in January and July and corresponding peaks in March and

Table 1: Intussusception rates (cases/100,000 child-years) in New Zealand children 0–36 months, 1998–2013.

Period	Duration	Age Group	Cases	Population	Incidence	Confidence Intervals
Jan 1998–Jun 2003	5.5 years	<1 year	181	307,388	58.9	49.2–80.7
Jan 1998–Jun 2003	5.5 years	1–2 years	53	309,362	17.1	9.9–26.6
Jan 1998–Jun 2003	5.5 years	2–3 years	22	312,194	7.0	2.4–13.4
Jan 1998–Jun 2003	5.5 years	Total	256	928,944	27.6	19.4–41.0
Jul 2003–Dec 2013	9.5 years	<1 year	350	639,226	54.8	37.9–66.2
Jul 2003–Dec 2013	9.5 years	1–2 years	123	650,304	19.3	10.3–27.3
Jul 2003–Dec 2013	9.5 years	2–3 years	65	603,715	10.3	3.7–16.0
Jul 2003–Dec 2013	9.5 years	Total	538	1,893,245	28.2	16.8–37.2
Jan1998–Dec 2013	16 years	<1 year	531	946,614	56.1	41.7–71.2
Jan1998–Dec 2013	16 years	1–2 years	176	959,666	18.6	9.4–25.9
Jan1998–Dec 2013	16 years	2–3 years	87	915,909	9.2	3.3–15.2
Jan1998–Dec 2013	16 years	total	794	2,822,189	28.0	17.5–38.2

Figure 2: The effect of season on intussusception rates (New Zealand children 0–36 months, 1998–2013).
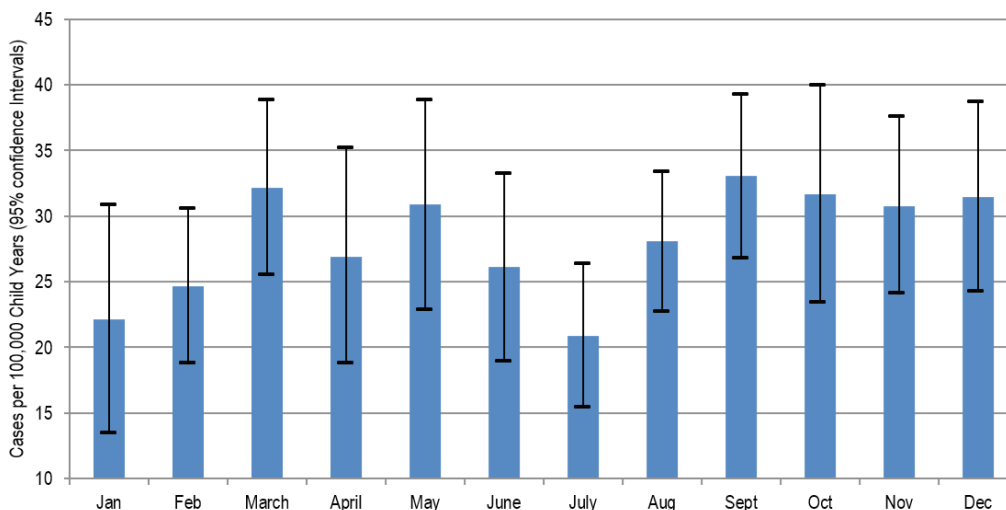
September (22.1/100,000 and 20.8/100,000 versus 32.1/100,000 and 33.0/100,000 child years, Kruskal Wallis, *p*<0.001, Figure 2).

Intussusception was commoner in younger children, with 531 (56%) cases occurring in infants aged 0–12 months. The youngest infant was ten days old. Median age was 8.95 months (IQR 5.80–14.53), with incidence peaking between six–nine months (79.8 cases/100,000 child-years), and falling by 33–36 months of age (<6.0/100,000 child-years; Kruskal Wallis, *p*<0.001, Figure 3).

Intussusception was more common in males (male:female ratio 1.87). Male incidence was 36.4/100,000 child-years, while female incidence was 19.5/100,000 child-years, (Mann Whitney U, p<0.001). There was no significant difference between males and females in the age at which intussusception occurred.

Figure 3: The effect of age on intussusception rates (New Zealand children 0–36 months, 1998–2013).
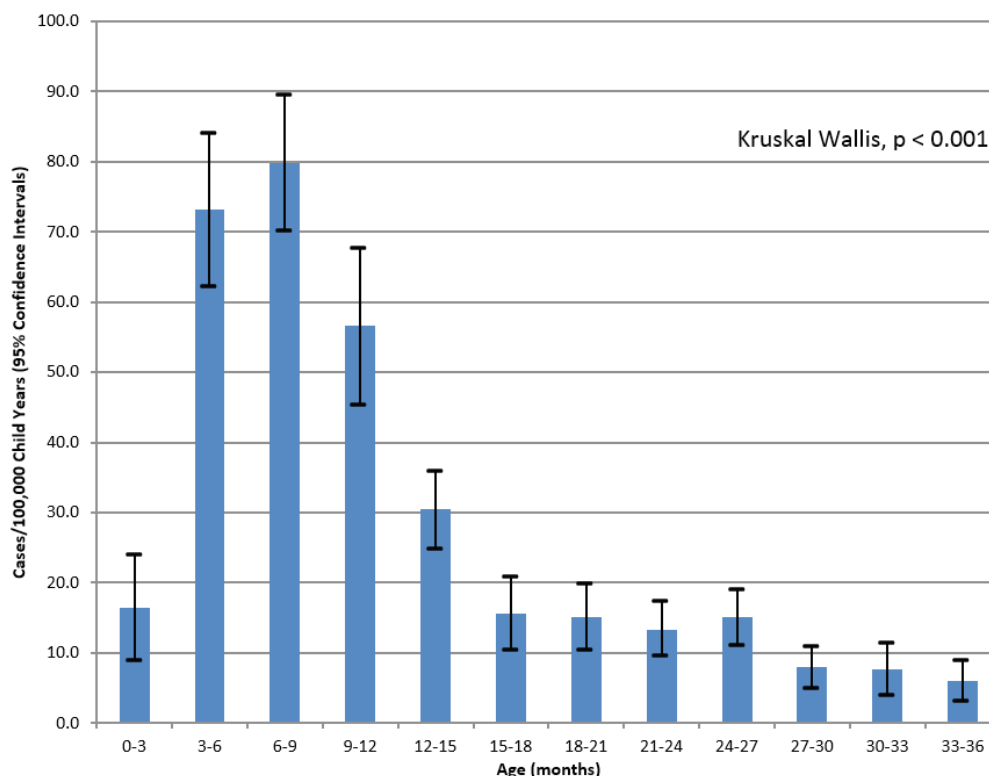
Table 2: The effect of ethnicity on intussusception rates (New Zealand children 0–36 months, 1998–2013).

Ethnicity	Incidence (cases/100,000 child-years)	95% Confidence Intervals
European	24.5	13.6–32.4
Māori	23.0	12.9–31.4
Pacific	22.1	14.8–34.2
Asian	26.7	16.6–36.9

There was no significant difference in intussusception rates between ethnic groups (Table 2).

New Zealand census allows more than one ethnic group, thereby increasing total population numbers and decreasing calculated intussusception rates.

However, the age at which intussusception occurred varied with ethnicity, occurring significantly earlier in Māori and Pacific infants than other ethnicities (Kruskal Wallis, p<0.03, Figure 4).

There was wide variation across District Health Board (DHB) regions, with rates reflecting primary care referral patterns. For example, Auckland DHB performs the majority of the region's paediatric surgery and had a markedly elevated rate of 49.0/100,000 while nearby, Waitemata DHB and Counties Manukau DHB had low rates of 7.1/100,000 and 7.2/100,000 respectively. There was no significant difference in incidence between the North and South Islands.

Most patients were admitted on a single occasion to a single hospital for treatment (81%). However, a significant minority (16.6%) required transfer to another hospital and a few patients (3.7%) required readmission to the same hospital within the same week.

Discussion

Over the last 16 years, the incidence of intussusception in those aged between 0–36 months has remained constant in New Zealand, with an average rate of 28 cases/100,000 child-years. These data will be critical for ongoing monitoring of intussusception rates, following the recent introduction of a rotavirus vaccine into the New Zealand national immunisation schedule.

The current study calculated an incidence rate of 56.1/100,000 years in children <1 year from January 1998 to December 2013. The earlier New Zealand study[14] reported

Figure 4: Age of intussusception by ethnicity (New Zealand children age 0–36 months, 1998–2013).
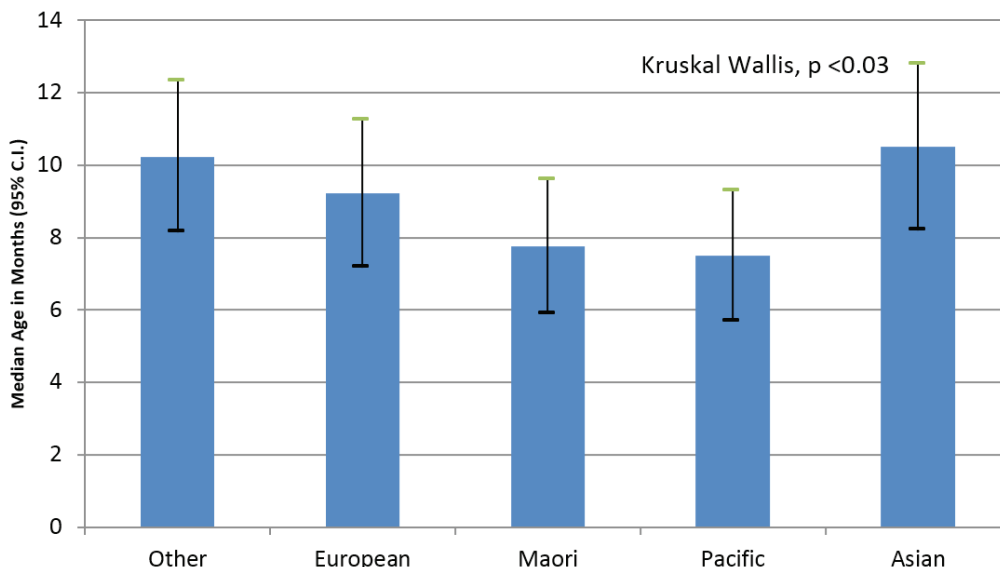
a rate of 65/100,000 child-years and identified 21 more cases over the period January 1998 to June 2006. The extra cases may have resulted from their concomitant use of surgical and radiological databases in addition to NMDS data. Alternatively, their methodology for defining non-concurrent episodes may have varied from ours. Nonetheless, their incidence rate was statistically indistinguishable from ours for the same period (Chi^2=0.64).

As with other international datasets we found intussusception incidence peaked in the first year of life. Our incidence rate of 56.1/100,000 child-years in infants <1 year is in the lower/mid range of international comparisons (Bangladesh 9/100,000 child-years, North America 33/100,000 child-years, England 66/100,000 child-years, Australia 101/100,000 child-years, Vietnam 302/100,000 child-years and South Korea 328/100,000 child-years).[2]

The reasons for this regional variation are poorly understood. Some variation is undoubtedly attributable to differences in data quality and diagnostic criteria. However, other factors such as genetic influences, dietary factors and precipitant infectious diseases (particularly gastroenteritis) have been suggested.[18] Regardless of underlying mechanism, the existence of regional differences emphasises the need for local data to monitor for vaccine-associated adverse events. Our study has clearly established this data for New Zealand.

Infection is hypothesised to precipitate intussuseption; aggregated lymphoid tissue in the gut wall (Peyer's Patches) hypertrophies following infection and may subsequently function as a 'lead point'. Recent episodes of viral[19] or bacterial[20] gastroenteritis have been consistently linked to intussusception. Several studies have found adenovirus at higher rates in the faeces of children with intussusception than in controls.[21,22] Others report a temporal association between recent respiratory tract infection and intussusception.[23] No studies have identified an association between rotavirus gastroenteritis and intussusception,[22,24] including an earlier New Zealand study.[14]

Previous authors have investigated the association between intussusception and the spring and autumn epidemics of viral gastroenteritis typical of temperate countries.[25] In contrast with earlier studies, two recent large literature reviews did not identify seasonal patterns.[2,23] Although contributing studies were separated according to geographic origin, and the data for Oceania showed a winter trough, it did not reach statistical significance.[2] Our study found summer and winter troughs and corresponding spring and autumn peaks.

The absence of strong seasonal patterns argues against rotavirus as a significant precipitant of intussusception[23], although the presence of multiple viral agents may obscure a rotavirus-associated seasonal effect. The relative potency of different viral precipitants remains to be elucidated.[22]

The current New Zealand vaccination schedule recommends completion of rotavirus vaccinations prior to peak age of intussusception occurrence. There is preliminary evidence to support this strategy; a recent Australian study found a weaker association between rotavirus vaccination and intussusception when patients vaccinated after recommended age limits were excluded from their data.[7]

Existing data do not clarify whether vaccination associated intussusceptions represent an overall increase in incidence or an earlier age of onset among those in whom intussusception would have occurred later in infancy, regardless of vaccine exposure.

The possibility that vaccination does not increase cumulative incidence is supported by a large Rotarix pre-licensure trial which found a significantly lower risk of intussusception in recipients of the vaccine compared with recipients of placebo after one year of follow-up (relative risk 0.28; 95%CI, 0.1–0.81).[26] These findings suggest that the short-term increase in intussusception risk after rotavirus vaccination may be offset by a decrease in the longer-term risk of intussusception during the first year of life.

We found that intussusception incidence peaked in infants aged six months, with a median age of 8.95 months. Our data are very similar to the international literature,[2,23] with peak incidence older than the recommended age for completion of rotavirus vaccination.

We found no ethnic difference in intussusception rates. This finding contrasts with the earlier New Zealand study which identified a lower incidence among Māori compared to other ethnic groups.[14] Ethnic differences have been identified in other settings, with lower rates noted in indigenous versus non-indigenous children in Australia,[27] Bedouin Arab versus Jewish children in Israel[28] and white versus black/hispanic children in the US.[29]

We did identify a difference in median age of occurrence, with earlier intussusception in Pacific and Māori children compared to those of Asian or other ethnic origin. This association has not been reported previously and could be associated with the variable burden of infectious diseases, with Pacific and Māori children more likely to be admitted to hospital with gastroenteritis or respiratory infections.[30] An alternative factor could be infant weight, as Pacific and Māori infants are typically heavier than other ethnic groups.[31] It is uncertain whether earlier intussusception in Māori and Pacific infants increases their risk of vaccine associated events.

Timeliness of vaccine delivery remains problematic for New Zealand infants. A recent study found 23% of six week infants received the first vaccination of their primary series more than four weeks late, with 27% of three-month infants receiving their second vaccination more than six weeks late.[32] Unpublished data suggest that Māori infants are more likely than other ethnicities to receive their primary series late.[33] It remains to be clarified whether this delay extends to rotavirus vaccinations, and how the delay affects intussusception risk.

Vaccination associated intussusception is a rare event. Extrapolating Australian rates, and assuming a birth cohort of 60,000 and a vaccination rate of 95% (MoH HealthTarget), New Zealand might expect three extra intussusception cases per year. Given year-to-year variability as well as New Zealand's small immunisation cohort, it would take a number of years to detect this small increase in intussusception cases. However, the ethnic differences in timing of intussception combined with later immunisation delivery in Māori, occurring closer to the peak incidence of intussception, make a compelling case for continued intussception surveillence in New Zealand.

Intussusception symptoms include the baby having intermittent crying/screaming episodes, curling up or pulling the knees to the chest, vomiting +/- passing bloody, pink or red coloured jelly-like stools; this information is provided in several forums for parents as part of vaccination (http://www.immune.org.nz/vaccines/rotate-q%C2%AE, http://www.kidshealth.org.nz/intussusception).

Our study has a number of shortcomings. The data is anonymous, making it impossible to audit case notes to ascertain diagnostic accuracy or management details. We do not have length of stay nor outcome data.

Our study relies on retrospective coding data rather than prospectively identified cases. Coding data compiles data from large patient populations over significant time periods. It also has recognised weaknesses, including variable coding practices between institutions, changes in coding practice over time and simple coding errors. Hospitals are required to load discharge data into the NMDS within 21 days of the month of discharge, so our data are likely to be complete up to December 2013.

A Canadian study examining the reliability of ICD-9 and ICD-10 coding data in identifying intussusception calculated a sensitivity of 89.3%, and a specificity of >99.9%.[34] This study implies that ICD coding data miss intussusception cases and thus under-estimate the background incidence rate. Comparing an under-estimate of incidence with prospectively collected data would over-estimate the risk of vaccine associated adverse events.

Conversely, some study infants may have received Rotarix privately. If so, comparing our data with prospectively collected data would under-estimate vaccine risk. As our data were blinded and rotavirus vaccination was not systematically recorded prior to 2014, we were unable to assess this possibility.

Conclusion

The benefits of rotavirus vaccines vary between populations and similarly the disadvantages need to be balanced. In high-income countries such as Australia and New Zealand, most intussusception cases are diagnosed early and treated by enema with good outcomes.

In settings of high rotavirus mortality, particularly those with low background rates of intussusception, risk-benefit analyses overwhelmingly favour vaccination. In settings where rotavirus mortality is uncommon, and the background rates of intussusception are relatively high such as Australia, the benefits may be less dramatic. Nonetheless, recent Australian work supports vaccination, estimating >6500 fewer gastroenteritis hospitalisations compared to 14 additional intussusception cases annually.[7] Rotavirus hospitalization rates are higher in New Zealand than Australia[35] and intussusception rates are lower and thus the relative benefits of vaccination are likely to be greater.

It remains important for New Zealand to monitor the impact of rotavirus vaccination, specifically any increase in intussusception. It is particularly important to assess the impact on Māori and Pacific infants in whom intussusception occurs at an earlier age. By describing pre-vaccination epidemiology, our study will inform ongoing analyses of the rotavirus vaccine programme.

Author information:
Bronwyn Rosie, Paediatric Registrar, Starship Children's Health, Auckland; Stuart Dalziel, Paediatric Emergency Consultant, Starship Children's Health, Auckland; Elizabeth Wilson, Paediatric Infectious Diseases Consultant, Starship Children's Health, Auckland; Emma J Best, Senior Lecturer, Department of Paediatrics, University of Auckland, Auckland.
Corresponding author:
Emma J Best, Senior Lecturer, Department of Paediatrics, University of Auckland, Paediatric Infectious Diseases Consultant, Starship Children's Health, ADHB, Level 5 offices, Starship Children's Health, ADHB, Park Road, Grafton, Auckland.
e.best@auckland.ac.nz
URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7015

REFERENCES:

1. Bines JE, Kohl KS, Forster J, et al. Acute intussusception in infants and children as an adverse event following immunization: case definition and guidelines of data collection, analysis, and presentation. Vaccine. 2004 Jan 26;22:569–574.

2. Jiang J, Jiang B, Parashar U, et al. Childhood Intussusception: A Literature Review. PLoS ONE. 2013 Jul 22; 8(7): e68482. doi:10.1371/journal.pone.0068482. Print 2013.

3. Tate JE, Burton AH, Boschi-Pinto C, et al. 2008 estimate of worldwide rotavirus-associated mortality in children younger than 5 years before the introduction of universal rotavirus vaccination programmes: a systematic review and meta-analysis. Lancet Infect Dis. 2012 Feb. doi 10.1016/S1473-3099(11)7023–5. Epub 2011 Oct 24.

4. Grimwood K, Lambert SB. Rotavirus vaccines: opportunities and challenges. Hum Vaccin. 2009 Feb; 5(2):57–69. Epub 2009 Feb 8. Review.

5. Milne RJ, Grimwood K. Budget impact and cost-effectiveness of including a pentavalent rotavirus vaccine in the New Zealand childhood immunization schedule. Value Health. 2009 Sep;12(6):888–898. doi 10.1111/j.1524-4733.2009.00534.x. Epub 2009 Mar 18.

6. Centers for Disease Control and Prevention. Withdrawal of rotavirus vaccine recommendation. MMWR Morb Mortal Wkly Rep. 1999; 48:1007.

7. Carlin JB, MacCartney KK, Lee KJ, et al. Intussusception risk and disease prevention associated with rotavirus vaccines in Australia's national immunization program. Clin Infect Dis. 2013 Nov; 57:1427–1434. doi:10.1093/cid/cit520. Epub 2013 Aug 26.

8. GlaxoSmithKline. Data on file: Annual sales reports. 2009–2014.

9. Richardson V, Hernandez-Pichardo J, Quintanar-Solares M, et al. Effect of rotavirus vaccination on death from childhood diarrhea in Mexico. N Eng J Med. 2010 Jan 28; 362(4):299–305. doi: 10.1056/NEJMoa0905211.

10. Sheridan S, Lambert S, Grimwood K. 2012. Impact of rotavirus vaccination on childhood gastroenteritis. Microbiology Australia 33. URL: http://microbiology.publish.csiro.au/?act=view_file&file_id=MA12056.pdf (accessed 5 November 2013).

11. Payne DC, Baggs J, Zerr DM, et al. Protective association between rotavirus vaccination and childhood seizures in the year following vaccination in US children. Clin Infect Dis. 2014; 58 (2): 173-177. doi: 10.1093/cid/cit671. Epub 2013 Nov 20

12. Pardo-Seco J, Cebey-Lopez M, Martinon-Torres N, et al. Impact of Rotavirus Vaccination on Childhood Hospitalization for Seizures. Pediatr Infect Dis J. 2015 July; 34(7):769–73.

13. Buttery JP, Danchin MH, Lee KJ, et al. Intussusception following rotavirus vaccine administration: post-marketing surveillance in the National Immunization Program in Australia. Vaccine. 2011 Apr 5;29(16):3061–6. doi: 10.1016/j.vaccine.2011.01.088

14. Chen YE, Beasley S, Grimwood K. Intussusception and rotavirus associated hospitalisation in New Zealand. Arch Dis Child. 2005 Oct;90(10):1077–81. Epub 2005 Jun 17

15. National Health Board Business Unit. National Minimum Dataset (Hospital Events) Data Dictionary. Wellington: Ministry of Health; 2010.

16. Ministry of Health. 2004. Ethnicity Data Protocols for the Health and Disability Sector. Wellington: Ministry of Health.

17. Statistics New Zealand Infoshare. http://www.stats.govt.nz/infoshare, accessed 1 May 2014.

18. Bines JE. Rotavirus vaccines and intussusception risk. Curr Opin Gastro. 2005; 21:20–25.

19. Mansour AM, El Koutby M, El Barbary MM, et al. Enteric viral infections as potential risk factors for intussusception. J Infect Dev Ctries. 2013 Jan 15;7(1):28–35. doi: 10.3855/jidc.2321

20. Nylund CM, Denson LA, Noel JM. Bacterial enteritis as a risk factor for childhood intussusception: a retrospective cohort study. J Pediatr. 2010 May;156(5):761–5. doi: 10.1016/j.jpeds.2009.11.026. Epub 2010 Feb 6.

21. Lappalainen S, Ylitalo S, Arola A, et al. Simultaneous presence of human herpesvirus 6 and adenovirus infections in intestinal intussusception of young children. Acta Paediatr. 2012 Jun;101(6):663-70. doi: 10.1111/j.1651-2227.2012.02616.x. Epub 2012 Feb 21.

22. Bines JE, Liem NT, Justice FA, et al. Risk factors for intussusception in infants in Vietnam and Australia: adenovirus implicated, but

not rotavirus. J Pediatr. 2006;149:452–460.

23. Sáez-Llorens X, Velázquez FR, Lopez P, et al. A multi-country study of intussusception in children under 2 years of age in Latin America: analysis of prospective surveillance data. BMC Gastroenterol. 2013 May 27;13:95. doi: 10.1186/1471-230X-13-95.

24. Velazquez FR, Luna G, Cedillo R, et al. Natural rotavirus infection is not associated to intussusception in Mexican children. Pediatr Infect Dis J. 2004 Oct;23(10 Suppl):S173–8.

25. Tate JE, Simonsen L, Viboud C, et al. Trends in intussusception hospitalizations among US infants, 1993–2004: implications for monitoring the safety of the new rotavirus vaccination program. Pediatrics. 2008 May;121(5):e1125–32. doi: 10.1542/peds.2007–1590.

26. Macias M, Lopez P, Velazquez FR, et al. The Rotavirus vaccine RIX 4414 (Rotarix) is not associated with intussusception in one year old infants. In: Program and abstracts of the 45th Interscience Conference on Antimicrobial Agents and Chemotherapy, Washington, DC, 2005, Dec 16–19.

27. Justice F, Carlin J, Bines J. Changing epidemiology of intussusception in Australia. J Paediatr Child Health. 2005 Sep-Oct;41(9-10):475–8.

28. Muhsen K, Kassem E, Efraim S, et al. Incidence and risk factors for intussusception among children in Norther Israel from 1992 to 2009: A retrospective study. BMC Pediatr. 2014 Aug 31;14:218. doi: 10.1186/1471-2431-14-218.

29. Tate JE, Simonsen L, Viboud C, et al. Trends in intussusception hospitalizations among US infants, 1993–2004: Implications for monitoring the safety of the new rotavirus vaccination program. Pediatrics. 2008 May;121(5):e1125–32. doi: 10.1542/peds.2007–1590.

30. Baker MG, Barnard LT, Kvalsvig A, et al. Increasing incidence of serious infectious disease and inequalities in New Zealand: a national epidemiological study. Lancet. 2012 Mar 24;379(9821):1112–9. doi: 10.1016/S0140-6736(11)61780-7. Epub 2012 Feb 20.

31. Howe LD, Ellison-Loschmann L, Pearce N, et al. Ethnic differences in risk factors for obesity in New Zealand. J Epidemiol Community Health. 2015 Jun;69(6):516-22. doi: 10.1136/jech-2014-204464. Epub 2015 Mar 20.

32. Petousis-Harris H, Grant CC, GoodyearSmith F, et al. What contributes to delays? The primary care determinants of immunisation timeliness in New Zealand. J Prim Health Care. 2012 Mar 1;4(1):12–20.

33. Sinclair O. Ethnic inequities in health: Have we made progress? Pertussis mortality and morbidity in New Zealand for Māori and non-Māori over the past century. Unpublished Masters of Public Health Thesis, University of Auckland, 2015.

34. Ducharme R, Benchimol E, Deeks SL, et al. Validation of diagnostic codes for intussusception and quantification of childhood intussusception incidence in Ontario, Canada: a population-based study. J Pediatr. 2013 Oct;163(4):1073-9. e3. doi: 10.1016/j. jpeds.2013.05.034. Epub 2013 Jul 1.

35. Grimwood K, Huang QS, Cohet C, et al. Rotavirus hospitalisation in New Zealand children under 3 years of age. J Paediatr Child Health. 2006 Apr;42(4):196–203.

Publication rates and characteristics of undergraduate medical theses in New Zealand

Ibrahim Saleh Al-Busaidi, Yassar Alamri

ABSTRACT

AIM: Publication in peer-reviewed journals is widely regarded as the preferred vehicle for research dissemination. In New Zealand, the fate and publication rates of theses produced by medical students is unknown. The aim of this study was to examine the frequency and characteristics of publications derived from research conducted by Bachelor of Medical Sciences (BMedSc(Hons)) students at the three campuses of the University of Otago Medical School, New Zealand.

METHODS: A total of 153 BMedSc(Hons) theses accepted at the Otago Medical School during the period of January 1995 to December 2014 were analysed. Using standardised search criteria, PubMed and Google Scholar databases were searched in October 2015 to examine the number and characteristics of publications.

RESULTS: Overall, 50 (32.7%) out of 153 included theses resulted in 81 scientific publications. Ten (12.3%) publications featured in Australasian journals. The majority of publications were original articles (84%), with pathology and molecular biology (19%) being the most common research area. Although they did not reach statistical significance, publications in higher impact factor journals trended towards having a senior first author as opposed to a student first author (p=0.06).

CONCLUSION: Although higher than reported figures from previous studies, publication rates of BMedSc(Hons) theses remain lower than expected. To improve our understanding of medical student publishing in New Zealand, formal examination of the factors hindering medical students from publishing their theses is imperative.

Ongoing health research is crucial to improve medical knowledge and clinical practice. Appropriate dissemination and publication of research findings is the key and final step in the scientific communication process. In fact, making all research findings, positive or otherwise, publicly available to potential users is an ethical obligation mandated by the international scientific community.[1] This is usually achieved through articles in peer-reviewed journals, conference presentations and academic theses or dissertations. Inaccessible information may result in a number of unfavourable consequences including waste of resources and medical knowledge, unnecessary duplication of studies and loss of scientific integrity/trust.[2]

Publishing in refereed journals is widely regarded by funders and academic institutions as the preferred vehicle for research dissemination.[3] The number of peer-reviewed publications is a significant indicator commonly used to assess individual and institutional research performance.[4,5]

Exposure to undergraduate research improves critical thinking skills among medical students, stimulates their interest in academic medicine and is associated with increased postgraduate research productivity.[6] Career progression and future involvement in research were reported by medical students to be the main motivations for participating in undergraduate medical research.[6,7] Furthermore, research experience is taken

into account by specialty training colleges in the selection process for competitive specialist training posts and additional points are awarded for publications.[6,8]

In addition to other undergraduate medical research activities, interested students may choose to undertake any of several intercalated research degrees, often eventuating in a thesis.[8] At the University of Otago, the Bachelor of Medical Sciences with Honours 'BMedSc(Hons)' course is an intercalated degree involving a full-time one-year research available to medical students who have satisfactorily completed three or more years of their Bachelor of Medicine and Bachelor of Surgery (MB ChB) programme.[9] The submission of a satisfactory thesis describing the results of supervised research is a prerequisite for the completion and award of the degree. Publishing the project findings in peer-reviewed journals, while encouraged, is not required for the award of the degree.

It has been argued that research-degree theses must be made publicly available.[10] Publication of resultant research in indexed peer-reviewed journals reflects its quality and scientific value, and the acceptability of its content to the scientific community.[5,11] Despite the recognised importance of student research and publishing,[8] few studies have evaluated research productivity of medical student theses.[5,11–13] The fate and publication patterns of undergraduate medical thesis-related research in New Zealand is unknown. This study was aimed to assess the characteristics and publication pattern of BMedSc(Hons) theses in peer-reviewed journals conducted by medical students at the University of Otago Medical School.

Methods

Search strategy

The electronic Otago University Research Archive[14] (ourarchive.otago.ac.nz) was searched for all BMedSc(Hons) theses. Furthermore, the Faculty of Medicine was contacted for additional theses that were not electronically archived. Theses accepted between 1 January 1995 and 31 December 2014 were included. The latter cut-off was set to allow for a reasonable time for any submitted publication upon

thesis completion to go through the peer-review process.

During October 2015, PubMed and Google Scholar databases were searched by both authors for publications using the student's first and last names. A publication was considered relevant if the student was one of the co-authors and the publication title/abstract was related to the student's thesis topic.

Data collection

Confirmed theses were examined more closely. Information pertaining to projects' start and end dates, number of supervisor(s) as well as subject areas were collected. The main subject area was based on key words reported in the thesis. If there were no key words reported, the main research filed was defined by consensus between the authors based on the title and content of the thesis.

For each corresponding publication, author (number of authors and order of authorship) and article-related data (type of and year of publication, journal name and impact factor) were collected. Journals' impact factor was obtained using the Journal Citation Reports of the Thomson Reuters Web of Science. Journals not included in the Thomson Reuters Web of Science were allocated an impact factor of zero.

Statistical analysis

Collected data were inputted onto an Excel sheet. Descriptive statistics were used to analyse most of the data. Comparisons were conducted using an independent-samples Student t-test. Regression analysis was used to test for correlations. Statistical significance was determined if type I error rate was <5% (p-value<0.05). All analyses were performed using the Statistical Package for Social Sciences software (SPSS Statistics®, version 22.0.0.0).

Results

Study sample

A total of 154 theses were examined. Only one thesis (from 1965) was excluded as it fell beyond the time-limit set for the study. There was a mean of 7.7 theses submitted per year (range, 4–17). The number of students enrolling in and submitting BMedSc(Hons) theses has gradually increased through the study period (Figure 1). The most common subject areas of research

Figure 1: Number of medical students enrolled in BMedSc(Hons) programme at the University of Otago (1995–2014).
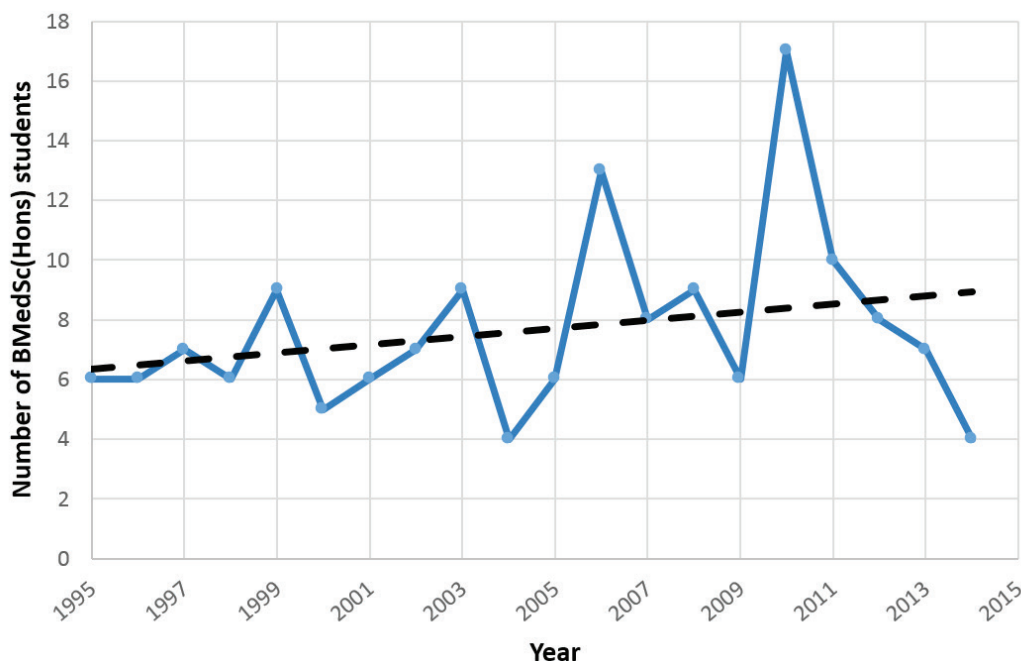
were pathology and molecular biology (19%), followed by community medicine (including general practice and public health, 9.8%) and endocrinology and reproductive physiology (9.8%).
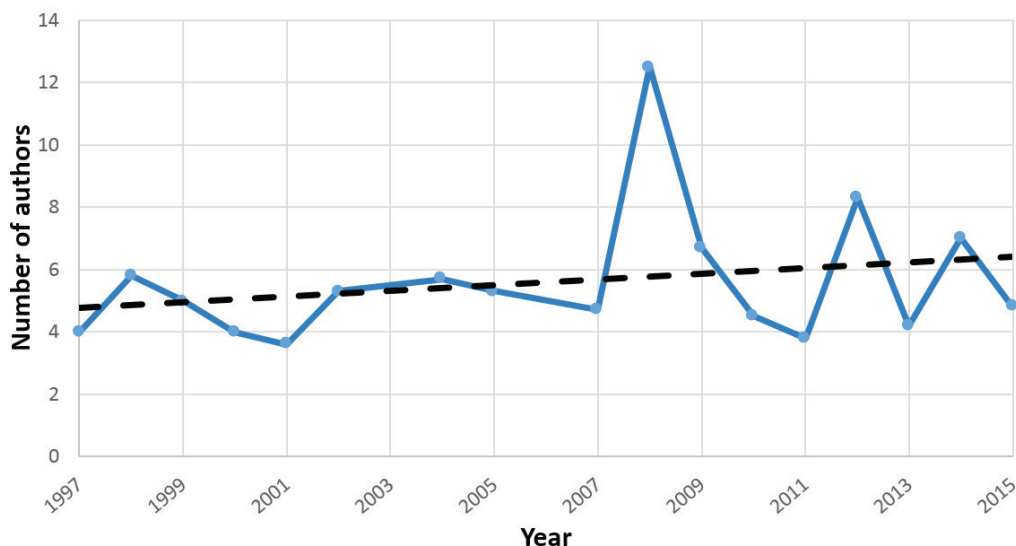
Publication data

Overall, 50 (32.7%) out of 153 theses resulted in at least one article published in indexed peer-reviewed journals. A total of 81 publications, all of which were in English, were identified (range, 1–9 publications per thesis). Original articles (84%) and reviews (14.8%) constituted the most common types of publication. In almost all publications,

the student was the first (50.6%) or second (48.1%) author. The median lag between the start of research and publication of a manuscript was 176 weeks (range, 43–652 weeks).

Five supervisors were co-authors of about a third (32.1%) of the publications. Moreover, the number of academic staff supervising students (but not collaborators) has steadily increased through the years, which could explain the upward trend in the mean number of authors per publication (Figure 2). Students who published did not differ in the mean number of supervisors compared with students who did not publish (1.6 vs 1.4, p=0.08).

Figure 2: Mean number of authors per publication over time.

Publishing journals

Ten articles (12.3%) were published in Australasian journals. The mean impact factor of publishing journals was 1.67 (range, 0–30.3). Publications in higher impact factor journals trended towards having a senior first author as opposed to a student first author, although this did not reach statistical significance (p=0.06). There was a moderate but significant, direct correlation between the total number of the authors and the impact factor of the publishing journal (adjusted R^2=0.14, p<0.001).

Discussion

Findings from this study provide, for the first time, an insight into the number and characteristics of publications from undergraduate theses produced by medical students in New Zealand. Adding to the existing literature on New Zealand medical student research and publishing,[8,15] this study sheds light on their contribution to international scientific literature.

There are several compelling ethical[1] and professional reasons for supervisors[3,4] and their students[6–8] to publish in refereed journals. Publication rates from BMedSc(Hons) theses in New Zealand have been found to be pleasingly high; almost one-third (32.7%) of theses led to a publication in a peer-reviewed journal. This rate is higher than figures reported by studies from developing and developed countries, with publication rates ranging from 13.9% and 30%.[5,10–12,16]

Nearly half of medical and health-related studies remain unpublished.[2] Although regarded as a piece of literature, the visibility of a thesis is low without appropriate dissemination of its content to the wider scientific community. A recent study evaluated publication rates of Master's in Public Health theses at the University of Auckland, New Zealand, found time constraint (63%), lack of support from supervisors (35%) and low confidence in writing for peer-reviewed publication (29%) were the most commonly cited reasons for non-publication of a thesis.[17]

In our study, the average time taken from commencing research to publication was more than three and a half years; a finding similar to previous research.[10,12] The long delay for theses to appear in publication can be explained by study demands as students commence their medical studies immediately upon the end of the one-year research programme. Moreover, student's supervisor(s) were one of the main drivers behind publication. Five supervisors were co-authors of about a third of published articles. It was, however, the quality not the quantity of supervisors (or collaborators) that influenced publication rates.

Findings from this study have implications for medical schools and funders of undergraduate medical research. Medical students undertaking BMedSc(Hons) research projects are supported financially by scholarships and awards from the Faculty of Medicine at the University of Otago as well as local trusts.[13] Thesis writing is a valuable opportunity to teach medical students the scientific method of writing and stimulate their interest in a career in academia.[6] Failure to transform a thesis into peer-reviewed publications raises questions about the scientific value of generated knowledge and the role of undergraduate medical theses in research training and education.[10] Sustained commitment from supervisors, practical support in academic writing (such as writing courses) and setting the expectation that publication is part of the thesis writing process might be reasonable measures to increase publication rates from medical theses.[4,17]

Results from our study may underestimate publication rates from medical student theses. First, articles which are published in journals that are not indexed in the two databases used in this study may have been missed. Second, findings from medical theses might have been disseminated by other means than peer-reviewed publications including conference papers or posters, book chapters and technical reports. Third, our study examined research outputs from undergraduate medical theses produced by only one medical school in New Zealand. The BMedSc(Honours) at the University of Auckland is a full-time one-year research programme offered to medical students, similar to that of Otago's.

The overall research productivity of medical students in New Zealand cannot be judged merely based on this study. Medical student publications may arise from research activities not examined in this study which include formal research

training projects,[18] summer studentships, research electives/selectives and independent research.[7,8] Notwithstanding the aforementioned limitations, we believe findings from this study give a fair reflection of undergraduate medical theses in New Zealand.

Conclusions

To the best of our knowledge, this is the first study to explore medical student research production from undergraduate theses in New Zealand. Publication rates of BMedSc(Hons) theses were higher than reported figures from previous studies. The role of undergraduate medical theses in research training and productivity has to be clearly defined and interventions to increase publication rates should be implemented. To improve our understanding of medical student publishing in New Zealand, formal examination of the factors hindering medical students from publishing their theses is imperative.

Author information:
Ibrahim Saleh Al-Busaidi, Christchurch Public Hospital, Canterbury District Health Board, Christchurch; Yassar Alamri, Department of Medicine, New Zealand Brain Research Institute, Christchurch.
Corresponding author:
Ibrahim Saleh Al-Busaidi, Christchurch Public Hospital, Canterbury District Health Board, Riccarton Avenue, Christchurch.
ibra.3sk@gmail.com

URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7016

REFERENCES:

1. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. J Am Coll Dent. 2014;81:14–8.

2. Chan AW, Song F, Vickers, A et al. Increasing value and reducing waste: addressing inaccessible research. Lancet. 2014;383:257–66.

3. Lowcay B, McIntyre E, Hale M, Ward AM. Peer reviewed publication rates. An indication of research output. Aust Fam Physician. 2004;33:284–6.

4. McGrail MR, Rickard CM, Jones R. Publish or perish: a systematic review of interventions to increase academic publication rates. Higher Educ Res Dev. 2006;25:19–35.

5. Arriola-Quiroz I, Curioso WH, Cruz-Encarnacion M, Gayoso O. Characteristics and publication patterns of theses from a Peruvian medical school. Health Info Libr J. 2010;27:148–54.

6. Griffin MF, Hindocha S. Publication practices of medical students at British medical schools: experience, attitudes and barriers to publish. Med Teach. 2011;33:e1–8.

7. Park SJ, McGhee CN, Sherwin T. Medical students' attitudes towards research and a career in research: an Auckland, New Zealand study. N Z Med J. 2010;123:34–42.

8. Al-Busaidi IS, Al-Shaqsi SZ. Students' contribution to the New Zealand Medical Journal: a 14-year review. N Z Med J. 2015;128:47–52.

9. Bachelor of Medical Science with Honours (BMedSc(Hons)). University of Otago; Available from: http://www.otago.ac.nz/courses/qualifications/bmedschons.html#regulations

10. Chalmers I. For ethical, economic and scientific reasons, health-relevant degree theses must be made publicly accessible. Evid Based Med. 2012;17:69-70.

11. Salmi LR, Gana S, Mouillet E. Publication pattern of medical theses, France, 1993-98. Med Educ. 2001;35:18–21.

12. Nieminen P, Sipilä K, Takkinen HM, Renko M, Risteli L. Medical theses as part of the scientific training in basic medical and dental education:

---

experiences from Finland. BMC Med Educ. 2007;7:51.

13. Dhaliwal U, Singh N, Bhatia A. Masters theses from a university medical college: publication in indexed scientific journals. Indian J Ophthalmol. 2010;58:101–4.

14. Otago University Research Archive. University of Otago; Available from: http://ourarchive.otago.ac.nz

15. Al-Busaidi IS. Trends and patterns in medical student research and publishing in New Zealand. N Z Med J. 2015;128:116–18.

16. Munung N, Vidal L, Ouwe-Missi-Oukem-Boyer O. Do Students Eventually Get to Publish their Research Findings? The Case of Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome Research in Cameroon.

Ann Med Health Sci Res. 2014;4:436–41.

17. Bullen CR, Reeve J. Turning postgraduate students' research into publications: a survey of New Zealand Master's in public health students. Asia Pac J Public Health. 2011;23:801–9.

18. Wilson N, Peace J, Edwards R. Research outputs by medical students from routine public health training projects. Med Teach. 2011;33:254.

How effective is our current Orthopaedic Prioritisation Tool for scoring patients for arthroplasty surgery?

Neal Singleton, Lewis Agius, Sudhindra Rao

ABSTRACT

AIM: To compare those patients who are being accepted onto the waiting list for total hip and knee arthroplasty surgery with those patients who are being declined surgery, using a validated functional questionnaire.

METHOD: The clinic records from all patients seen for consideration of total hip or knee arthroplasty at Hawkes Bay Hospital during the preceding four months were reviewed. We sent the Oxford Hip and Knee Score questionnaire to all patients who had been put forward for consideration of surgery.

RESULTS: Of the 150 patients we surveyed, 81 had been accepted onto the waiting list and received a date for surgery within the next four months and 69 had been declined surgery. Of the 81 patients who had been accepted onto the waiting list for surgery, 61 returned the Oxford questionnaire with an average score of 10.9. Of the 69 patients who had been declined surgery, 59 returned the Oxford questionnaire with an average score of 10.8. Thus the mean Oxford score was not statistically different between those patients being accepted onto the waiting list for surgery and those being declined surgery (p=0.925).

CONCLUSION: No difference was found between those patients being accepted onto the waiting list for total hip or knee arthroplasty and those being declined surgery in Hawkes Bay after using the Oxford Hip and Knee Score as a measure of functional impairment. The average Oxford score indicates that patients being seen in Hawkes Bay Hospital for consideration of total hip or knee arthroplasty are severely functionally impaired as a result of their condition.

Osteoarthritis is a common condition affecting about 15% of adult New Zealanders.[1] The incidence increases with advancing age, with a significant rise after the age of 60 years. Given the ageing population it is likely that New Zealand will have to contend with an increasing socioeconomic burden from osteoarthritis in the future.[2] In the US it is projected that by 2030 demand for total hip arthroplasty (THA) and total knee arthroplasty (TKA) will have increased by 174% and 673%, respectively.[3] During the last 13 years in New Zealand the total number of THA cases has increased by 75% and TKA 158% and it estimated that by 2026 the absolute number of THA and TKA cases will increase by 84% (8,950 cases) and 183% (8,613 cases) respectively.[4]

THA and TKA are common procedures which are used to treat end-stage osteoarthritis when all non-surgical management options have been exhausted. The goal of arthroplasty surgery is to alleviate pain and restore function. However, given the costs associated with arthroplasty surgery and the limited resources available, public hospitals cannot offer surgery to all patients seen for consideration of joint replacement. As such, there must be a just and fair way to determine who should be offered surgery. Determining this though, raises a number of ethical, social, economic and surgical quandaries. There are a number of different methods currently in use for prioritising patients for surgery. The goal of all such scoring tools is to prioritise patients according to their symptoms and likely benefit from surgery.

Hawkes Bay DHB uses a prioritisation tool which is based on the New Zealand

Orthopaedic Association hip and knee prioritisation tool but has three categories:

1. potential deterioration (if surgery were to be delayed),

2. expected benefit from surgery and

3. an overall surgeon assessed severity rating with a score of 1–5 being given (with 5 being the most severe).

A score can be overridden if the planned surgery is for malignancy, impending spinal cord/nerve compression or for a loose/infected joint prosthesis. There is provision for clinical override in exceptional circumstances whereby a patient can be added to the waiting list at the surgeon's discretion regardless of their prioritised score. Based on the score a patient achieves utilising the prioritisation tool, they are then either placed onto the waiting list for surgery or surgery is declined. This is dependent on what score is deemed the threshold score. A financial threshold score is the minimum score that a patient needs to achieve in order to be listed for surgery ie, if the patients score above the threshold score then they are listed for surgery and if they score below the threshold score then surgery is declined. This financial threshold is subject to adjustment by individual DHBs' commensurate with the Health Ministry's waiting time target of four months.

The aim of this study was to compare those patients who had been accepted onto the waiting list for THA and TKA with those who had been declined surgery, using the Oxford score as a measure of disability in order to determine whether the current prioritisation tool is effective. The Oxford Hip and Knee Scores were devised as joint specific instruments for assessing function while minimising influence from other comorbidities. The Oxford Hip and Knee Scores have been evaluated independently and found to be the best and most reliable systems for the assessment of hip and knee replacement, respectively.[5,6,7]

We chose to use the Oxford score as it has been validated in a number of studies.[8,9,10] Furthermore, it is widely used in national joint registries including the New Zealand National Joint Registry. The score is comprised of twelve questions which assess pain and function. Patients complete each question giving a score of between 0 to 4 with 4 being the best outcome and 0 being the worst. A total score out of 48 is achieved with 48 being the best outcome score and 0 being the worst, indicating severe disability. Kalairajah et al have recommended a category of excellence for an Oxford score of >41, good for a score of 34–41, fair for 27–33 and poor for those <27.[11] The minimal clinically important difference (MCID) is the smallest change in score that patients perceive as meaningful and has been reported as 5 for the Oxford Hip Score and 4 for the Oxford Knee Score.[12] That the MCID is small indicates that even a small change in score may represent a clinically important change in function.

Method

All patients who are referred to Hawkes Bay DHB with a diagnosis of hip or knee osteoarthritis are assessed by a Consultant Orthopaedic surgeon or Registrar in an outpatient clinic. After clinical evaluation, if a patient is clearly not in need of arthroplasty or they do not desire surgery, they can either be discharged back to the care of their General Practitioner (for ongoing non-surgical management) or reviewed in clinic at a later date. These patients do not receive a prioritisation score. All other patients who desire surgery and who are deemed appropriate for surgery are scored utilising the prioritisation tool. The maximum score is 100, the higher the score the greater the level of perceived disability (and the potential need for and benefit from surgery). It is this score that determines those who are referred to the surgical waiting list and those who are declined surgery based on the predetermined threshold score. At the time of completing this study the threshold score in Hawkes Bay DHB was 80 points. The completed forms are forwarded to the Elective Services Manager for final determination of those that will be accepted onto the (four month) surgical waiting list. Those deemed to have not reached the threshold are not accepted onto the waiting list.

We reviewed clinic records for the four-month period, 1st July 2015 to 31st October 2015. This four-month period was selected at random by the primary author who noted an increasing number of patients with severe disability being declined THA and TKA due to not meeting the threshold score.

All data was collected prospectively. All patients who had an application for THA or TKA put forward for review by the Elective Services Manager were sent the Oxford Hip or Knee Score questionnaire to complete. No patient was aware if they had been accepted or declined surgery at the time of completing the questionnaire. We tabulated the results in order to perform a direct comparison of Oxford scores between those patients who had been accepted onto the waiting list for surgery and those patients who had been declined surgery after receiving the list of accepts and declines from the Elective Services Manager.

Ethics approval was obtained from the hospital's research committee.

Statistical analysis

Standard descriptive statistics including means, ranges, standard deviations, frequencies and percentages were used to summarise the data between those accepted and those declined surgery. Outcomes were compared between those accepted and those declined surgery using t-tests and Chi-square tests. A two-tailed p-value of <0.05 was taken to indicate statistical significance. Data were analysed using SPSSv23.0.

Results

During the four-month study period, 150 patients had an application for THA or TKA put forward for review by the Elective Services Manager (73 applications for THA and 77 applications for TKA). Of these 150 patients, 81 met the financial threshold (average prioritisation tool score 85.4) and were accepted onto the waiting list for surgery (46 for THA and 35 for TKA), and 69 failed to meet the financial threshold (average score 74.2) and were declined inclusion onto the waiting list (27 for THA and 42 for TKA).

Oxford Hip and Knee Score questionnaires were returned by 120/150 (80% of patients) made up of 61/81 (75%) who had been accepted for surgery and 59/69 (86%) who had been declined for surgery. Those who returned the questionnaire are further described in Table 1. This table also shows the associations between being accepted or declined for surgery based on mean score

Table 1:

Variable	Accepted for surgery N=61			Declined for surgery N=59			Mean Difference (95% CI)
	Mean (SD)	Median (IQR)	Range	Mean (SD)	Median (IQR)	Range	
Oxford score	10.9 (6.0)	10 (7 to 13)	2 to 35	10.8 (6.3)	9 (7 to 14)	0 to 31	0.1 (-2.1 to 2.3) P=0.925
Age	70.5 (9.3)	70 (66.5 to 77.5)	47 to 94	69.2 (10.2)	71 (62 to 77)	46 to 88	1.3 (-2.2 to 4.8) P=0.458
	Accepted N/61 (%)		Declined N/59 (%)		Odds ratio (95% CI)		
Female gender	32 (50.8%)		31 (49.2%)		1.00		
Male gender	29 (50.9%)		28 (49.1%)		(0.49 to 2.04) P=0.993		
Māori ethnicity	9 (52.9%)		8 (47.1%)		1.10		
Non-Māori ethnicity	52 (50.5%)		51 (49.5%)		(0.39 to 3.08) P=0.851		
THA	39 (67.2%)		19 (32.8%)		3.73		
TKA	22 (35.5%)		40 (64.5%)		(1.75 to 7.95) P=0.001		

differences for continuous variables and odds ratios for categorical variables. There was no association between Oxford score and age (r=0.06, P=0.53). As can be seen in Table 1 there was no statistically significant associations between whether or not patients were accepted for surgery except for hip compared to knee surgery.

The mean Oxford score was not significantly different between those patients being accepted onto the waiting list for surgery and those being declined surgery, indicating no significant functional difference between these two groups (Table 1).

Gender, age and ethnicity were not related to acceptance onto the waiting list for surgery. However, those patients seen for consideration of THA were significantly more likely to be accepted for surgery than those seen for TKA (Table 1).

Conclusion

We found that when using the Oxford Hip and Knee Score as a measure of functional impairment there was no statistical difference between those patients being accepted onto the waiting list for THA and TKA and those being declined surgery in Hawkes Bay (p=0.925). One could therefore question the effectiveness of our current prioritisation tool.

Furthermore, the average Oxford score in our study among those patients being accepted for surgery and those being declined surgery suggests that as a region, Hawkes Bay patients who are seen for consideration of arthroplasty surgery are severely functionally impaired secondary to their osteoarthritis with an overall Oxford score of 10.9.

Discussion

In 2004 the Ministry of Health introduced the joint initiative in an effort to increase the number of arthroplasty cases being performed. A recent review by the Director General of Health reported that waiting list times had reduced and the absolute number of joint replacements being performed in New Zealand is increasing. In reality however, given the ageing population, more patients are being seen for consideration of THA and TKA. Blackett et al reported that of 858 patients where surgery was both desired by the patient and deemed appropriate by the surgeon, 307 (36%) were declined for being below the financial threshold.[13] Currently the over 65-year age group represents around 13% of the New Zealand population.[14] This is expected to almost double to 25% by 2031 which will place an increasing burden on healthcare resources, particularly arthroplasty surgery, as the elderly live longer, lead more active lives and are less accepting of disability.[14]

Although the goal of all scoring tools is to prioritise patients according to their symptoms and likely benefit from surgery, it is likely that they struggle to differentiate between patients with greater disability. This may account for the results of our study in which there was no difference between those patients being accepted onto the waiting list for surgery and those being declined surgery despite those patients who were accepted for surgery exceeding the prioritisation threshold score with those who were declined surgery falling below it. A more sensitive scoring tool may be necessary in order to differentiate between patients with higher levels of disability. Furthermore, the prioritisation tool in use is a generic tool and not specific to any particular orthopaedic condition (it is used to score all patients seen for consideration of elective orthopaedic surgery) which makes targeted assessment of a patient's symptoms difficult. Individualised scoring tools tailored to specific orthopaedic conditions may be necessary to achieve greater accuracy.

The MCID has been reported as being 5 for the Oxford hip score and 4 for the Oxford knee score indicating that not only is there no statistical difference in scores between those patients being accepted for surgery and those being declined but there is no clinical difference either and patients are equally disabled.[12]

One of the major determinants of post-operative outcome is pre-operative function and, therefore, those patients who are significantly impaired at the time of surgery are still left with a degree of disability post-operatively and are unlikely to ever achieve the same functional outcome as those patients who are less impaired at the time of surgery.[15] In this study, patients with significant disability (mean Oxford score 10.8) are being declined surgery and are

therefore either left with significant pain and dysfunction which will no doubt have an effect on their quality of life or if they do eventually make it onto the waiting list (and undergo surgery) they are unlikely to ever achieve the same functional outcome as those who have their surgery at an earlier stage. Furthermore, it is accepted that operating on end-stage osteoarthritis can be more surgically challenging and possibly require the use of more expensive implants.

Williams et al reported that following THA, the mean Oxford score can be expected to increase by an average of 20 points and 14 points following TKA.[16] The mean Oxford score for those being accepted onto the waiting list for surgery was 10.9 in this study. When we separated THA and TKA the mean Oxford score for those being accepted for THA was 11.1 and those being accepted for TKA was 10.5. If the THA group increased by 20 points they would still only achieve a post-operative Oxford score of 31.1 meaning most are, potentially, only achieving a fair outcome. Similarly, in the TKA group, provided patients improve by 14 points, as has been suggested, most patients would only be expected to achieve an outcome of 24.5 points resulting in a poor outcome. In contrast, the New Zealand Joint Registry suggests that 89% of New Zealanders have good or excellent Oxford scores at five years after THA and TKA.[17] These results would suggest that patients in Hawkes Bay (over the study period) are having their surgeries undertaken when they are severely functionally impaired and they are, therefore, potentially unlikely to ever achieve outcomes comparable with those who have surgery earlier. This situation is unlikely to be unique to Hawkes Bay and it is likely that DHBs across New Zealand will encounter similar situations where the threshold score necessary to be listed for surgery will need to be increased to cope with the current burden of disease but with the financial and resource constraints.

Methodological considerations

All data was collected prospectively and the rate of returned questionnaires was similar between those accepted (75%) and those declined surgery (86%), which adds strength to the study. Potential weaknesses of the study include the small number of patients included but even with a larger sample size the results would unlikely change.

In 2013 Gwynne-Jones highlighted the existence of regional variations in the provision of arthroplasty surgery within New Zealand.[18] Variations between regions can be attributed to a number of factors including the age of the population, ethnic diversity, average household income and thus access to privately funded surgery as well as type of employment. This paper has further highlighted variations between regions and suggests that taking these factors into consideration for future planning may be a worthy consideration. In Gwynne-Jones' paper 96% of patients listed for arthroplasty surgery in Otago had an Oxford score of 20 or less, 74% less than 15 and 37% less than 10 points.[18] In the Hawkes Bay population, during our four-month study period, among those patients who had been declined surgery 90% had an Oxford score of less than 20, 80% less than 15, and 51% less than 10 points indicating that even the patients being declined surgery in Hawkes Bay are equally, if not more, functionally disabled than the Otago population of patients who are being accepted onto the waiting list for surgery. Similarly, in Canterbury the mean Oxford score in 726 patients who underwent THA between 2009 and 2011 was 18 compared with an average score of 10.9 in our study.[19] This could be attributed to the age of the population; 16.8% of Hawkes Bay's population are over the age of 65 years compared with 15.7% in Otago and 15.5% in Canterbury (and 14.3% for New Zealand as a whole).[20] These results could also be attributed to the ethnic makeup of the regions. Māori are known to present late and with more severe disability secondary to osteoarthritis (and consequently have poorer outcomes following surgery).[21] In Hawkes Bay Māori make up 24.3% of the population compared with just 7.5% in Otago and 8.1% in Canterbury.[20]

Blackett et al reported that the average NZOA score for those patients being listed for surgery (70.62) was higher than those being declined surgery (55.38, p<0.001) at Whangarei Hospital.[13] Similarly, at Hawkes Bay Hospital, the average NZOA score in his paper was higher for those being listed for surgery (76.96) compared with those being declined (64.66, p<0.001).[13] This would

indicate that the scoring tools are working well in differentiating patients being seen for consideration of arthroplasty surgery. However, our study found that when using the Oxford score as a measure of disability, there was no difference between patients being accepted onto the waiting list for surgery and those being declined surgery. Blackett et al also reported that 36% of patients in whom surgery was both desired by the patient and recommended by the surgeon were declined surgery.[13] In our study 46% of patients (69 out of 150) were declined surgery where it was both desired by the patients and deemed appropriate by the assessing surgeon, meaning that close to half of all patients being seen for consideration of arthroplasty surgery in Hawkes Bay are being declined.

Gwynne-Jones reported that in Otago the intervention rate for THA and TKA was 20.4 cases per 10,000 per year publically funded and 17.5/10,000 per year funded by ACC (compared with 33.0/10,000 in New Zealand as a whole) ie, a total intervention rate of 37.9/10,000 per year in Otago (public and ACC funded private).[18] The actual demand in Otago is 41.7/10,000 per year meaning an unmet demand of 3.8/10,000 per year or 73 cases per year.[18] Our results would suggest that the unmet demand in Hawkes Bay is even higher with 69/150 being declined surgery where it is was both desired by the patient and suggested as appropriate by the surgeon over just a four-month period indicating a potential unmet need of 207 cases per year.

This issue of 'unmet need' is a common theme in orthopaedic literature and addressing this issue is challenging. Royal Australasian College of Surgeons figures (2011) recommend one orthopaedic surgeon for 15,000–20,000 population .The current New Zealand average is 1/17,700 population. The present situation in Hawkes Bay region is 1/24,200. This suggests significant under-resourcing of orthopaedic specialists in Hawkes Bay.

A nationwide prioritisation tool would help to compare regions in a uniform way and therefore enable for a fairer provision of resources. A trial is currently underway to implement a nationwide orthopaedic prioritisation tool. Its use will become mandatory later this year. The tool is based on a functional impairment questionnaire ("How does your condition affect your life?"), which patients complete themselves. This is based on six categories:

1. social interactions,
2. personal relationships,
3. ability to meet your responsibility to others,
4. personal care,
5. personal safety, and
6. leisure activities.

This score is combined with the score from a surgeon-completed questionnaire based on five categories:

1. surgeon perceived impact on patient,
2. likelihood that significant deterioration in symptoms/function will occur in the next six months,
3. consequences (or significance) of deterioration insymptoms/function occurring in the next six months,
4. amount of benefit from the proposed surgery for this patient,
5. risk of surgery for this patient—death or significant complications.

The combined scores give an overall score (out of 100) which is then used to determine who is placed on the waiting list for surgery based on the current threshold score. This nationwide prioritisation tool is web based and will therefore enable for a comparison between regions and may help government planning for the future. It does not, however, collect data on the type of surgery proposed. It is yet to be seen whether this new prioritisation tool will enable for differentiation between severely disabled patients and therefore effectively score patients for arthroplasty surgery. A study similar in design to this one comparing patients being accepted and declined surgery using the new prioritisation tool with the Oxford score would be a worthy consideration.

In conclusion, this study has shown that there is no difference between those patients being accepted onto the waiting list for THA and TKA and those being declined surgery in Hawkes Bay using the Oxford score as a measure of disability. Nearly half of all patients seen for consideration of THA or TKA are declined surgery despite being severely functionally impaired with

an average Oxford score of 10.8. This study has highlighted the current situation in New Zealand where the ageing population is placing an increasing demand on healthcare services. Even with the introduction of a nationwide prioritisation tool it is unlikely that we will be able to differentiate between those patients who are significantly disabled and as a result there will likely be a large number of patients unable to receive surgery and who are significantly disabled from their orthopaedic condition.

Author information:
Neal Singleton, Orthopaedic Registrar, Hawkes Bay Hospital; Lewis Agius, Orthopaedic Registrar, Hawkes Bay Hospital; Sudhindra Rao, Consultant Orthopaedic Surgeon, Hawkes Bay Hospital.
Corresponding author:
Neal Singleton, Orthopaedic Registrar, Hawkes Bay Hospital.
nealsingleton@hotmail.com
URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7018

REFERENCES:

1. Ministry of Health: A portrait of health – key results from the 2006/7 New Zealand health survey.

2. Litwic A, Edwards M, Dennison E, Cooper C. Epidemiology and burden of osteoarthritis. Br Med Bull. 2013;105:185–99.

3. Kurtz S, Ong K, Lau E et al. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. J Bone Joint Surg Am. 2007;89:780–85.

4. Hooper G, Lee A, Rothwell A, Frampton C. Current trends and projections in the utilisation rates of hip and knee replacement in New Zealand from 2001 to 2026. NZMJ 335 2014;127:82–93.

5. Dunbar M, Robertson O, Ryd L, Lidgren L. Appropriate questionnaires for knee arthroplasty: results of a survey of 3600 patients from The Swedish Knee Arthroplasty Registry. J Bone Joint Surg [Br] 2001;83-B:339–44.

6. Dunbar M, Robertson O, Ryd L, Lidgren L. Translation and validation of the Oxford-12 item knee score for use in Sweden. Acta Orthop Scand 2000;71:268–74.

7. Ostendorf M, van Stel H, Buskens, E et al. Patient-reported outcome in total hip replacement: a comparison of five instruments of health status. J Bone Joint Surg [Br] 34 2004;86-B:801–8.

8. Rothwell AG, Hooper GJ, Hobbs A, Frampton CM. An analysis of the Oxford hip and knee scores and their relationship to early joint revision in the New Zealand Joint Registry. J. Bone Joint Surg. Br. 2010; 92: 413–8.

9. Dawson J, Fitzpatrick R, Carr A, Murray D. Questionnaire on the perceptions of patients about total hip replacement. J. Bone Joint Surg. Br. 1996; 78: 185–90.

10. Field RE, Cronin MD, Singh PJ. The Oxford hip scores for primary and revision hip replacement. J. Bone Joint Surg. Br. 2005; 87: 618–22.

11. Kalairajah Y, Azura K, Hulme C et al. Health outcome measures in the evaluation of total hip arthroplasties: a comparison between the Harris hip score and the Oxford hip score. J. Arthroplasty 2005; 20: 1037–41

12. Beard D, Harris K, Dawson J, Doll H, Murray D, Carr A, Price A. Meaningful changes for the Oxford hip and knee scores after joint replacement surgery. J Clin Epidemiol. 2015 35 Jan;68(1):73–9.

13. Blackett J, Carslaw A, Lees D, Fargher S, Rao S, Pohl M. The impact of the 6-month waiting target for elective surgery: a patient record study. NZMJ 2014;127:45–53.

14. Ministry of Social Development data (www.msd.govt.nz)

15. Roder C, Staub LP, Eggli S et al. Influence of preoperative functional status on outcome after total hip arthroplasty. J Bone Joint Surg Am. 2007;89:11–7.

16. Williams D, Blakey C, Hadfield S et al. Long-term trends in the Oxford knee score following total knee replacement. Bone Joint J. 2013;95-B(1):45–51.

17. New Zealand Joint Registry. www.cdhb.govt.nz/njr/

18. Gwynne-Jones D. Quantifying the demand for hip and knee replacement in Otago, New 366 Zealand. NZMJ 2013;126;7–17.

19. Allen C, Hooper G, Frampton C. Do larger femoral heads improve the functional outcomes in total hip arthroplasty? J Arthoplasty (2014);29:401–4.

20. Statistics New Zealand data (www.stats.govt.nz)

21. Singleton N, Buddicom E, Vane A, Poutawera V. Are there differences between Māori and non-Māori patients undergoing primary total hip and knee arthroplasty surgery in New Zealand? A registry-based cohort study. NZMJ 2013;126:23–30.

Parental smoking during pregnancy: findings from the Growing Up in New Zealand cohort

Gayl Humphrey, Fiona Rossen, Natalie Walker, Chris Bullen

ABSTRACT

AIMS: To investigate patterns of exposure to tobacco smoke in pregnancy among a representative sample of New Zealand women.

METHODS: Analyses of smoking-related data from the first wave of the Growing Up in New Zealand cohort study, ie from the first data-collection point during the antenatal period in 2009–10.

RESULTS: Twenty percent of mothers reporting smoking before pregnancy and 9.9% of mothers continued during pregnancy. These figures were higher in younger women (p<.0001), women with lower educational achievement (p<.001) and Māori women (p<.001). Similarly, being Māori (p<.0001) and having a lower education achievement (p<.0029) were associated with smoking during an unplanned compared to a planned pregnancy. Multiparous mothers were more likely to be smokers than primaparous mothers (11%: 95% Confidence Interval [CI] 10.0–12.1 vs 8.3%: 95% CI 7.2–9.4). Second-hand smoke exposure was more common for younger women (Odds Ratio [OR] 3.2: 95% CI 1.6–6.4), Māori women (OR 1.9: 95% CI 1.4–2.5), and women with unplanned pregnancies (OR 3.4 95% CI 12.0–14.8).

CONCLUSIONS: There are differences in a range of contextual and behavioural factors related to smoking before and during pregnancy. Low educational achievement, being young, Māori and multiparous were all associated with smoking during pregnancy. A better understanding of why these differences exist is needed in order to find appropriate interventions to support women in becoming smoke-free.

Tobacco smoke exposure in pregnancy (maternal smoking and second-hand smoke [SHS] exposure) is one of the single most important preventable risks for maternal, fetal and infant health.[1–3] In this paper we present new data on tobacco smoke exposure in pregnancy for New Zealand women.

Maternal smoking during pregnancy

Smoking prevalence in the general population in developed countries has declined more rapidly in recent years compared to developing countries.[4] However, globally 22% of the world's adult population are estimated to be current smokers (36% men, 8% women).[5] A similar picture is evident in New Zealand, with the prevalence of current smoking declining from 25% in 1996/97 to 18% 2012/13.[6] However, the rates of decline in smoking prevalence has been slower for Māori and Pacific Peoples during this period.[6] Furthermore, there has been little change in smoking rates over time among pregnant women in New Zealand (19.5% in 2008 versus 18.4% in 2010[7]), particularly if they are Māori, aged 20 years and under, living in the most deprived areas and/or multiparous.[7,8]

Smoking during pregnancy is associated with a range of health risks for the baby and pregnancy, including adverse fetal development,[9–11] birth complications,[12,13] antepartum haemorrhage[14] and pre-term delivery.[15] Smoking during pregnancy also has deleterious effects on children in the early neonatal and preschool periods, with respiratory morbidity being more common.[16,17] Harms have also been reported to continue through the child's

life course into adulthood. For example, maternal smoking during pregnancy is associated with adolescent-onset of mental illness[18] and an increased risk in adulthood of obesity,[19] metabolic disorders[19] and cardiovascular disease.[3]

Exposure to SHS

Exposure of non-smokers to SHS is also associated with harms.[20] The 2008–2010 Global Adult Tobacco Survey found that almost one half (n=470 million) of reproductive-aged women (15–49 years) from 14 low- and middle-income countries were exposed to SHS in their homes.[21] At a global level it is estimated that of all deaths attributable to SHS, 28% occur in children and 47% in women.[22] When non-smoking pregnant women are exposed to SHS there is evidence of harmful effects on fetal development and on the health of the child, such as asthma, low birth weight and neural tube defects.[17,23–24] An increased risk of cardiovascular disease among adult offspring exposed to SHS during pregnancy and infancy has also been reported.[25] Higher exposure to SHS, both in the home and at work, is seen for those who are socioeconomically disadvantaged.[26] In US and Australia studies, women with lower educational achievement and from marginalised ethnicities are more exposed to SHS when pregnant than their counterparts.[27,28]

In New Zealand, non-smokers with the highest exposure to SHS are pre- and school aged children, Māori and those of low socioeconomic status.[6,29–30] Data on SHS exposure among pregnant New Zealand women are limited. Given the high rates of daily smoking among males aged 25–54 years,[31] and the high rates of smoking in the home,[29,32,33] it is highly likely that many non-smoking pregnant women are exposed to SHS. Exposure is likely to be disproportionally greater for Māori women, due to the higher proportion of Māori that smoke

and similarly for those experiencing high levels of deprivation. In a small study of pregnant Māori women who smoked (n=60), all lived with smokers and smoking was the norm among their Whānau, friends and co-workers.[34] Participants remarked that their environment made being smoke-free a difficult position to adopt.[34] Exposure to SHS in the home has almost halved between 2006/07 and 2012/2013 for New Zealand adults (7.5% to 3.7%) and for children aged 0–14 years (9.6% to 5%). The decrease experienced by Māori (adults 16% to 9.4%: children 18.9% to 9.2%) was substantial but not enough.[35] The Māori and Māori children are disproportionately affected in terms of disability adjusted life years (DALYs) due to SHS exposure.[35]

The Growing Up in NZ (GUiNZ) cohort study offers a unique opportunity to examine smoking behaviour and exposure to SHS over time. This paper focuses on the data reported at the first data-collection point (antenatal) which ended in June 2010. Our aim is to present the patterns of pregnancy and exposure to tobacco smoke in this cohort, to better understand the profile of smokers and the at-risk groups.

Methods

The methodology of GUiNZ is reported elsewhere[36] but in brief, GUiNZ is a longitudinal study that has recruited and collected information from pregnant mothers and their partners from before children are born. All participants had an expected delivery date between 25th April 2009 and 25th March 2010. In total, 6,822 pregnant women enrolled and completed a computer-assisted face-to-face antenatal interview. The cohort is comparable to the most recent New Zealand national birth statistics with regard to maternal age, ethnicity, parity and socioeconomic indicators.[37]

Figure 1: GUiNZ smoking questions for the mother.

Own smoking.	Exposure to SHS.
1. Did you smoke regularly—that is, every day—before you were aware you were pregnant? [Yes; no; don't know]	1. Does anyone currently regularly smoke in the same room as you? [Yes; no; don't know]
2. How many cigarettes did you smoke per day, on average, before this pregnancy?	2. How often? [Rarely (less than once a week); occasionally (a few times a week); often (almost or every day of the week); don't know]
3. Are you currently smoking? [Yes; no; don't know]	
4. How many cigarettes do you smoke per day, on average?	

Measurements

Smoking

The smoking questions specifically for the mother were used (Figure 1).

Ethnicity

Ethnicity was self-prioritised and coded into six Level 1 categories in line with Statistics New Zealand's coding criteria.[38] For the purpose of presenting smoking data, we combined the categories of MELAA and Other due to small numbers.

Social-economic position

Socio-economic deprivation was measured using the 2006 New Zealand Deprivation Index (NZDep2006) and area-level (neighbourhood) index constructed from nine Census 2006 variables (means-tested benefits; household income; home ownership; single-parent family; employment; qualifications; household overcrowding; access to a telephone and access to a car).[39,40] We aggregated summary deprivation scores as quintiles, with '1' representing the least deprived neighbourhoods and '5' the most deprived neighbourhoods. Highest educational qualification was coded as: no qualifications; secondary school completion; diploma/trade certification; bachelors' degree; or higher degree.

Statistical analyses

All statistical analyses used SAS version 9.3 (SAS Institute, Cary, Indiana,). We used descriptive statistics to examine associations between mothers' smoking with demographics, pregnancy period (before or during), planned/unplanned pregnancy and parity. Where multiple regression modelling was used, variables were entered only if they were significant covariates in univariate analyses. Outcomes with less than 10 people in each cell are not presented.

Results

In total, 1,946 mothers reporting smoking either before or during pregnancy—20.4% (n=1,387) smoked before pregnancy and 9.9% (n=559) reported that they smoked during pregnancy. In univariate analyses, being younger, Māori or Pacific, more deprived and less educated were all associated with smoking before and during pregnancy (Table 1).

NZMA
New Zealand Medical Association

Table 1: Mothers smoking pre- and during pregnancy by demographic characteristics.

Demographic characteristics		MOTHERS SMOKING							
		Before pregnancy[1]				During pregnancy[2]			
		n (N=6,807)	% (95% CI)	Odds ratio (95% CI)	p-value	n (N=5,664)	% (95% CI)	Odds ratio (95% CI)	p-value
Total		1,387	20.4 (19.4–21.3)	-	-	559	9.9 (9.1–10.6)	-	-
Age group	19 or less	190	57.9 (52.6–63.3)	4.4 (2.8–6.9)	<0.0001	85	31.1 (25.6–36.6)	1.7 (0.9–2.9)	<0.0001
	20–29	758	28.5 (26.8–30.2)	2.4 (1.6–3.5)		299	13.6 (12.2–15.1)	1.4 (0.9–2.3)	
	30–39	405	11.5 (10.4–12.5)	1.2 (0.8–1.7)		162	5.4 (4.6–6.2)	0.9 (0.5–1.4)	
	40 or older	34	12.1 (8.3–15.9)	1		13	6.4 (3.0–9.7)	1	
Ethnicity	Māori	444	46.8 (43.7–50.0)	2.2 (1.8–2.6)	<0.0001	236	31.6 (28.3–34.9)	3.1 (2.5–3.9)	<0.0001
	Pacific	316	31.7 (28.8–34.6)	1.1 (0.9–1.3)		98	13.5 (11.0–16.0)	1.0 (0.7–1.3)	
	Asian	35	3.5 (2.4–4.6)	0.2 (0.1–0.3)		-	-	-	
	MELAA & Other	18	11.5 (6.5–16.4)	0.6 (0.3–1.0)		-	-	-	
	New Zealand European	572	15.5 (14.3–16.7)	1		220	6.8 (5.9–7.7)	1	
NZDep2006	1 (least deprived)	106	9.7 (7.9–11.4)	1	<0.0001	31	3.3 (2.2–4.5)	1	<0.0001
	2	175	14.2 (12.2–16.1)	1.4 (1.1–1.9)		66	6.1 (4.7–7.5)	1.8 (1.1–2.7)	
	3	182	15.6 (13.5–17.7)	1.4 (1.0–1.8)		64	6.3 (4.8–7.8)	1.6 (1.0–2.5)	
	4	284	20.0 (17.9–22.0)	1.4 (1.1–1.9)		112	9.6 (7.9–11.2)	1.9 (1.3–3.0)	
	5 (most deprived)	640	34.0 (31.8–36.1)	2.0 (1.6–2.6)		286	19.5 (17.5–21.6)	2.9 (1.9–4.4)	
Highest education	No sec school qualification	284	58.0 (53.6–62.3)	1	<0.0001	143	40.6 (35.5–45.8)	1	<0.0001
	Sec school / NCEA 1–4	423	26.0 (23.9–28.2)	0.4 (0.3–0.5)		158	11.9 (10.2–13.7)	0.3 (0.2–0.4)	
	Diploma / Trade cert / NCEA 5–6	532	25.6 (23.7–27.5)	0.4 (0.4–0.6)		222	12.9 (11.3–14.5)	0.4 (0.3–0.5)	
	Bachelor's degree	101	6.6 (5.3–7.8)	0.1 (0.1–0.2)		21	1.6 (0.9–2.3)	0.1 (0.0–0.1)	
	Higher degree	41	3.9 (2.7–5.0)	0.1 (0.1–0.1)		13	1.4 (0.6–2.1)	0.1 (0.0–0.1)	

1. Relates to question: "Did you smoke regularly—that is every day—before you were aware you were pregnant?"
2. Relates to question: "Are you currently smoking?" NB: These results relate to mothers who were interviewed during pregnancy—mothers who were interviewed post-partum were excluded from these analyses.

Of the women who reported they were currently smoking (n=533), 40.1% (n=222) reported that they smoked ≤4 cigarettes per day (CPD), 31.1% (n=172) smoked between 5–9 CPD and 28.8% smoked ≥10 CPD. Given the small numbers in each subgroup, we used regression analyses to investigate differences between smoking <10 and ≥10 CPD (Table 2). The findings show that when all factors were controlled for, older women (aged 30–39 years; OR=0.7, 95% CI 0.2–2.6; p=0.0004) and being Māori (OR=1.2, 95% CI: 0.8–1.9, p<0.0001)) were associated with smoking ≥10 CPD.

Table 2: Average number of cigarettes smoked per day by demographic characteristics.[1]

Demographic characteristics		NUMBER OF CIGARETTES PER DAY				Odds ratio (95% CI)	p-value[3]
		9 or less[2]		10 or more			
		n (N=553)	% (95% CI)	n (N=553)	% (95% CI)		
Total		394	71.2 (67.5–75.0)	159	28.8 (25.0–32.5)	-	-
Age group	19 or less	70	84.3 (76.5–92.2)	13	15.7 (7.8–23.5)	0.2 (0.0–0.7)	0.0004
	20–29	216	73.0 (67.9–78.0)	80	27.0 (22.0–32.1)	0.4 (0.1–1.5)	
	30–39	101	62.7 (55.2–70.2)	60	37.3 (29.8–44.8)	0.7 (0.2–2.6)	
	40 or older	-	-	-	-	1	
Ethnicity	Māori	151	64.8 (58.7–71.0)	82	35.2 (29.0–41.3)	1.2 (0.8–1.9)	<.0001
	Pacific	82	84.5 (77.3–91.8)	15	15.5 (8.2–22.7)	0.3 (0.2–0.7)	
	Asian	-	-	0	-	-	
	MELAA & Other	-	-	0	-	-	
	New Zealand European	156	71.6 (65.6–77.6)	62	28.4 (22.4–34.4)	1	
NZDep2006	1 (least deprived)	24	77.4 (62.7–92.2)	-	-	1	0.37
	2	49	75.4 (64.9–85.9)	16	24.6 (14.1–35.1)	1.0 (0.4–2.9)	
	3	45	72.6 (61.4–83.7)	17	27.4 (16.3–38.6)	1.4 (0.5–3.9)	
	4	79	70.5 (62.1–79.0)	33	29.5 (21.0–37.9)	1.6 (0.6–4.2)	
	5 (most deprived)	197	69.6 (64.2–75.0)	86	30.4 (25.0–35.8)	1.9 (0.7–4.9)	

Table 2: Average number of cigarettes smoked per day by demographic characteristics.[1] (Continued.)

Highest education	No sec school qualification	86	61.0 (52.9–69.1)	55	39.0 (30.9–47.1)	1	0.0002
	Sec school / NCEA 1–4	133	84.2 (78.5–89.9)	25	15.8 (10.1–21.5)	0.3 (0.2–0.5)	
	Diploma / Trade cert / NCEA 5–6	149	68.0 (61.8–74.2)	70	32.0 (25.8–38.2)	0.6 (0.3–0.9)	
	Bachelor's degree	13	65.0 (44.0–86.0)	-	-	-	
	Higher degree	11	84.6 (64.9–100.0)	-	-	-	

These results relate to:

1. The question: 'How many cigarettes do you smoke per day, on average?'; mothers who indicated that they were <u>currently</u> smoking; and, mothers who were interviewed during pregnancy–others who were interviewed post-partum were excluded from these analyses.
2. As this question was only asked of mothers who indicated that they currently smoke, 'nine or less' includes those who responded 'zero'.
3. Outcome being modelled is '10 or more'.

Tables 3 and 4 present the unadjusted findings for the number of cigarettes smoked by planned or unplanned pregnancy and by parity. Among women with unplanned pregnancies unplanned smoking ≥10 CPD was more common (31.2%) than among women with planned pregnancies (20.7%) (Table 3) Table 4 shows that multiparous women were more likely to smoke ≥10 CPD (32.1%) than their primipara counterparts (22.6%, n=195).

Table 3: Number of cigarettes per day by planned/unplanned pregnancy.[1]

Maternal self-reported average daily cigarette consumption	Planned pregnancy		Unplanned pregnancy	
	n (N=135)	% (95% CI)	n (N=414)	% (95% CI)
9 or less	107	79.3 (72.4–86.1)	285	68.8 (64.4–73.3)
10 or more	28	20.7 (13.9–27.6)	129	31.2 (26.7–35.6)

These results relate to:

1. The question: 'How many cigarettes do you smoke per day, on average?';
2. Mothers who indicated that they were currently smoking; and,
3. Mothers who were interviewed during pregnancy—mothers who were interviewed post-partum were excluded from these analyses.

Table 4: Number of cigarettes per day by parity.[1]

Maternal self-reported average daily cigarette consumption	First pregnancy		Subsequent pregnancies	
	n (N=195)	% (95% CI)	n (N=358)	% (95% CI)
9 or less	151	77.4 (71.6–83.3)	243	67.9 (63.0–72.7)
10 or more	44	22.6 (16.7–28.4)	115	32.1 (27.3–37.0)

These results relate to:

1. The question: 'How many cigarettes do you smoke per day, on average?';
2. Mothers who indicated that they were currently smoking; and,
3. Mothers who were interviewed during pregnancy - mothers who were interviewed post-partum were excluded from these analyses.

Planned versus unplanned pregnancy on continued smoking

Smoking before pregnancy was greater when that pregnancy was unplanned, particularly for younger women (p<.0001), those with lower education achievement (p<.001) and Māori women (p<.001).

After adjusting for all covariates in the regression analysis, being Māori (p<.0001) and having lower education achievement (p<.0029) were found to be significantly associated with continuing to smoke during an unplanned pregnancy compared to planned, while age was less important (p<.015) (Table 5).

Table 5: Mothers smoking <u>during</u> pregnancy by planned/unplanned pregnancy.

Demographic characteristics		DURING pregnancy[1]				Odds ratio (95% CI)	p-value[2]
		Planned		Unplanned			
		n (N=3,488)	% (95% CI)	n (N=2,156)	% (95% CI)		
Total		135	3.9 (3.2–4.5)	420	19.5 (17.8–21.2)	-	-
Age group	19 or less	8	9.4 (3.2–15.6)	77	90.6 (84.4–96.8)	2.6 (0.7–10.8)	0.01
	20–29	73	24.7 (19.8–29.6)	223	75.3 (70.4–80.2)	0.7 (0.2–2.4)	
	30–39	50	31.1 (23.9–38.2)	111	68.9 (61.8–76.1)	0.7 (0.2–2.4)	
	40 or older	4	30.8 (5.7–55.9)	9	69.2 (44.1–94.3)	1	
Ethnicity	Māori	30	12.8 (8.5–17.0)	205	87.2 (83.0–91.5)	3.4 (2.1–5.4)	<.0001
	Pacific	26	26.8 (18.0–35.6)	71	73.2 (64.4–82.0)	1.3 (0.8–2.2)	
	Asian	0	-	-	-	-	
	MELAA & Other	-	-	0	-	-	
	New Zealand European	77	35.3 (29.0–41.7)	141	64.7 (58.3–71.0)	1	
NZDep2006	1 (least deprived)	10	32.3 (15.8–48.7)	21	67.7 (51.3–84.2)	1	0.16
	2	19	30.2 (18.8–41.5)	44	69.8 (58.5–81.2)	0.8 (0.3–2.0)	
	3	25	39.1 (27.1–51.0)	39	60.9 (49.0–72.9)	0.5 (0.2–1.3)	
	4	29	25.9 (17.8–34.0)	83	74.1 (66.0–82.2)	0.7 (0.3–1.8)	
	5 (most deprived)	52	18.2 (13.8–22.7)	233	81.8 (77.3–86.2)	1.1 (0.5–2.5)	
Highest education	No sec school qualification	23	16.2 (10.1–22.3)	119	83.8 (77.7–89.9)	1	0.002
	Sec school / NCEA 1–4	44	28.0 (21.0–35.1)	113	72.0 (64.9–79.0)	0.4 (0.3–0.8)	
	Diploma / Trade cert / NCEA 5–6	51	23.1 (17.5–28.6)	170	76.9 (71.4–82.5)	0.7 (0.4–1.2)	
	Bachelor's degree	10	50.0 (28.1–71.9)	10	50.0 (28.1–71.9)	0.2 (0.1–0.6)	
	Higher degree	-	-	7	-	-	

1. Relates to question: "Are you currently smoking?" NB: These results relate to mothers who were interviewed during pregnancy—mothers who were interviewed post-partum were excluded from these analyses.
2. Outcome being modelled is 'Unplanned pregnancy'.

Parity and Smoking

While the survey did not capture if mothers smoked during earlier pregnancies, Table 6 reports unadjusted smoking responses by parity status. There was little difference in smoking between the parity groups (first-born: 20.5%, 95% CI 19.0–22.0 vs subsequent: 20.3%, 95% CI 19.0–21.5) or during pregnancy (first-born: 8.3%, 95% CI 7.2-9.4 vs subsequent: 11%, 95% CI 10.0–12.1).

However, after controlling for age, ethnicity, deprivation and educational achievement; continuing to smoke during pregnancy was more common in multiparous women who were Māori and Pacific (Table 6).

Table 6: Mothers smoking <u>during</u> pregnancy by parity.

Demographic characteristics		DURING pregnancy[1]				Odds ratio (95% CI)	p-value[2]
		First-born		Subsequent			
		n (N=2,396)	% (95% CI)	n (N=3,268)	% (95% CI)		
Total		198	8.3 (7.2–9.4)	361	11.0 (10.0–12.1)	-	-
Age group	19 or less	67	78.8 (70.1–87.5)	18	21.2 (12.5–29.9)	0.0 (0.0–0.1)	<.0001
	20–29	100	33.4 (28.1–38.8)	199	66.6 (61.2–71.9)	0.2 (0.0–0.6)	
	30–39	30	18.5 (12.5–24.5)	132	81.5 (75.5–87.5)	0.6 (0.1–2.3)	
	40 or older	-	-	12	92.3 (77.8–100.0)	1	
Ethnicity	Māori	77	32.6 (26.6–38.6)	159	67.4 (61.4–73.4)	1.9 (1.2–2.9)	<.0001
	Pacific	27	27.6 (18.7–36.4)	71	72.4 (63.6–81.3)	2.2 (1.3–3.9)	
	Asian	0			-	-	
	MELAA & Other	-	-	-	-	-	
	New Zealand European	92	41.8 (35.3–48.3)	128	58.2 (51.7–64.7)	1	
NZDep2006	1 (least deprived)		-	24	77.4 (62.7–92.1)	1	0.16
	2	25	37.9 (26.2–49.6)	41	62.1 (50.4–73.8)	0.5 (0.2–1.4)	
	3	24	37.5 (25.6–49.4)	40	62.5 (50.6–74.4)	0.9 (0.3–2.7)	
	4	46	41.1 (32.0–50.2)	66	58.9 (49.8–68.0)	0.4 (0.1–1.1)	
	5 (most deprived)	96	33.6 (28.1–39.0)	190	66.4 (61.0–71.9)	0.6 (0.2–1.5)	

Table 6: Mothers smoking <u>during</u> pregnancy by parity. (Continued.)

Highest education	No sec school qualification	39	27.3 (20.0–34.6)	104	72.7 (65.4–80.0)	1	<.0001
	Sec school / NCEA 1–4	65	41.1 (33.5–48.8)	93	58.9 (51.2–66.5)	0.4 (0.2–0.6)	
	Diploma / Trade cert / NCEA 5–6	81	36.5 (30.2–42.8)	141	63.5 (57.2–69.8)	0.3 (0.2–0.5)	
	Bachelor's degree	-	-	16	76.2 (58.0–94.4)	0.3 (0.1–0.9)	
	Higher degree	-	-	-	-	-	

1. Relates to question: "Are you currently smoking?" NB: These results relate to mothers who were interviewed during pregnancy—mothers who were interviewed post-partum were excluded from these analyses.
2. Outcome being modelled is 'subsequent pregnancy'.

Exposure to SHS

Seven percent of the 5,664 women reported being exposed to SHS from someone smoking in the same room. For planned pregnancies, someone else smoking in the same room as the mother was substantively less (3%, 95% CI 2.4–3.6) than for unplanned pregnancies (13.4%, 95% CI 12.0–14.8). However, when parity was examined irrespective of planned or unplanned, no difference was apparent, (primipara mothers: 8.1%, CI 7.0–9.1 versus multiparous mothers: 6.2%, 95% CI 5.3–7.0).

Adjusting for age, ethnicity, deprivation and educational status, being ≤ 19 years of age (OR 3.2, 95% CI 1.6–6.4; p<.0001), being Māori (OR 1.9: 95% CI: 1.4–2.5; p<.0001), living in an area of high deprivation (OR 3.5 CI: 2.0–5.7; p<.0001) and having a low educational achievement (p<.0001) were significantly associated with mothers reporting having someone smoking in the same room as them (Table 7).

Table 7: Exposure to SHS by demographic characteristics.

Demographic characteristics		EXPOSURE TO SECOND-HAND SMOKE DURING PREGNANCY				Odds ratio (95% CI)	p-value[2]
		Yes		No			
		n	% (95% CI)	n	% (95% CI)		
Total		394	7.0 (6.3–7.6)	5,270	93.0 (92.4–93.7)	-	-
Age group	19 or less	77	28.2 (22.9–33.5)	196	71.8 (66.5–77.1)	3.2 (1.6–6.4)	<0.0001
	20–29	228	10.4 (9.1–11.7)	1,967	89.6 (88.3–90.9)	1.6 (0.9–3.0)	
	30–39	79	2.6 (2.1–3.2)	2,913	97.4 (96.8–97.9)	0.6 (0.3–1.2)	
	40 or older	10	4.9 (1.9–7.9)	194	95.1 (92.1–98.1)	1	
Ethnicity	Māori	135	18.1 (15.3–20.8)	612	81.9 (79.2–84.7)	1.9 (1.4–2.5)	<0.0001
	Pacific	84	11.6 (9.2–13.9)	642	88.4 (86.1–90.8)	1.1 (0.8–1.5)	
	Asian	27	3.4 (2.1–4.6)	775	96.6 (95.4–97.9)	0.7 (0.5–1.1)	
	MELAA & Other	-	-	134	96.4 (93.3–99.5)	0.6 (0.3–1.6)	
	New Zealand European	142	4.4 (3.7–5.1)	3,100	95.6 (94.9–96.3)	1	

Table 7: Exposure to SHS by demographic characteristics. (Continued.)

NZDep2006	1 (least deprived)	18	1.9 (1.0–2.8)	918	98.1 (97.2–99.0)	1	<0.0001
	2	38	3.5 (2.4–4.6)	1,042	96.5 (95.4–97.6)	1.6 (0.9–2.9)	
	3	49	4.9 (3.5–6.2)	960	95.1 (93.8–96.5)	1.8 (1.0–3.2)	
	4	78	6.7 (5.2–8.1)	1,094	93.3 (91.9–94.8)	2.0 (1.1–3.4)	
	5 (most deprived)	211	14.4 (12.6–16.2)	1,254	85.6 (83.8–87.4)	3.4 (2.0–5.7)	
Highest education	No sec school qualification	94	26.7 (22.1–31.3)	258	73.3 (68.7–77.9)	1	<0.0001
	Sec school / NCEA 1–4	113	8.5 (7.0–10.0)	1,213	91.5 (90.0–93.0)	0.4 (0.3–0.6)	
	Diploma / Trade cert / NCEA 5–6	144	8.4 (7.1–9.7)	1,573	91.6 (90.3–92.9)	0.5 (0.3–0.7)	
	Bachelor's degree	31	2.4 (1.5–3.2)	1,277	97.6 (96.8–98.5)	0.2 (0.1–0.3)	
	Higher degree	-	-	941	99.1 (98.4–99.7)	0.1 (0.0–0.2)	

These results relate to:
1. The question: 'Does anyone currently regularly smoke in the same room as you?'; Mothers who were interviewed during pregnancy—mothers who were interviewed post-partum were excluded from these analyses.
2. Outcome being modelled is 'Does anyone currently regularly smoke in the same room as you?—Yes''.

Discussion

Being younger (<20 years), being less well educated and living in an area of high deprivation continue to be strongly related to smoking before and during pregnancy. These factors are similar to those reported internationally[41,42,43] and nationally.[7,8] The finding that multiparous women were more likely to continue to smoke during pregnancy and smoke more CPD than primipara women has also been previously reported.[44] While first time pregnancy appears to be a motivator for smoking cessation, it does not seem to hold true for multiparous women. This finding has also been reported before for New Zealand,[7] and may be related to both smoking behaviour (eg being more cigarette dependent) and contextual factors (eg less social support, financial pressures and low self-confidence).[45] Understanding these factors is important, as this group is highly likely to be contributing to the wider family's (including older children) exposure to SHS, as well as their unborn child.

A planned pregnancy was positively associated with not smoking during pregnancy or if still smoking, a lower consumption of cigarettes (<9 CPD). This finding may signal that women (and families) may have planned a wider "healthy" strategy which included smoking cessation when planning to start or add to their family. It is not known if these women (and families) also have greater and/or earlier interactions with health professionals and as such are exposed to early cessation advice, support and treatment. Until relatively recently, cutting down rather than quitting smoking was the dominant message to pregnant smokers by health professionals.[46,47]

While there is a high awareness of the harms of smoking on themselves and their unborn child, the lived context of the pregnant women plays a large part in smoking cessation. It is not possible to determine who actively cut down their CPD in this study but research suggests that adoption of a cutting down approach versus quitting is more common in women with low educational achievement and living in areas of greater deprivation.[46,48,49] It is critical that a consistent message and a subsequent supportive environment is provided if changes to these rates are to happen. It will also be important to explore the smoking data in subsequent GUiNZ waves, as international research suggests

women often resume smoking in the days or weeks following the birth of their child.[7,50]

Second Hand Smoke

Wider social contexts (friends, family, work) are important factors in supporting or impeding behavioural change activities.[51] While only 7% of our cohort reported another person smoking in the same room, this finding was correlated with being younger, living in the most deprived area, lower educational achievement and Māori. Once again, understanding these contexts in more detail is important for intervention strategies to be successful. Exploration of the GUiNZ partner responses and the other contextual details captured in GUiNZ data (including stressors) is needed. It is clear that New Zealand's current smoke-free strategies are not proving to be as effective for multiparous women and it is unclear why this is so. More in-depth qualitative research is needed to explore their motivations and situational contexts. Such research will help identify where additional interventions could be focused so as to reduce the burden of SHS on other children living at home.[52]

Equity

The impact of high rates of smoking is evident for Māori mortality and health-related outcomes across the life course from the new-born through to adulthood.[35,53] Māori reportedly receive antenatal care later in pregnancy.[7] In repeated smoking surveys, Māori youth report having their first cigarette significantly earlier than their non-Māori counterparts, and smoking prevalence in young Māori females (15–24 years) was significantly higher than for non-Māori.[54] This finding may partly account for the low smoking cessation rate for Māori during pregnancy.[34] However it does not explain the contextual drivers that influenced earlier initiation or higher consumption. Arguably, it will not be until a deeper understanding of this issue is obtained, that effective interventions can emerge.

It is clear that efforts to support young Māori and Pacific women at their first pregnancy to quit are pivotal, as both groups were positively associated with smoking during subsequent pregnancies. The ability to act on information given about smoking in pregnancy has been reported as low by Māori women.[47,55] This finding should emphasise that the effectiveness of the current suite of interventions is suboptimal for pregnant Māori women regardless of parity, and new strategies are needed to reduce significant life course harms.

Interventions

There is a bourgeoning literature on the effectiveness of cessation activities for the general smoking population. However, there is less research on smoking cessation interventions for pregnant women (prima and multiparious) and indigenous populations. Indigenous research by Glover et al[56,57] and Walker et al[33] have set some of the ground work for identifying successful directions for smoking cessation interventions, such as coaching models and using incentives as motivators for change. It is clear that more work is urgently needed to evaluate these and other data in more detail, as it remains unclear why some of these strategies work and others do not.

A Cochrane review specifically examining smoking cessation interventions for indigenous populations concluded that more rigorous trials are required to bridge the gap between tobacco-related health disparities in Indigenous and non-Indigenous populations.[58] Another Cochrane Review[59] reported that using a mix of interventions was most effective in helping pregnant women that smoke to quit, and highlighted the positive findings around the use of incentives. Use of incentives is a strategy rarely used in New Zealand, but is showing promise particularly for younger Māori mothers (ie <30 years)[60] and with one context of a team competition.[60] However, the cost-effectiveness of such strategies is unclear.

Engaging with mothers and families early in their antenatal care in another strategy that shows promise in the New Zealand context, in reducing smoking rates.[50] While New Zealand has a significant array of smoking cessation intervention programmes based on and contributing to the evidence base, there are few that have a specific focus on pregnant women.[61] Evaluations of these programmes appear to be scarce in the published and grey literature, or are small in scale and duration[62] and as such are often not widely adopted. While not specific to mothers, a study of the awareness and perceived effectiveness of smoking cessation services for those living in high deprivation areas in New Zealand, reported these to

be low.[63] This finding is important as our findings show that being Māori and living in high deprivation areas were associated with smoking during pregnancy.

Conclusion

Reducing maternal tobacco smoke exposure has the potential to have a positive health effect that far exceeds the immediate health of both mother and infant. There is a paucity of local evidence on the effectiveness of smoking cessation interventions for Maori women. Without effective interventions to reduce tobacco smoke exposure in pregnancy, intergenerational health equalities will be come more entrenched.

Author information:

Gayl Humphrey, Co-Lead Health Information and Technology Programme and Research Fellow, National Institute for Health Innovation, University of Auckland, Auckland; Fiona Rossen, Senior Research Fellow, Growing Up in New Zealand, University of Auckland, Auckland; Natalie Walker, Programme Leader, National Institute for Health Innovation, and Member of Centre for Addiction Research, University of Auckland, Auckland; Chris Bullen, Director, National Institute for Health Innovation, and Member of Centre for Addiction Research, University of Auckland, Auckland.

Corresponding author:

Gayl Humphrey, National Institute for Health Innovation, University of Auckland, Private Bag 92019, Auckland.

g.humphrey@auckland.ac.nz

URL:

https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7019

REFERENCES:

1. Al-Sahab B, Saqib M, Hauser G, Tamim H. Prevalence of smoking during pregnancy and associated risk factors among Canadian women: a national survey. BMC Pregnancy and Childbirth. 2010;10(1):24.

2. Castles A, Adams EK, Melvin CL, Kelsch C, et al. Effects of smoking during pregnancy: Five meta-analyses. American Journal of Preventive Medicine. 1999;16(3):208–15.

3. Mamun AA, O'Callaghan MJ, Williams GM, Najman JM. Maternal Smoking During Pregnancy Predicts Adult Offspring Cardiovascular Risk Factors – Evidence from a Community-Based Large Birth Cohort Study. PLoS ONE. 2012;7(7):e41106.

4. OECD. OECD Factbook 2013. 2013.

5. WHO. Geneva: World Health Organization. 2013.

6. Ministry of Health. Tobacco Use 2012/13: New Zealand Health Survey. Wellington Ministry of Health 2014.

7. Andrews A, Dixon L, Eddy A, Guillialand K, et al. Smoking prevalence trends: An analysis of smoking at pregnancy registration and at discharge from a midwife Lead Maternity Carer - 2008 to 2010. New Zealand College of Midwives Journal. 2014;49:17–22.

8. Ministry of Health. Report on Maternity 2012. Wellington: Ministry of Health 2015.

9. Cornelius MD, Day NL. Developmental consequences of prenatal tobacco exposure. Current Opinion in Neurology. 2009;22(2):121–5.

10. Bouwland-Both MI, van Mil NH, Tolhoek CP, Stolk L, et al. Prenatal parental

tobacco smoking, gene specific DNA methylation, and newborns size: the Generation R study. Clin Epigenetics. 2015;7(1).

11. Koshy G, Delpisheh A, Brabin BJ. Dose response association of pregnancy cigarette smoke exposure, childhood stature, overweight and obesity. Eur J Public Health. 2011;21(3):286–91.

12. Blatt K, Moore E, Chen A, Van Hook J, et al. Association of reported trimester-specific smoking cessation with fetal growth restriction. Obstet Gynecol. 2015;125(6):1452–9.

13. Leite M, Albieri V, Kjaer S, Jensen A. Maternal smoking in pregnancy and risk for congenital malformations: Results of a Danish register-based cohort study. Maternal Child Health Journal 2014;19:1010–2.

14. Van Altvorst M, Chan E, Taylor R, Kenny L, et al. Antepartum haemorrhage of unknown origin and maternal cigarette smoking beyond the first trimester. Australian and New Zealand Journal of Obstetrics and Gynaecology. 2012;52:161–6.

15. Mei-Dan E, Walfisch A, Weisz B, Hallak M, et al. The unborn smoker: Association between smoking during pregnancy and adverse perinatal outcomes. J Perinat Med. 2015;43(5):553–8.

16. Neuman Å, Hohmann C, Orsini N, Pershagen G, et al. Maternal Smoking in Pregnancy and Asthma in Preschool Children: A Pooled Analysis of Eight Birth Cohorts. American Journal of Respiratory and Critical Care Medicine. 2012;186(10):1037–43.

17. Burke H, Leonardi-Bee J, Hashim A, Pine-Abata H,

et al. Prenatal and passive smoke exposure and incidence of asthma and wheeze: systematic review and meta-analysis. Pediatrics. 2012;129(4):735–44.

18. Mitrou F, Gaudie J, Lawrence D, Silburn SR, et al. Antecedents of hospital admission for deliberate self-harm from a 14-year follow-up study using data-linkage. BMC Psychiatry. 2010;10.

19. Ino T. Maternal smoking during pregnancy and offspring obesity: meta-analysis. Pediatrics international : official journal of the Japan Pediatric Society. 2010;52(1):94–9.

20. Öberg M, Jaakkola MS, Woodward A, Peruga A, et al. Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. The Lancet Respiratory Medicine. 2010;6736(10):61388–8.

21. Centers for Disease Control and Prevention Current tobacco use and second-hand smoke exposure among women of reproductive age - 14 countries, 2008-2010. Morbidity and Mortality Weekly Report 2012;61 877–82.

22. Öberg M, Woodward A, Jaakkol MS, Peruga A, et al. Global estimate of the burden of disease from second-hand smoke. Geneva: World Health Organization; 2010.

23. Wang M, Wang ZP, Zhang M, Zhao ZT. Maternal passive smoking during pregnancy and neural tube defects in offspring: A meta-analysis. Archives of Gynecology and Obstetrics. 2014;289(3):513–21.

24. Hawsawi AM, Bryant LO, Goodfellow LT. Association between exposure to secondhand smoke

during pregnancy and low birthweight: A narrative review. Respiratory Care. 2015;60(1):135–40.

25. West HW, Gall SL, Juonala M, Magnussen CG. Is Passive Smoking Exposure in Early Life a Risk Factor for Future Cardiovascular Disease? Current Cardiovascular Risk Reports. 2015;9(9).

26. WHO. Environmental health inequalities in Europe: Assessment Report. Geneva: World Health Organisation; 2012. Report No.: ISBN 978 92 890 0260 8.

27. Hawkins SS, Dacey C, Gennaro S, Keshinover T, et al. Secondhand smoke exposure among nonsmoking pregnant women in New York City. Nicotine Tob Res. 2014;16(8):1079–84.

28. Bonevski B, Paul C, Jones A, Bisquera A, et al. Smoky homes: gender, socioeconomic and housing disparities in second hand tobacco smoke (SHS) exposure in a large population-based Australian cohort. Prev Med. 2014;60:95–101.

29. Thomson G, Wilson W, P H-C. Smoky homes: a review of the exposure and effects of secondhand smoke in New Zealand homes. New Zealand Medical Journal 2005;118(1213):1404–15.

30. Glover M, Kira A, Cowie N, Wong R, et al. Health consequences of tobacco use for Māori—cessation essential for reducing inequalities in health. New Zealand Medical Journal. 2013;126(1379).

31. Statistics NZ. Tobacco smoking. online: Statistics New Zealand; 2014.

32. Glover M, Hadwen G, Chelimo C, Scragg R, et al.

Parent versus child reporting of tobacco smoke exposure at home and in the car. The New Zealand Medical Journal (Online). 2013;126(1375):37–47.

33. Walker N, Johnston V, Glover M, Bullen C, et al. Effect of a family-centered, secondhand smoke intervention to reduce respiratory illness in indigenous infants in Australia and New Zealand: A randomized controlled trial. Nicotine Tob Res. 2015;17(1):48–57.

34. Glover M, Kira A. Why Māori women continue to smoke while pregnant. New Zealand Medical Journal. 2011;124(1339):22–31.

35. Mason K, Borman B. Burden of disease from second-hand smoke exposure in New Zealand. NZMJ. 2016;129(1432).

36. GUiNZ. http://www.growingup.co.nz/en/about-the-study.html [cited 2015]. Available from: http://www.growingup.co.nz/en/about-the-study.html

37. Morton SMB, Ramke J, Kinloch J, Grant CC, et al. Growing Up in New Zealand cohort alignment with all New Zealand births. Australian and New Zealand Journal of Public Health. 2014;39(1):82–7.

38. Statistics New Zealand. Statistical standard for ethnicity [Internet]. Wellington: Statistics New Zealand; 2005 [cited 15 June 2010]. Available from: http://www2.stats.govt.nz/domino/external/web/carsweb.nsf/55d63ae38ba3a25e-4c2567e6007f6686/35d-9b7e17a1d6151c-c25701100031353?OpenDocument

39. Salmond C, Crampton P, Atkinsonn J. NZDep2006 Index of Deprivation.

University of Otago.: Department of Public Health,; 2007.

40. White P, Gunston J, Salmond C, Atkinson J, et al. Atlas of Socioeconomic Deprivation in New Zealand NZDep2006. Wellington: Ministry of Health; 2008.

41. Cui Y, Shooshtari S, Forget EL, Clara I, et al. Smoking during Pregnancy: Findings from the 2009–2010 Canadian Community Health Survey. PLoS ONE. 2014;9(1):e84640.

42. Jaddoe VW, Troe EJ, Hofman A, Mackenbach JP, et al. Active and passive maternal smoking during pregnancy and the risks of low birthweight and preterm birth: the Generation R Study. Paediatric and perinatal epidemiology. 2008;22(2):162–71.

43. Tabb KM, Huang H, Menezes PR, Azevedo e Silva G, et al. Ethnic differences in tobacco use during pregnancy: findings from a primary care sample in São Paulo, Brazil. Ethnicity & Health. 2014;20(2):209–17.

44. Mohsin M, Bauman A. Socio-demographic factors associated with smoking and smoking cessation among 426,344 pregnant women in New South Wales, Australia. BMC Public Health. 2005;5:138.

45. Meernik C, Goldstein AO. A critical review of smoking, cessation, relapse and emerging research in pregnancy and post-partum. British Medical Bulletin. 2015;114(1):135–46.

46. Graham H, Flemming K, Fox D, Heirs M, et al. Cutting down: insights from qualitative studies of smoking in pregnancy. Health & Social Care in the Community. 2014;22(3):259–67.

47. Glover M, Paynter J, Bullen C, Kristensen K. Supporting pregnant women to quit smoking: postal survey of NZ general practitioners and midwives' smoking cessation knowledge and practices. The New Zealand Medical Journal (Online). 2008;121(1270):53–64.

48. Greaves L. The meanings of smoking to women and their implications for cessation. International Journal of Environmental Research and Public Health. 2015;12(2):1449–65.

49. Pledger AB. Exploring the experiences of pregnant women using an NHS stop smoking service: A qualitative study. Perspect Public Health. 2015;135(3):138–44.

50. Barnes HB, Barnes AM, Baxter J, Crengle S, et al. Hapu Ora: Wellbeing in the Early Stages of Life. Auckland: Whariki Resaerch Group and SHORE, Massey University; 2013.

51. Hitchman SC, Fong GT, Zanna MP, Thrasher JF, et al. The relation between number of smoking friends, and quit intentions, attempts, and success: Findings from the International Tobacco Control (ITC) Four Country Survey. Psychology of Addictive Behaviors. 2014;28(4):1144–52.

52. Pisinger C, Hammer-Helmich L, Andreasen A, Jorgensen T, et al. Social disparities in children's exposure to second hand smoke at home: a repeated cross-sectional survey. Environmental Health. 2012;11(1):65.

53. Ministry of Health. Māori Health Chart Book 2015 (3rd edition). Wellington: Ministry of Health; 2015.

54. Ministry of Health. Māori Smoking and Tobacco Use 2011. Wellington Ministry of Health; 2011.

55. Glover M, Nosa V, Gentles D, Watson D, et al. Do New Zealand Māori and Pacific 'walk the talk' when it comes to stopping smoking? A qualitative study of motivation to quit. Journal of Smoking Cessation. 2014;9(2):68–75.

56. Glover M, Cowie N. Increasing delivery of smoking cessation treatments to Māori and Pacific smokers. The New Zealand Medical Journal (Online). 2010;123(1308).

57. Glover M, Kira A, Walker N, Bauld L. Using Incentives to Encourage Smoking Abstinence Among Pregnant Indigenous Women? A Feasibility Study. Matern Child Health J. 2015;19(6):1393–9.

58. Carson KV, Brinn MP, Peters M, Veale A, et al. Interventions for smoking cessation in Indigenous populations. The Cochrane database of systematic reviews. 2012;1:CD009046.

59. Chamberlain C, O'Mara-Eves A, Oliver S, Caird Jenny R, et al. Psycho-social interventions for supporting women to stop smoking in pregnancy. Cochrane Database of Systematic Reviews. 2013.

60. Glover M, Kira A, Gentles D, Cowie N, et al. The WERO group stop smoking competition: main outcomes of a pre- and post- study. BMC Public Health. 2014;14:599.

61. Aukati Kaipaipa: Hapu Mama. http://www.aukatikaipaipa.conz/2016.

62. Jakob-Hoff M, Fa'alau F, Spee K, Postlethwaite J. Smokefree Pregnancy Incentives Project Final Evaluation Report. Counties Manukau: Counties Manukau DHB; 2015.

63. Cowie N, Glover M, Scragg R, Bullen C, et al. Awareness and perceived effectiveness of smoking cessation treatments and services among New Zealand parents resident in highly deprived suburbs. The New Zealand Medical Journal (Online). 2013;126(1378):48–59.

Low FODMAP diet efficacy in IBS patients—what is the evidence and what else do we need to know?

Tim Kortlever, Clarice Hebblethwaite, Julie Leeper, Leigh O'Brien, Chris Mulder, Richard B Gearry

ABSTRACT

Irritable Bowel Syndrome (IBS) is a common and significant health problem which may be treatable with dietary interventions. Here we aim to explain the principles of the low Fermentable Oligo-, Di-, Monosaccharides and Polyol diet, and discuss both the limitations and opportunities of the diet in those with IBS, a common cause of presentation to primary and secondary care in New Zealand.

Background

Irritable Bowel Syndrome (IBS) is a functional gastrointestinal disorder characterised by abdominal pain, altered bowel habits (diarrhoea or constipation) and bloating. The Rome III criteria are the current standard test for the diagnosis of IBS being validated and used frequently (Figure 1).[7] In industrialised countries, IBS is one of the most common gastrointestinal disorders presenting to general practitioners (GPs). IBS prevalence in New Zealand is 10–18%, with a higher prevalence in women.[1] The highest rates of IBS are seen in those less than 50 years of age.[2] Data on the distribution of IBS among different ethnic and age groups in New Zealand are not available.

While not requiring major medical or surgical interventions, the impact of IBS on quality of life is significant. Health-related quality of life is reduced in IBS patients who present to doctors,[3] more so than controls and to similar levels as individuals with diabetes, end stage renal failure and gastroesophageal reflux disease.[4] Furthermore, comorbidities such as anxiety and depression are more common in IBS patients.[5] The impact on daily activities is significant and it is estimated that the yearly direct costs of IBS may range up to NZ$1,000 (or around US$750) per patient.[6] Indirect costs, such as absence from work and impaired productivity, are more difficult to calculate but have been acknowledged as substantial.[6]

Figure 1:[7]

Diagnosis of Irritable Bowel syndrome is based on the following criteria:

- A patient must have recurrent abdominal pain or discomfort for at least three days/month in the last three months with symptom onset at least six months prior to diagnosis
- Abdominal pain or discomfort must be associated with at least two of the following:
1. Improvement with defaecation
2. Onset associated with change in frequency in stool
3. Onset associated with change in form (appearance) of stool

---

Although the pathophysiology of IBS is unclear, a number of putative mechanisms have been identified. These include visceral hypersensitivity, brain-gut axis dysregulation, altered gastrointestinal microbiota, increased gut mucosal immune activity and the direct action of specific food chemicals.[7] Visceral hypersensitivity has received significant attention. Functional MRI brain studies, for example, support the importance of this mechanism.[8] However, without a clear understanding of IBS aetiology and pathogenesis, a symptom-based approach has been adopted to palliate symptoms. Current therapeutic options include pharmaceuticals (eg antidepressants, antispasmodics, laxative agents or 5-HT$_3$ receptor antagonists), fibre supplements, regular exercise, probiotics, psychological and dietary therapy.[7] Often simple pharmacotherapy may be safe and effective but many treatments only achieve partial relief and may have serious adverse effects. For example, laxatives improve stool frequency and consistency in patients with constipation-predominant IBS, but do not have an effect on abdominal pain or bloating.[7] Furthermore, antidepressants may improve abdominal pain, but may have side effects and are, therefore, not suitable for all IBS patients.[7]

Over the past decade, a new dietary therapy has emerged with convincing results in multiple clinical trials. In studies from a range of populations, up to three-quarters of IBS patients experience symptom improvement when adhering to a diet low in Fermentable Oligo-, Di-, Monosaccharides and Polyols (FODMAPs).[9–12] Key symptoms such as abdominal pain, bloating, stool frequency and flatulence improve significantly in most IBS patients adherent to this diet. Restricting certain short-chain carbohydrates from the diet involves guidance from skilled dietitians to ensure that the diet is nutritionally adequate and balanced, and presented in a practical way.

Here we aim to explain the principles of the low FODMAP diet and to discuss both the limitations and opportunities of the diet in those with IBS, a common cause of presentation to primary and secondary care in New Zealand.
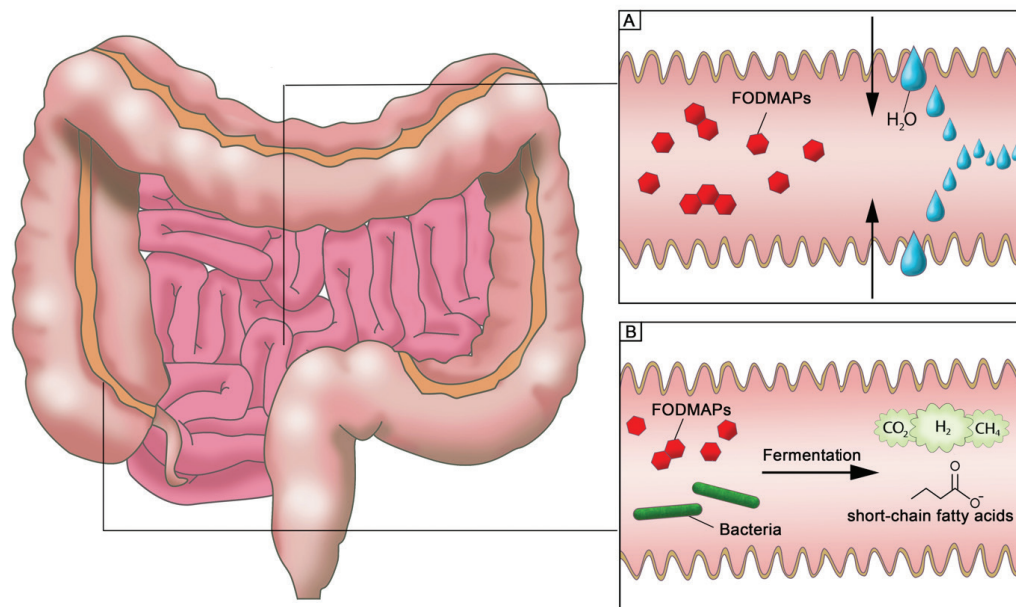
The low FODMAP diet
Origin and mechanisms

Up to 84% of patients with IBS attribute specific foods as triggers of their abdominal symptoms.[13] Up to 70% of all patients believe that incompletely absorbed carbohydrates may be associated with their GI symptoms.[13] This view is supported by observations of symptom improvement after dietary exclusion of solitary short-chain carbohydrates such as: fructose, lactose, fructans and sorbitol.[14] A number of these carbohydrates even share an additive effect, increasing the severity of symptoms when ingested together.[12] In the last decade, a restriction diet that incorporates these and other carbohydrates, the low FODMAP diet, has been developed.

The key characteristics shared by foods that are restricted in the low FODMAP diet are, firstly, that they are osmotically active.[15] Secondly, they are malabsorbed due to the absence (relative or absolute) of specific hydrolases to effectively digest them or due to an absorption rate that is slower than intestinal transit time.[16] Consequently, these molecules remain in the small intestine, attracting water into the gastrointestinal tract via osmosis (Figure 2A). FODMAPs then pass into the colon where they are fermented by the colonic microbiota. FODMAP fermentation releases gases (primarily hydrogen, carbon dioxide and in some cases methane) and short-chain fatty acids (Figure 2B).

Small intestinal water accumulation following high FODMAP consumption has been confirmed by imaging and experimental studies.[15,17] In one study, twelve ileostomates consumed a low and high FODMAP diet in a randomised, single-blinded cross-over study. Effluent weight, dry weight and volume increased by approximately 20% on the high FODMAP diet.[15] A MRI study by Murray et al found an increase in small bowel water content (SBWC) after ingestion of large amounts of fructose.[17] The addition of glucose lessened the increase in SBWC via co-transport of glucose and fructose.

The fermentation of FODMAPs by intestinal microbiota has been studied by Ong

Figure 2: The effects of FODMAPs in the small (A) and large intestine (B). Subsequent luminal distention caused by an increased luminal volume may trigger symptoms in patients with visceral hypersensitivity, including: abdominal pain or discomfort, diarrhoea, constipation and bloating.

et al, who found that both IBS patients and healthy subjects produced more breath hydrogen while consuming a high rather than low FODMAP diet.[18] In addition, breath hydrogen levels of IBS patients were significantly higher than that of healthy subjects, suggesting differences in the microbiota composition and metabolism, as described previously.[19]

Increased small intestinal water delivery and colonic fermentation leads to luminal distension and a more liquid effluent. In IBS patients (who have visceral hypersensitivity) this may trigger symptoms such as abdominal pain and bloating, diarrhoea and wind. Constipation while digesting osmotically active carbohydrates may be explained by a slower transit time due to the production of methane gas in some individuals.[14]

Principles of the low FODMAP diet

A diet low in FODMAPs minimises foods with a high content of fermentable short-chain carbohydrates. These include fructose (in excess of glucose), lactose, fructans, galacto-oligosaccharadies and polyols. As the name suggests, the low FODMAP diet does not eliminate FODMAPs completely, but rather reduces gastrointestinal FODMAP concentration to an extent where symptoms are controlled. This threshold is, however, variable between individuals. Therefore, dietitians will advise a significant reduction in FODMAP containing foods with a subsequent re-introduction of specific FODMAPs until a symptom threshold is reached.

While there is no ability for most FODMAPs to be absorbed in the small intestine, lactose and fructose can be transported across the intestinal barrier in some individuals. Absorption of lactose, the natural sugar in milk and other dairy products, depends on the persistence of lactase enzymes in the brush border of the small intestinal mucosa. Lactase non-persistence is common after weaning in the majority of the global population. However, high lactase levels may persist to adulthood in some populations, especially those of Northern European descent.[20] In those with lactase non-persistence and IBS, lactose acts as a FODMAP leading to significant gastrointestinal symptoms.

Fructose may be absorbed in one of two ways.[16] Fructose is generally absorbed slowly via the low capacity GLUT5 transporter. However, this mechanism may be overwhelmed when there are larger quantities of fructose ingested over a short period (eg by drinking a glass of orange juice). Alternatively, when fructose and glucose are ingested simultaneously in

Table 1: Summary of the key studies of low FODMAP diet efficacy. IBS = Irritable Bowel Syndrome, LFD = low FODMAP diet, HFD = high FODMAP diet, NCGS = non-coeliac gluten sensitivity, LGG = Lactobacillus rhamnosus GG, GI = gastrointestinal.

Reference	Country	Cohorts	Methodology	Intervention and duration	Results	Limitations and remarks
Shepherd et al (2008)[12]	Australia	IBS patients with Fructose Malabsorption (n=25)	Randomised, double-blinded, quadruple arm, controlled rechallenge trial	2 week treatment arms with fructose, fructans, mix and glucose drinks each, whilst adherent to the LFD at all times	Symptoms were adequately controlled in the glucose drink arm (86%), but significantly worsened during each of the other arms: % of patients experiencing adequately controlled symptoms was 30%, 23% and 21% for the fructose, fructans and mixture drinks respectively.	All food was provided to the participants, cohort consisted only of patients who had a marked response to the LFD diet prior to recruitment.
Ong et al (2010)[18]	Australia	IBS patients (n=15) healthy subjects (n=15)	Randomised, single-blinded, controlled crossover trial	2 days HFD 2 days LFD (7 day washout period)	In IBS patients all symptoms (Abdominal pain, bloating, flatus, nausea, heartburn and tiredness) were significantly worse when adherent to the HFD. Healthy subject only had a significant increase in flatus with the HFD.	Short dietary window, all food was provided to the participants.
Staudacher et al (2011)[10]	UK	IBS patients on standard IBS dietary advice (NICE-guidelines) (n=39), IBS patients on LFD (n=42)	Prospective, controlled	2–6 months	Significantly more patients in the LFD group reported improvement in bloating, abdominal pain, nausea, energy levels and flatulence. Improvement in composite symptom score was greater in LFD than in the standard group (86% vs. 49%). More patients in the LFD group were satisfied with their symptom response (76% vs. 54% in the group standard group).	Standard dietary advice and LFD overlapped, adherence was not objectively measured, no control group.
De Roest et al (2013)[9]	New Zealand	IBS patients (n=90)	Prospective, uncontrolled	Mean 15.7 months	Key symptoms improved with the LFD. Most patients (72.1%) reported satisfaction with their symptoms. Most patients (75.6%) also were found adherent to the diet, which was associated with greater symptom improvement.	Variable follow-up duration, adherence was not objectively measured.
Halmos et al (2014)[11]	Australia	IBS patients (n=30), Healthy subjects (n=8)	Randomised, single-blinded, controlled crossover trial	21 days LFD 21 days Australian diet (21 day wash-out period)	Patients with IBS had lower overall symptoms scores while on LFD compared to the typical Australian diet and baseline. Symptoms were unaltered in the control group for both diets. A majority of IBS patients (70%) had an improvement in overall gastrointestinal symptoms that was considered clinically significant.	All food was provided to the participants.
Pedersen et al (2014)[24]	Denmark	IBS patients on LFD (n=42), LGG (n=41) and normal Danish diet (n=40), all supplemented with a web-based monitoring programme.	Randomised, unblinded, controlled trial	6 weeks	Significant improvement in symptom score of patients on the LFD compared to patients on the normal Danish diet and to baseline. The average symptom score was reduced more in patients who used both IBS medication and were in the LFD group.	Study was unblinded, intended control group (normal Danish diet) had significant improvement too (possible bias), adherence was not measured.
Bohn et al (2015)[26]	Sweden	IBS patients on standard IBS dietary advice (n=37), IBS patients on LFD (n=38)	Randomised, single-blinded trial	4 weeks	Symptom score in both diets reduced significantly compared to baseline. No difference was observed in symptom score reduction between the two diets. The proportion of patients having a clinically significant response was similar in both diets.	Standard IBS dietary advice and LFD overlapped, use of probiotics, lactose-reduced diet and IBS medication was allowed in both groups.
Chumpitazi et al (2015)[25]	US	IBS patients aged 7–17 years (n=33)	Randomised, double-blinded, crossover trial	2 days LFD 2 days typical American childhood diet (5 day washout period	Patients had fewer daily abdominal pain episodes and a lower composite GI score in the LFD arm compared to the typical American childhood diet arm. However, composite GI score was not significantly different compared to baseline in any of the two diets, and pain severity decreased evenly in both diets.	Limited duration of intervention, all food was provided to the participants.

equimolar quantities (eg kiwifruit), a more efficient co-transporter, GLUT2, is upregulated, facilitating the absorption of glucose and fructose simultaneously. The total capacity of both GLUT5 and GLUT2 are variable, hence fructose malabsorption can be considered as a physiological feature in healthy people and IBS patients alike.

The three other restricted carbohydrates are fructans, galacto-oligosaccharides and polyols.[16] Fructans, polymers of fructose molecules, are found in wheat, onions, garlic and other foods such as legumes. Galacto-oligosaccharides are also found in onions and legumes, and in a number of vegetables. The human gut is unable to hydrolyse fructans and galacto-oligosaccharides, leading to non-absorption and subsequent delivery to the colon where they are readily fermented. Polyols are found in fruits such as apples, apricots and watermelon, and in avocado, mushrooms and cauliflower. In addition, a specific polyol, sorbitol, is frequently used as alternative sweetener. Absorption of these carbohydrates occurs mainly in the jejunum and is variable between individuals. When the amount of polyol in the diet exceeds the absorptive capacity, the non-absorbed carbohydrate will be osmotically active in the small intestine and fermented in the colon leading to symptoms in IBS patients with visceral hypersensitivity.

Implementation

In current clinical practice, the low FODMAP diet is taught by dietitians trained in the delivery of the low FODMAP diet in a 30-minute to one-hour session.[16] Food diaries and sometimes breath testing are undertaken before the session to personalise the diet to the patient's specific circumstances. After 4–6 weeks on a low FODMAP diet, a follow-up session is important. If symptoms are controlled, the dietitian and patient can both work towards reintroduction of restricted food items to a tolerated level. This ensures that the patient's diet is as complete and unrestricted as possible, reducing the risk of long term nutritional deficiencies (eg calcium, dietary fibre, B vitamins).

In studies of free-living individuals, low FODMAP diet adherence rates have been high (approximately 70%), and adherence has been strongly associated with efficacy.[9] While up to 60% of those adhering to the diet find it easy to follow, some patients report less positively on taste, cost, maintaining the diet while eating away from home and the effort needed to incorporate the diet into their lives.[9,10]

Efficacy

Table 1 describes the key studies that have been published concerning the low FODMAP diet in the management of IBS symptoms. Whole diet intervention studies are difficult to design, perform and remain blinded. As such, it is difficult to compare the methodology of randomised placebo controlled drug trials with those of whole foods or diets where it is near impossible to blind participants to the intervention and placebo. This was noted in recent systematic reviews where the quality of studies in the FODMAP field was noted to be poor.[21,22] A number of criticisms have been raised against the quality of many of the low FODMAP dietary intervention studies, including small number in the studies, the lack of a control group, the short duration of follow-up and that many studies have come from the same investigators. However, interventions of a whole diet are notoriously difficult to perform. The ideal study would blind participants and investigators to the diet, the participants would have the entire diet provided to them and the study would continue for years. Clearly the practicalities of adequately addressing any of these issues are very difficult to manage and have not been addressed in other whole-diet studies to our knowledge.

However, across a range of retrospective and prospective intervention studies, there has been a consistent effect demonstrated, supporting the use of low FODMAP diet in those with IBS. Nearly all studies found that 70–80% of IBS patients improved while on the low FODMAP diet, usually within 1–4 weeks.[23] Furthermore, the low FODMAP diet was favoured over usual IBS dietary interventions, local dietary habits and/or subject's baseline diet.[10,11,24,25] These findings are supported by the studies that underpin the mechanism of action of low FODMAP diet described earlier. Finally, data suggests a prolonged response to low FODMAP diet in those who remain adherent.[9]

Potential limitations and health-related drawbacks

So are there potential problems with a low FODMAP diet? Oligosaccharides are also prebiotics, which are a useful energy source to saccharolytic and probiotic bacteria in the gut (eg *Lactobacillus* and *Bifidobacterium*). Some of these genera have demonstrated in animal studies health-promoting effects on the host by influencing both the innate and adaptive immune system, the composition of the microbiota (preventing dominance of harmful species), and reducing the risk of developing colorectal cancer.[27] Moreover, the products of FODMAP fermentation, short-chain fatty acids (SCFA) supress pathogens and increase the absorption of minerals by acidifying luminal pH, modulate energy homeostasis and support apoptosis.

The low FODMAP diet eliminates the major source of dietary prebiotics, thus the source of energy for saccharolytic bacteria. This has a marked effect on the microbiota as was demonstrated in studies were the total abundance of major groups of bacteria decreased after a period of low FODMAP consumption.[28,29] Most notably among these were *Bifidobacteria* and other butyrate-producing species, which both have been widely described for their anticarcinogenic and anti-inflammatory effects. The relative decrease in the proportion of these bacteria may predispose to long-term adverse effects that have not been studied or demonstrated. Reassuringly, the few studies in this area have found that SCFA concentration and pH remain largely unchanged and had conflicting results on the change in *Bifidobacterium* after the institution of a low FODMAP diet.

Finally, there are no data investigating the long-term impact of low FODMAP diet on the GI microbiota, or after the reintroduction of high FODMAP foods. Given that the intestinal microbiota reacts rapidly to dietary changes,[30] adequate reintroduction of FODMAP foods could abolish any potential detrimental effects of the initial diet. Such re-introduction should be done in a slow stepwise manner, to determine whether there may be threshold effects whereby smaller quantities of FODMAP containing foods can be safely included without triggering symptoms. In conclusion, the short-term use of the low FODMAP diet is widely considered as a safe treatment, provided that patients alleviate their level of restriction after satisfactory symptom control and do not commence the diet when asymptomatic.

Future developments

Individualising dietary advice

Approximately a quarter of patients trialling the low FODMAP diet do not experience an improvement in symptoms. This could be partly due to poor adherence, but may reflect IBS pathogenic mechanisms independent of FODMAP triggers. A method of predicting the outcome of dietary intervention could prevent unnecessary treatment, reducing costs and potential risks.

With this in mind, Chumpatzi et al have attempted to discover if microbiome patterns can predict outcome and compared metagenome in responders and non-responders.[25] Responders had higher concentrations of bacteria with high saccharolytic metabolic capacity (thus were more able to ferment carbohydrates) and also had more markers of FODMAP-specific carbohydrate metabolism, than non-responders. Interestingly, microbial diversity was not significantly different between the two groups.

While the field of nutritional genomics has led to the prediction of individual postprandial blood sugar response, enabling important individual lifestyle adjustments that reduce the risk of developing type II diabetes mellitus,[31] there has been less progress in the field of IBS. Once again, this may be a fertile area for developing personalised medicine solutions for people with functional gastrointestinal disorders.

Streamlining dietary information delivery

At this point in time, the most evidence for the efficacy and safety of low FODMAP diet for the management of IBS symptoms is when it is delivered by a dietitian with expertise and training in its implementation in a one-to-one interview. However, this approach is also costly. Different methods of dietary advice delivery may benefit the availability and economic impact of

the low FODMAP diet. A recent study by Whigham et al found considerable cost savings and a similar clinical efficacy in a programme that facilitated group treatment for eligible patients, for example.[32] Traditional one-to-one sessions were still needed for patients with atypical symptoms and other nutritional and medical concerns in this study. Another alternative could be an online-based treatment platform, potentially in collaboration with GPs, since a number of studies have reported that patients generally find any written information about the diet they receive easy to understand.[9,10] Similar to group treatment, this approach may be suitable for providing low FODMAP advice to patients with typical symptoms of IBS and without any nutritional or medical concerns.

However, research on the nutritional safety and efficacy of these ways of dietary advice is needed before such measures are implemented. Besides, one-to-one dietary consultation has clear benefits that are not found in the methods described earlier. Firstly, specialist dietitians are able to ensure they are delivering correct information based on the current status of testing for FODMAPs in foods. Over the years, FODMAP content measuring has been intensified and refined and has forced adjustments in the diet. Some foods that were initially tested as high in FODMAPs have subsequently been re- evaluated and allowed back into the diet, others on the other hand have been re-listed as FODMAP rich foods. This has led to a variety of information available both in print and online that can appear to be conflicting as regards to certain foods and their safe threshold. Furthermore, some sources of information have misinterpreted the FODMAP tables and incorrectly limited certain foods when they have never been classified as high FODMAP (eg coconut milk and coconut cream).

The one-to-one approach also has the benefit of personalising the diet to the individual patient, increasing the chance of adherence and efficacy while reducing the chance of adverse effects due to prolonged low FODMAP diet without the re-introduction of FODMAP containing foods or poor nutritional quality. A dietitian can also take into consideration the bowel function and in the case of constipation, monitor this carefully when on a low FODMAP diet. It is possible constipation can worsen in some cases due to the lowering of fibre and loss of stimulant effect of osmotically active FODMAPs. Further modifications can be made to the diet with inclusion of suitable fibre or alternative supplements to benefit the bowel frequency without increasing the burden of bloating or flatulence.

Conclusion

Up to three-quarters of IBS patients experience symptom improvement on the low FODMAP diet. Although it comprises considerable changes, the diet appears safe as long as reintroduction of higher FODMAP foods (to a tolerated) level is part of the diet plan. The current method of dietitian-guided intervention in combination with written resources delivers high adherence among patients. However, exploring the possibilities of group, online or GP-based treatment in patients in formalised studies is warranted. Research should also concentrate on the effects of low FODMAP diet on quality of life, changes in tolerance and sensitivity towards certain foods after dietary intervention, and long-term nutritional and microbiotal health. Finally, the development of predictorary tools that enable dietitians to tailor the low FODMAP diet based on individuals' microbiota or genetics could increase efficacy and reduce unnecessary treatment. However, despite an interesting and promising outlook for the low FODMAP diet in IBS patients, it is important to realise that the solution for IBS might be beyond FODMAPs alone and that future treatments may be more potent in reducing symptoms.

Author information:

Tim Kortlever, Department of Medicine, University of Otago, Christchurch; Clarice Hebblethwaite, Dietitian, Dietary Specialists, New Zealand; Julie Leeper, Dietitian, Dietary Specialists, New Zealand; Leigh O'Brien, Dietitian, Dietary Specialists, New Zealand; Chris Mulder, Gastroenterology and Hepatology, VU University Medical Centre, Netherlands; Richard B Gearry, Department of Medicine, University of Otago, Christchurch.

Corresponding author:

Richard B Gearry, Department of Medicine, University of Otago, 2 Riccarton Avenue, Christchurch.

richard.gearry@cdhb.govt.nz

URL:

https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7021

REFERENCES:

1. Barbezat G, Poulton R, Milne B, et al. Prevalence and correlates of irritable bowel symptoms in a New Zealand birth cohort. N Z Med J. 2002 Oct 25;115(1164):U220.

2. Lovell RM, Ford AC. Global prevalence of and risk factors for irritable bowel syndrome: a meta-analysis. Clin Gastroenterol Hepatol. 2012 Jul;10(7):712–721.e4.

3. El-Serag HB, Olden K, Bjorkman D. Health-related quality of life among persons with irritable bowel syndrome: a systematic review. Aliment Pharmacol Ther. 2002 Jun;16(6):1171–85.

4. Gralnek IM, Hays RD, Kilbourne A, et al. The impact of irritable bowel syndrome on health-related quality of life. Gastroenterology. 2000 Sep;119(3):654–60.

5. Hausteiner-Wiehle C, Henningsen P. Irritable bowel syndrome: relations with functional, mental, and somatoform disorders. World J Gastroenterol. 2014 May 28;20(20):6024–30.

6. Inadomi JM, Fennerty MB, Bjorkman D. Systematic review: the economic impact of irritable bowel syndrome. Aliment Pharmacol Ther. 2003 Oct 1;18(7):671–82. Review.

7. Chey WD, Kurlander J, Eswaran S. Irritable bowel syndrome: a clinical review. JAMA. 2015 Mar 3;313(9):949–58.

8. Mertz H, Morgan V, Tanner G, et al. Regional cerebral activation in irritable bowel syndrome and control subjects with painful and nonpainful rectal distention. Gastroenterology. 2000;118(5):842–8.

9. De Roest RH, Dobbs BR, Chapman BA, et al. The low FODMAP diet improves gastrointestinal symptoms in patients with irritable bowel syndrome: a prospective study. Int J Clin Pract. 2013 Sep;67(9):895–903.

10. Staudacher HM, Whelan K, Irving PM, Lomer MC. Comparison of symptom response following advice for a diet low in fermentable carbohydrates (FODMAPs) versus standard dietary advice in patients with irritable bowel syndrom. J Hum Nutr Diet. 2011 Oct;24(5):487–95.

11. Halmos EP, Power VA, Shepherd SJ, et al. A diet low in FODMAPs reduces symptoms of irritable bowel syndrome. Gastroenterology. 2014;146(1):67–75 e5.

12. Shepherd SJ, Parker FC, Muir JG, Gibson PR. Dietary triggers of abdominal symptoms in patients with irritable bowel syndrome: randomized placebo-controlled evidence. Clin Gastroenterol Hepatol. 2008 Jul;6(7):765–71.

13. Bohn L, Storsrud S, Tornblom H, et al. Self-reported food-related gastrointestinal symptoms in IBS are

common and associated with more severe symptoms and reduced quality of life. Am J Gastroenterol. 2013 May;108(5):634–41.

14. Barrett JS. Extending our knowledge of fermentable, short-chain carbohydrates for managing gastrointestinal symptoms. Nutr Clin Pract. 2013 Jun;28(3):300–6.

15. Barrett JS, Gearry RB, Muir JG, et al. Dietary poorly absorbed, short-chain carbohydrates increase delivery of water and fermentable substrates to the proximal colon. Aliment Pharmacol Ther. 2010 Apr;31(8):874–82.

16. Gibson PR, Shepherd SJ. Evidence-based dietary management of functional gastrointestinal symptoms: The FODMAP approach. J Gastroenterol Hepatol. 2010 Feb;25(2):252–8.

17. Murray K, Wilkinson-Smith V, Hoad C, Costigan C, Cox E, Lam C, et al. Differential effects of FODMAPs (fermentable oligo-, di-, mono-saccharides and polyols) on small and large intestinal contents in healthy subjects shown by MRI. Am J Gastroenterol. 2014 Jan;109(1):110–9.

18. Ong DK, Mitchell SB, Barrett JS, et al. Manipulation of dietary short chain carbohydrates alters the pattern of gas production and genesis of symptoms in irritable bowel syndrome. J Gastroenterol Hepatol. 2010 Aug;25(8):1366–73.

19. Kassinen A, Krogius-Kurikka L, Makivuokko H, et al. The fecal microbiota of irritable bowel syndrome patients differs significantly from that of healthy subjects. Gastroenterology. 2007 Jul;133(1):24–33.

20. Misselwitz B, Pohl D, Fruhauf H, Fried M, Vavricka SR, Fox M. Lactose malabsorption and intolerance: pathogenesis, diagnosis and treatment. United European Gastroenterol J. 2013 Jun;1(3):151–9.

21. Moayyedi P, Quigley EM, Lacy BE, et al. The Effect of Dietary Intervention on Irritable Bowel Syndrome: A Systematic Review. Clin Transl Gastroenterol. 2015 Aug 20;6:e107.

22. Ford AC, Vandvik PO. Irritable bowel syndrome: dietary interventions. BMJ Clin Evid 2015 Jul 30;2015.

23. Marsh A, Eslick EM, Eslick GD. Does a diet low in FODMAPs reduce symptoms associated with functional gastrointestinal disorders? A comprehensive systematic review and meta-analysis. Eur J Nutr. 2015 May 17. [Epub ahead of print]

24. Pedersen N, Andersen NN, Vegh Z, et al. Ehealth: low FODMAP diet vs Lactobacillus rhamnosus GG in irritable bowel syndrome. World J Gastroenterol. 2014 Nov 21;20(43):16215–26.

25. Chumpitazi BP, Cope JL, Hollister EB, et al. Randomised clinical trial: gut microbiome biomarkers are associated with clinical response to a low FODMAP diet in children with the irritable bowel syndrome. Aliment Pharmacol Ther. 2015 Aug;42(4):418–27.

26. Bohn L, Storsrud S, Liljebo T, et al. Diet Low in FODMAPs Reduces Symptoms of Irritable Bowel Syndrome as Well as Traditional Dietary Advice: A Randomized Controlled Trial. Gastroenterology. 2015;149(6):1399–407 e2.

27. Roberfroid M, Gibson GR, Hoyles L, et al. Prebiotic effects: metabolic and health benefits. Br J Nutr. 2010 Aug;104 Suppl 2:S1–63.

28. Staudacher HM, Lomer MC, Anderson JL, et al. Fermentable carbohydrate restriction reduces luminal bifidobacteria and gastrointestinal symptoms in patients with irritable bowel syndrome. J Nutr. 2012 Aug;142(8):1510–8.

29. Halmos EP, Christophersen CT, Bird AR, et al. Diets that differ in their FODMAP content alter the colonic luminal microenvironment. Gut. 2015;64(1):93–100.

30. David LA, Maurice CF, Carmody RN, et al. Diet rapidly and reproducibly alters the human gut microbiome. Nature. 2014;505(7484):559–63.

31. Zeevi D, Korem T, Zmora N, et al. Personalized Nutrition by Prediction of Glycemic Responses. Cell.163(5):1079–94.

32. Whigham L, Joyce T, Harper G, et al. Clinical effectiveness and economic costs of group versus one-to-one education for short-chain fermentable carbohydrate restriction (low FODMAP diet) in the management of irritable bowel syndrome. J Hum Nutr Diet. 2015 Dec;28(6):687–96.

Gingival lipoma

Makoto Adachi, Shinichiro Sumitomo

A 67-year old woman was referred to the Oral and Maxillofacial Surgery Clinic with painless swelling at the gingiva of the lower-left canine. The patient did not notice the lesion, but it was pointed out by her dentist. Intra-oral findings showed soft yellowish masses with a smooth surface without erosions at this site (Figure 1A). The lesion was removed under local anesthesia (Figure 1B). Histopathological examination confirmed the diagnosis of a lipoma. There was no complication during this operation or after surgery.

Gingival lesions are commonly observed in dental clinics; apical periodontitis, periodontal abscesses, epulides, cysts and tumors should be included in a differential diagnosis.

Figure 1:

Author information:
Makoto Adachi, Department of Oral and Maxillofacial Surgery, Asahi University, Japan;
Shinichiro Sumitomo, Department of Oral and Maxillofacial Surgery, Asahi University,
Japan.
Corresponding author:
Makoto Adachi, Department of Oral and Maxillofacial Surgery, Asahi University, 1851
Mizuho, Gifu, Japan.
madachi_dds_phd@yahoo.co.jp

URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7022

Rare coexistence of dermatomyositis and smooth muscle antibodies, with abnormal liver function tests

Vicki Quincey, Kamal Solanki, Duncan Lamont, Marius Rademaker

Smooth muscle antibodies have rarely been reported in dermatomyositis. Its coexistence is not well known nor whether there is an association of autoimmune hepatitis with dermatomyositis. Also, the rash in dermatomyositis can be refractory to standard treatment of dermatomyositis.

We report such a case of new onset florid dermatomyositis with skin features and myositis along with abnormal liver function tests and presence of anti-smooth muscle antibody.

A 19 year-old woman presented with a month history of constitutional symptoms including fatigue, pruritus and a macular eruption over the lateral aspect of her left arm. The rash progressed to involve nasal and periorbital regions with swollen eyelids (Figure 1a) as well as characteristic Gottron papules over her knuckles and erythematous plaques on the arms, knees and legs (Figure 1b). She had progressive aching in the muscles, which she described as if she had 'run a marathon', and had difficulty going up and down the stairs, dressing and combing her hair. There was no concurrent fever, chills, jaundice, joint, bladder or bowel symptoms nor any respiratory symptoms. She did not have hair loss or sicca symptoms.

On clinical examination apart from the characteristic rash, she had tenderness of her forearms which were swollen and taut. She had difficulty in squatting and abducting her arms overhead with moderate muscle weakness. There was no hepatosplenomegaly.

Her full blood count, renal function, glucose, thyroid function, rheumatoid factor, immunoglobulin levels, ANCA, LKM antibodies, anti-mitochondrial antibodies (AMA), thyroid antibodies, ESR and CRP were all normal. Her hepatitis A, B, C, CMV and EBV viral serology were negative. Her echocardiogram, chest x-ray and pulmonary function tests were normal.

She had positive antinuclear antibodies (homogenous and cytoplasmic staining at 1/640) and positive smooth muscle antibodies (at 1/640 titre). The extractable nuclear antibodies and dsDNA antibodies were negative, C3 and C4 complement levels were normal, myositis panel (which tests for Mi2, Ku, PM-Scl 75 & 100, Jo1, SRP, PL7 & 12, EJ, OJ and Ro52) were negative. Her alanine transaminase (ALT) was 237U/L (n=<20) and aspartate transaminase (AST) was 247U/L (n=<40) and creatinine kinase (CK) was 1479 U/L (n=30–180). The serum albumin, serum globulin, immunoglobulins and protein electrophoresis were normal. Nerve conduction studies showed marked myopathic motor units consistent with myopathy and the deltoid muscle biopsy confirmed inflammatory myositis (Figure 1c and d). Liver biopsy was not felt to be indicated by gastroenterology at this time. A diagnosis of dermatomyositis was made.

She was commenced on methotrexate 15mg weekly and prednisone (1mg/kg/day) and bone protection. Her transaminases and creatinine kinase normalized. Muscle weakness resolved to baseline slowly. Her skin failed to improve until hydroxychloroquine 400mg daily was added.

Dermatomyositis is an uncommon heterogenous autoimmune inflammatory disease characteristically affecting the skin.

Specific pathognomonic patterns of rash occur (heliotrope discoloration around eyes, Gottron papules, V-sign, Holster sign, Shawl sign, etc.) as well as inflammation of skeletal muscles. Current data strongly suggest a pivotal role of both the innate and adaptive immune systems in its pathogenesis.[1]

Anti-smooth muscle antibodies (ASMA) have only rarely been reported in dermatomyositis.[2–6] It is not clear if there is coexistence between dermatomyositis and autoimmune hepatitis. Though her liver function tests (LFTs) have normalised with the treatment, the possibility of autoimmune hepatitis cannot be ruled out. Her positive anti-smooth muscles antibodies have persisted which heightens the suspicions of its co-existence. Our patient is being closely followed up for any relapse of abnormal LFTs in which case liver biopsy for diagnostic confirmation and histological grading will be contemplated and justified.

Autoimmune conditions can cluster with autoimmune thyroid disease reported as the most common coexistent autoimmune disease.[7] Primary biliary cirrhosis[6,8,9] has been reported rarely with autoimmune inflammatory myositis. However, our patient had normal immunoglobulins and the anti-mitochondrial antibody (AMA) was not detected.
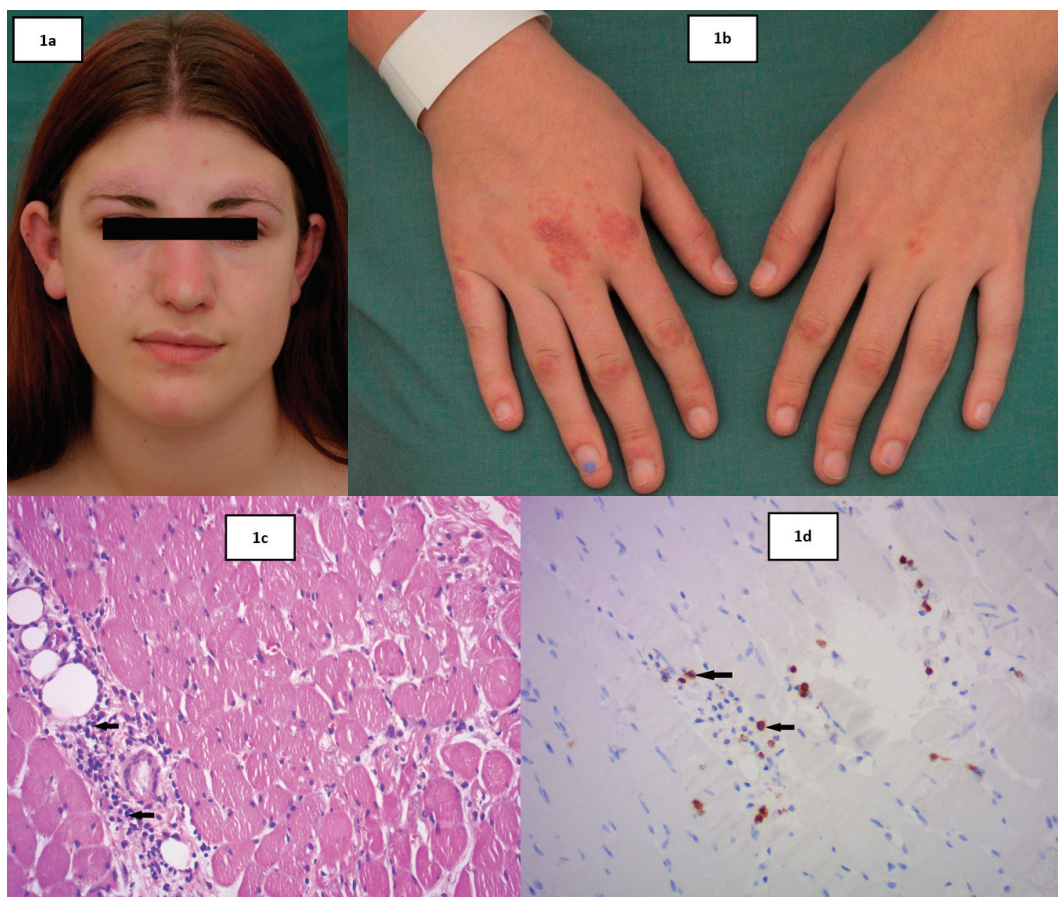
1a: Characteristic 'Heliotrope' rash of dermatomyositis, with scaly plaques on forehead, nose and ears.
1b: Characteristic "Gottron papules" on back of hands with ragged cuticles.
1c: Muscle biopsy (H&E stain at 200X magnification): arrows points to infiltrate of lymphocytes.
1d: Muscle biopsy (200X magnification) with arrows pointing to CD3 staining T Lymphocytes.

Author information:
Vicki A Quincey, Rheumatology Department, Waikato Hospital, Hamilton; Kamal K Solanki, Rheumatology Department, Waikato District Health Board, Hamilton; Duncan Lamont, Histopathology Department, Waikato District Health Board, Hamilton; Marius Rademaker, Dermatology Department, Waikato District Health Board, Hamilton.
Corresponding author:
Dr Vicki Quincey, Rheumatology Department, Waikato Hospital, Pembroke Street, Hamilton, New Zealand.
Vicki.quincey@waikatodhb.health.nz
URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7020

REFERENCES:

1. Tournadre A, Miossec. A critical role for immature muscle precursors in myositis. Nat Rev Rheumatol; 2013;9: 438–42.

2. Fujitake J, Saida K, Nishitani H, Suginoshita T, Osaki S. A case of polymyositis associated with chronic active hepatitis. Rinsho Shinkeigaku. 1990; 30: 55–61.

3. Hounoki H, Shinoda K, Ogawa R, Taki H, Tsuneyama K, Tobe K. Simultaneously developed polymyositis and autoimmune hepatitis. BMJ Case Rep. 2011 Nov 8; 2011. pii: bcr0920114763.

4. de Souza FH, Barros TB, de Moraes MT, Missumi LS, Lima FR, Levy-Neto M, Shinjo SK. Autoimmune hepatitis and dermatomyositis: a rare association. Acta Reumatol Port. 2012; 37: 264–7.

5. Schneeberger EE1, Arriola MS, Fainboim H, et al. Idiophatic inflammatory myophaties: its asociation with liver disorders. Rev Fac Cien Med Univ Nac Cordoba. 2012; 69: 139–43.

6. Kurihara Y, Shishido T, Oku K, et al. Polymyositis associated with autoimmune hepatitis, primary biliary cirrhosis, and autoimmune thrombocytopenic purpura. Mod Rheumatol. 2011; 21: 325–9.

7. McLeod DS, Cooper DS. The incidence and prevalence of thyroid autoimmunity. Endocrine. 2012; 42: 252–65.

8. Boki KA, Dourakis SP. Polymyositis associated with primary biliary cirrhosis. Clin Rheumatol. 1995; 14: 375–8.

9. Yasuda Y, Nakano S, Akiguchi I, Tanaka M, Kameyama M. A case of polymyositis associated with primary biliary cirrhosis. Rinsho Shinkeigaku. 1990; 30: 88–93.

10. Targoff IN. Dermatomyositis and polymyositis. In: Weston WL, MacKie RM, Provost TT (eds). Mosby Book; Current problems in Dermatology 1991: 131–81.

Exercise-induced rhabdomyolysis

Joseph Hutton, Daniel Wellington, Steven Miller

ABSTRACT

We report the case of a 34 year-old man who developed exercise-induced rhabdomyolysis after unaccustomed high-intensity exercise. Subclinical rhabdomyolysis is common after heavy exercise, yet it is uncommon for patients to seek medical advice. The presentation is variable and despite potentially life-threatening complications the diagnosis may be easily missed by patients and healthcare professionals. A high-index of suspicion is critical to avoid missing the diagnosis. We summarise the current knowledge, clinical course, complications and management of exercise-induced rhabdomyolysis.

Case Report

A 34 year-old man with unremarkable past medical and family histories was referred by his GP with a one-day history of pain and weakness affecting the lower back and thighs, and "tea-coloured" urine. Earlier that day, he had performed a strenuous weight training session to which he was unaccustomed. He denied use of steroids, supplements, or statins.

Examination was unremarkable aside from marked palpation tenderness in the quadriceps and lumbar para-spinal muscles, with 4+/5 weakness of hip flexion and knee extension bilaterally (limited by pain). There was no evidence of compartment syndrome.

The full blood count, coagulation screen, routine biochemistry and thyroid function were normal except for elevated creatine kinase (CK) of >14,000 U/L (60–220 U/L) and mildly elevated serum lactate of 1.6mmol/L (0.3–1.3mmol/L). His urine was tea coloured (Figure 1) and was strongly positive for blood on dipstick testing. However, urinary creatinine and protein was normal and the urine was acellular upon microscopy. These changes are suggestive of myoglobinuria due to the cross-reactivity of urine dipstick testing with myoglobin/haemoglobin.

The findings were consistent with exercise-induced rhabdomyolysis. Aggressive intravenous fluids were given and he was monitored daily for electrolyte disturbances and other sequelae. Electrolytes remained normal aside from transient asymptomatic hypocalcaemia 2.05 mmol/L (2.10–2.55mmol/L). CK remained >14,000 U/L for five days before falling. Symptoms improved over the same time-frame and he was discharged with GP follow-up.

Figure 1: Tea-coloured urine specimen from patient with exercise-induced rhabdomyolysis.

Table 1: The major categories of rhabdomyolysis and common causes for each category.

Type of Rhabdomyolysis	Common causes
Trauma	Crush syndrome, major trauma
Exertional	Strenuous or unaccustomed exercise, seizures (epilepsy, alcohol withdrawal)
Muscle hypoxia	Acute limb ischaemia/major artery occlusion, prolonged compression of limb during immobilisation or unconsciousness
Genetic	Disorders of glycolysis or glycogenolysis, disorders of lipid metabolism
Infection-related	Influenza A and B, Cocksackievirus, EBV, HIV, staph aureus pyomyositis, clostridium
Temperature-related	Heat stroke, malignant hyperthermia, malignant neuroleptic syndrome, hypothermia
Metabolic and electro-lyte disorders	Hypokalaemia, hypophosphataemia, hypocalcaemia, DKA, hyperosmolar hyperketotic state
Drugs/toxins	Fibrate, statins, alcohol, heroin, cocaine
Idiopathic	Unknown

Discussion

Rhabdomyolysis may be triggered by numerous insults (Table 1). Complications are less frequent in exercise-induced rhabdomyolysis than in other forms of rhabdomyolysis. Resolution is expected within one–two weeks. Severe exercise-induced rhabdomyolysis can cause acute kidney injury, electrolyte disturbances, disseminated intravascular coagulopathy, compartment syndrome and death.[2,4]

Excessive muscle activity induces rhabdomyolysis when myocyte energy demand exceeds production. Intracellular glycogen stores are exhausted and myocellular membranes become disrupted. The resulting release of intracellular skeletal muscle constituents into the circulation is responsible for complications observed.[13] Exercise-induced rhabdomyolysis is most commonly seen following strenuous physical activity but can sometimes occur after low-intensity exercise, especially in dehydrated individuals.[4] Risk factors include: male sex, being physically untrained, coexistent heat stroke, impaired sweating, sickle-cell trait, hypokalaemia and inherited muscle enzyme defects.[1,3–4]

Clinically, exercise-induced rhabdomyolysis is characterised by:[1]

- muscle tenderness, stiffness, cramping
- weakness in affected muscle groups
- "tea-coloured" urine
- other urinary symptoms: oliguria, anuria
- non-specific symptoms: malaise, fever, nausea, vomiting.

Baseline investigations include: urine dipstick, serum CK, electrolytes (including calcium), full blood count, coagulation screen, lactate, blood gas, TFTs and ECG.[1] The urine dipstick may be false-positive for blood due to cross-reactivity of myoglobin and haemoglobin (as in this case).[1–2] In recurrent cases, or if there is a family history of exercise-induced rhabdomyolysis, investigation for a predisposing genetic cause is suggested.[6] The conditions that predispose or cause rhabdomyolysis are varied and include disorders of lipid and carbohydrate metabolism, mitochondrial disease and myopathies.[1,3] Investigations should be targeted to the specific conditions of concern and may include skin and/or muscle biopsy, tissue histochemistry, muscle-exercise testing, biochemical tests and formal genetic testing.

The initial treatment of rhabdomyolysis is aggressive intravenous fluid resuscitation.[1–2,4] Normal saline is most commonly used. Care should be taken with fluid resuscitation, particularly in the context of prolonged anuria where patients are at greater risk of iatrogenic overload. Electrolyte disturbances should be corrected promptly, aside from hypocalcaemia which should only be treated if symptomatic or if concurrent severe hyperkalaemia is present due to risk of arrhythmia.[2] Sodium bicarbonate or mannitol may be considered under specialist guidance only.[2] Renal-replacement therapy may be used

for refractory complications, although this does not facilitate myoglobin elimination.[2] Plasmapheresis or haemofiltration with super-high-flux dialysers may have a role in severe cases.[7,8]

Conclusion

Exercise-induced rhabdomyolysis is an under-recognised phenomenon with potentially serious complications. The presentation, complications, investigations and management of exercise-induced rhabdomyolysis are summarised (Table 2). The most critical facet of making a diagnosis remains retaining a high-index of suspicion due to the uncommon nature of the presentation and its significant potential complications.

Table 2: Exercise-induced rhabdomyolysis: key points.

Summary of Exercise-induced Rhabdomyolysis	
Clinical features	• Muscle stiffness, tenderness, cramping • Weakness in affected muscles • Dark "tea-coloured" urine • Non-specific: malaise, fever, nausea, vomiting, fever
Risk factors	Male sex, unaccustomed exercise, heat stroke/hot weather, impaired sweating, hypokalaemia, inherited muscle enzyme defects, sickle cell trait
Investigations	• Urine dipstick and microscopy • Bloods: FBC, UEs, creatine kinase, lactate, calcium, phosphate, TFTs, clotting • Blood gas • ECG • May require specialist genetic testing if concerns about underlying cause

Management	• Early IV fluids (0.9% saline), large volumes may be required
	• Monitor:
	  - Fluid input/output: may require catheter
	  - Close electrolytes monitoring
	  - Signs of compartment syndrome/coagulopathy
	• Correct hypocalcaemia only if symptomatic or concurrent severe hyperkalaemia
	• Treat other electrolyte disturbances especially hyperkalaemia
	• Consider:
	  - Early involvement of intensive care/renal-services
	  - Consider use of Mannitol or Sodium Bicarbonate under specialist guideance
	  - Renal replacement therapy if resistant hyperkalaemia or other sequaelae of renal failure

Complications	• Acute kidney injury • Electrolyte disturbance: hyperkalaemia, hypocalcaemia, hyper/hypophosphataemia • Disseminated intravascular coagulopathy • Compartment syndrome • Death
Prognosis	Generally very good

Competing interests:
Nil.
Author information:
Joseph Hutton, General Medicine, North Shore Hospital, Auckland; Daniel Wellington, General Medicine, North Shore Hospital, Auckland; Steven Miller, Diabetes Centre, North Shore Hospital (WDHB), Auckland.
Corresponding author:
Joseph Hutton, General Medicine, North Shore Hospital, Shakespeare Road, Auckland.
jh7480@my.bristol.ac.uk
URL:
https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1442-23-september-2016/7023

REFERENCES:

1. Khan FY. Rhabdomyolysis: a review of the literature. Netherlands Journal of Medicine 2009; 67–272.

2. Bosch X, Poch E, Grau JM. Rhabdomyolysis and acute kidney injury. New England Journal of Medicine 2009; 361–362.

3. Kim J, Lee J, Kim S, Young Ryu H, Suk Cha K, Jun Sung D. Exercise-induced rhabomyolysis mechanisms and prevention: a literature review. Journal of Sport and Health Science xx 2015; 1–11.

4. Lee G. Exercise-induced rhabdomyolysis. Rhode Island Medical Journal 2014; 97 (11): 22–24.

5. Hill OT, Wahi MM, Carter R, Kay AB, McKinnon CJ, Wallace RF. Rhabdomyolysis in the US Active Duty Army, 2004–2006. Medicine and Science in Sports and Exercise 2012; 44 (3): 442–449.

6. Landau ME, Kenney K, Deuster P, Campbell W. Exertional rhabdomyolysis: a clinical review with a focus on genetic influences. J Clin Neuromuscul Dis. 2012 Mar;13(3):122–36.

7. Homsi E, Fernanda M, Barreiro L, Orlando J, Higa E. Prophylaxis of acute renal failure in patients with rhabdomyolysis. Renal Failure 1997; 19: 283–288.

8. Ronco C. Extracorporeal therapies in acute rhabdomyolysis and myoglobin clearance. Critical Care 2005; 9: 141–142.

Homeopathy—when self-regulation fails

Lance Gravatt

In July 2016 the Advertising Standards Authority (ASA) upheld a complaint against the Christchurch Homeopathy Centre for unsubstantiated therapeutic claims.[1] HCC declined to even participate in the self-regulatory process issuing a one line response "No thank you, I don't wish to respond". The ASA noted that this was the 22nd complaint upheld against homeopathic therapeutic claims since 2011.

The New Zealand Homeopathic Society advertises on their website the books *"Vaccine Free Prevention & Treatment with Homeopathy"*, *"Vaccination and Homeo-prophylaxis"* and *"Raising a Vaccine Free Child"*. The New Zealand Council of Homeopaths has an entire section of their website devoted to *"Homeopaths Answer to the Avian Flu"*.

The website for homeopath Dr Wendy Rose Isbell in Christchurch claims *"Well-chosen homeopathic remedies prescribed by trained practitioners can successfully treat infectious diseases even when there are no other alternatives"*.

The website for Flavell Homeopathy claims that conventional vaccinations are administered by *"injecting toxins directly into the bloodstream"* whereas homeopathic vaccination uses *"potentised nosodes"*.

The Canadian Paediatric Society review in 2015 states:

"There is scant evidence in the medical literature for either the efficacy or safety of nosodes, which have not been well studied for the prevention of any infectious disease in humans".[2]

In October 2015 the Federal Court of Australia issued a judgment in the case of the Australian Competition and Consumer Commission (ACCC) vs Homeopathy Plus! Australia PTY Limited regarding an article entitled "Whooping Cough—Homeopathic Prevention and Treatment." The Federal Court found that:[3]

"There is no reasonable basis, in the sense of an adequate foundation, in medical science… to state that Homeopathic Treatments are safe and effective as an alternative to the Vaccine for the prevention of whooping cough".

The Court imposed a fine of AUD 138,000.

At a time when global pandemics of infectious diseases such as Zika virus and Ebola virus seem more frequent and virulent, it is time for New Zealand to re-examine the effectiveness of its self-regulatory approach to therapeutic claims such as those made for homeoprophylaxis. Well before the 22nd upheld complaint by the ASA there must be the provision to impose significant fines that 'hurt'. The situation has gone beyond freedom of choice to one of societal harm through misleading, deceptive and unsubstantiated claims.

REFERENCES:

1. ASA Complaint 16/107

2. Rieder M J et al., 'Nosodes' are no substitute for vaccines. Paediatr Child Health. 2015: 20(4): 219–222.

3. Federal Court of Australia, NSD 256 of 2013.

Olanzapine for the prevention of chemotherapy-induced nausea and vomiting

Chemotherapy-induced nausea and vomiting are associated with a significant deterioration in quality of life and are perceived by patients as major adverse effects of cancer treatment.

Current international guidelines recommend a combination of a neurokinin receptor blocker, a serotonin receptor blocker and dexamethasone to alleviate these adverse reactions. However, even with the use of these three agents, nausea remains a problem for many patients.

Olanzapine, an antipsychotic agent, is known to be a multi-transmitter blocker. In this report its use as an antiemetic is tested. Three-hundred and eighty patients receiving highly emetogenic therapy were randomised to be treated with the three standard therapies plus olanzapine or a placebo.

The conclusions reached were that olanzapine, as compared with placebo, significantly improved nausea prevention, as well as the complete-response rate, among previously untreated patients who were receiving highly emetogenic chemotherapy.

N Engl J Med 2016;375:134–42

The cardiovascular safety of methylphenidate

Methylphenidate is often used in the management of children and young people with attention-deficit/hyperactivity disorder (ADHD). Such stimulants are known to be capable of causing tachycardia and slight elevation of blood pressure.

This analysis of case series data from South Korea reviews this issue. The data showed an increased risk of arrhythmia in those treated with methylphenidate which was highest in those with congenital heart disease. The researchers concluded that the use of methylphenidate in children and young people with ADHD is associated with an increased risk of arrhythmia and myocardial infarction. While there was an increased relative risk, the absolute risk is likely to be low.

A reviewer notes the results and points out the need to consider the severity of ADHD symptoms and the option of non-stimulants for children with high cardiovascular risk.

BMJ 2016;353:i2550 and BMJ 2016;353:i2874

Associations of urinary sodium excretion with cardiovascular events in individuals with and without hypertension

In this study the researchers aimed to explore whether the association between sodium intake and cardiovascular disease events and all-cause mortality is modified by hypertension status. Data from four large prospective studies conducted in 49 countries was reviewed. A pooled analysis of sodium excretion in 133,118 people (63,559 with hypertension and 69,559 without hypertension) with a median age of 55 years was performed.

Compared with moderate sodium intake, high sodium is associated with an increased risk of cardiovascular events and death in hypertensive populations (no association in normotensive population), while the association of low sodium intake with increased risk of cardiovascular events and death is observed in those with or without hypertension. These data suggest that lowering sodium intake is best targeted at populations with hypertension who consume high sodium diets.

Lancet 2016;388:465–75

Obituary—William Brown, M.D., M.A., of Dunedin

October 1916

On July 1st, Dr. Brown died suddenly in an attack of angina pectoris. No doctor in the South Island was more widely known, during the years of his active work, and none gained more widely the respect and affection of colleagues and patients. His connection with Dunedin began in 1875, when he commenced practice there and quickly became a prominent figure among his professional brethren, and for many years enjoyed a large practice among all classes of the community. This mile of his life was marked by continual charities and help to his poorer patients. They were always his favourites, and when he gave up practice in Dunedin it was his chief regret that he was leaving so many of the poor "old bodies" whom he had attended for so many years.

Professionally he quickly made his mark. His appointment to the staff of the Dunedin Hospital gave him the opportunity of surgical practice, which he desired, and later on, when the Medical School was established, he was appointed Lecturer on Surgery, this being the first appointment of a teacher in the practical side of medical education. In 1882 he made a visit to the Old Country, to examine methods of teaching and practice and returned in 1883, full of energy and enthusiasm in his work. For the next ten years he was a very prominent figure in the growing Medical School. His steady common sense was of great value in the somewhat adventurous experiment of launching a Medical School in so small a community as Otago, and the various changes which had to be made in local hospital government and construction were made easier by his tact and knowledge.

As a teacher he was clear and accurate and quickly gained the confidence of his pupils, and it was a matter of great regret to his colleagues when he decided to give up that branch of his work. Then followed two or three years' travel in Europe and America. He returned—about the middle nineties—to practise in Dunedin but did not begin University work again.

He was keenly interested in all educational questions, and served on the High School and Education Boards, besides doing much other public work. When he retired from practice about 1904 he went Home again to Scotland, but returned to New Zealand about 1912, settling first in Marton and later in Dunedin. He had always been a man of good physique and health, but it was observed by his friends on his return to Dunedin in 1914 that there were signs of heart failure. He did not resume practice, but took up work on the Patriotic Committee, and by a very general wish of many of the citizens he became a member of the Hospital and Charitable Aid Board, doing most valuable work for these bodies up to the day of his death.

A full and busy life ended suddenly, probably as he would have wished. He knew well that the end could not be far off and that it would probably come suddenly, but his courage never failed, nor did any complaint pass his lips. In trying to estimate his character and influence, the chief thing which struck those who knew him well was the universal feeling of friendliness which he inspired.

In whatever work he was engaged, private or professional, those who were his colleagues became his friends. He had a "genius for friendship"; all sorts and conditions of men and women who came in contact with him experienced the same feeling. It is difficult to define what is at the bottom of such an influence: charity, goodness, cleverness, public spirit, are not sufficient; all these may be present and leave us cold, but in Dr. Brown there was an immense gift of sympathy, invaluable to a doctor above all men, which probably was the secret of this universal feeling of personal friendliness to him. Among his

intimate friends he was always a delightful companion. He had travelled much and read much. His interests were catholic in the extreme—politics, literature, sociology, interested him to the last, and in the widest sense of the word he was a religious man, in the smallest and the greatest things placing duty first.

The following notice taken from the Dunedin "Evening Star" gives some further details of his life and of the esteem in which he was held by his fellow-citizens:—"He was born in 1845, in Banffshire, Scotland, the son of a farmer. He received his preliminary education at the gymnasium of Old Aberdeen, then graduated at the universities, taking the arts course at Aberdeen, where he distinguished himself in classics and mathematics, and gained the M.A. degree in 1867 and qualifying in the medical course at the Edinburgh University, which in 1870 bestowed on him the degrees of M.B. and C.M.

He went out to China as a medical missionary of the Baptist Church, and was for three years located at Che-Foo, where he conducted a hospital for the natives. He came to Dunedin in 1875. The ship by which he travelled from China was wrecked on the Queensland coast. Shortly after arrival here he started the practice of his profession, his surgery being in Princes Street, in the same building as that in which Mr. Downie Stewart the elder had his law chambers. Before long he removed to High Street. He joined the honorary medical staff of the Dunedin hospital, and was for some years lecturer on surgery at the Otago Medical School. He took a keen interest in education questions, and bestowed much time on his duties as chairman of the High Schools Board of Governors, whilst he also held a seat on the Otago Education Board, and became chairman of that body.

Outdoor sports also claimed his attention. He was a golfer, and at one time held the office of captain of the Otago Golf Club; he also became president of the New Zealand Amateur Athletic Association. After some years' residence in Dunedin the doctor paid a visit to the Old Country. Before his departure his many friends entertained him at a banquet.

On his return to Dunedin he resumed the practice of his profession, and eventually sold out to Dr. Church and went to Tauranga, that place being chosen on account of his wife's health. He stayed at Tauranga for a couple of years. Having some leisure, for he did not practise there, the doctor interested himself in education matters, and sat on the local school committee. Then he went Home once more, and stayed for some years. Whilst in Scotland he was closely associated with his brother-in-law, Mr. Thomas Johnston, managing director of Nobel's Explosives Company in Glasgow.

After returning to New Zealand, Dr. Brown went to live near Marton, but the climate did not suit Mrs. Brown, and they came back to Dunedin three or four years ago. The doctor then acquired an interest in the Ashburn Hall Asylum, and he occupied himself also in his duties on the Hospital and Charitable Aid Board and as a member of the Otago Patriotic Association. In 1871 he married a daughter of Mr. John Johnston, of Edinburgh, and they had one child, a daughter, who died whilst young. Mrs. Brown survives her husband.

The internment took place this morning, privately, but there were quite a number of close personal friends at the service in St. Matthew's Church. The deceased was a loveable man, very outspoken and sincere, but always most kind and considerate. His old clients invariably made him a personal friend. The feeling of respect entertained towards him by the community as a whole was shown on the occasion of his removal to Tauranga. There was a big meeting at His Majesty's Theatre, and all sorts of honours were showered upon him."

Proceedings of the 234th meeting of the Otago Medical School Research Society

2016

Adult born neurons: do cells "retire" and does developmental age matter?

SM Ohline,[1,3] RU Hegemann,[1,3] KL Wake,[1,3] MF Dinnunhan,[1,3] AD Wilson,[1,3] L Schoderböck,[1,2,3] SM Hughes,[2,3] WC Abraham.[1,3]

[1]Department of Psychology, [2]Department of Biochemistry and [3]Brain Health Research Centre, University of Otago, Dunedin.

Neurogenesis occurs throughout adulthood in mammals. However, there is controversy about whether or not old neurons 'retire' and newly born neurons take their place in neural circuitry.

In order to address this controversy, we 'birth-dated' newly born neurons in the dentate gyrus of Sprague Dawley rats using two thymidine analogues, (chloro-deoxyuridine (CldU) and iodo-deoxyuridine (IdU)), given at 35, 12, six or four weeks prior to study at 10 months of age. We then used immunofluorescence to identify active neurons as indicated by co-localisation of the thymidine analogue with the protein expressed by the immediate early gene *Zif268* (a marker of neuronal activity) and a neuronal marker, either calbindin or NeuN.

We found that the neurons born 35 weeks prior to study expressed Zif268 in the same proportion as those born only four weeks prior to study. However, neurons born at 12 weeks (% Zif268+/XdU+=1.1±0.20%) and six weeks (% Zif268+/XdU+=1.6±0.40%) before study expressed Zif268 in a significantly lower proportion to those born at four weeks (% Zif268+/XdU+=3.6±0.50%), where

P=0.002 (12 week vs four week) and P=0.01 (six week vs four week) in unpaired t-tests. We then asked the question: does the developmental age of the animal influence the relative activity of newly born cells? Using neurons birth-dated at 12 weeks (when the animal was two months old) and four weeks prior to study in animals aged only five months at perfusion, we found no difference in activity levels between the two groups (% Zif268+/XdU+=3.86±0.46% (12 week) and 4.48±1.0% (four week), P=0.48). Surprisingly, this level of activity was also not significantly different from the activity in the 35 week old cells or four week old cells in the 10 month old animals.

These results indicate that neurons born when a rat is young (two months of age) continue to be highly active in neuronal circuitry. If 'retirement' does occur, it appears to be specific to neurons born during middle-age.

Bacteria modulate goblet cell development in the colon; a mechanism compromised in Crohn's disease.

E Rodrigues,[1,2] M Schultz,[2] AG Butt.[1]

[1]Department of Physiology, Otago School of Medical Sciences and [2]Department of Medicine, Dunedin School of Medicine, University of Otago, Dunedin.

The intestinal epithelium and associated mucus form a physical barrier between the luminal bacteria and the intestinal immune system. The development of this barrier is dependent upon interaction between the epithelial cells and the luminal microbiota, and disruption of the barrier is a feature of Crohn's disease (CD). The objective of this study was to use human colonoids, an adult stem-cell derived representation of the gut crypt, to determine how the intestinal bacteria modulate the development of the epithelial barrier and whether this is disrupted in CD.

Colonoids were grown from colonic crypts from control and CD patients for 10 d ± lipopoly-saccharide (LPS) a bacterial cell-wall component. Organoid structure was assessed by light microscopy and gene expression by qPCR, Western Blotting (WB) and immunohistochemistry. Data are expressed as mean ± SEM of experiments performed from five different patient samples (n=5, unless stated otherwise). Statistical significance was determined using the paired t-test.

Colonoids exposed to LPS increased protein expression of MUC2 (P<0.05), a goblet cell marker and the number of goblet cells in colonoids from 2.7±1.2% to 20%± 3% (P<0.05). This involved LPS receptor-mediated (TLR4) modulation of pathways responsible for determining intestinal epithelial cell fate. Elevated IL8 transcript expression following TLR4 stimulation confirmed active LPS receptor (TLR4) and blocking the TLR4- receptor (CLI-095, 1 µg/ml) abrogated the LPS-driven MUC2 increase, indicating a TLR4-dependent pathway. Interestingly, IBD derived colonoids treated with LPS exhibited no increase in goblet cells (n=3). Additionally,

WB analysis showed less MUC2 protein (*P*<0.05).

The inability of the LPS to induce goblet cell development in CD is likely to contribute to the disruption of the intestinal barrier in CD, which contributes to the pathogenesis of CD by increasing the exposure of the immune system to the commensal bacteria.

Altered metabotropic glutamate receptor activity in early spinocerebellar ataxia type 1.

E Power, A Morales, R Empson.

Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

Spinocerebellar ataxia type 1 (SCA1) is a progressive and incurable degenerative movement disorder associated with loss of cerebellar function. A primary symptom of SCA1 is the progressive loss of Purkinje neurons (PNs), cells which provide the sole output from the cerebellar cortex. These neurons express a particularly high level of Type 1 metabotropic glutamate receptors (mGluR1). In this study we explored the potential for mGluR1 as a therapeutic target for the treatment of ataxia using a mouse model of human SCA1. This mouse model of SCA1 expresses an 82Q CAG repeat in the ataxin-1 gene which is expressed specifically in PNs.

SCA1 mice at early (six weeks) and mid-stage (12 weeks) of disease progression exhibited moderate motor impairment on an accelerating rotating rod task and altered gait (rotarod six weeks *P*<0.05, 12 weeks *P*<0.005, two-way ANOVA, Gait analysis 12 weeks *P*<0.008, one-way ANOVA). PNs from these mice exhibited excessively prolonged mGluR1-dependent synaptic currents and calcium signalling responses (six weeks *P*<0.01, 12 weeks *P*<0.001, two-way ANOVA).

Since careful control of mGluR1 signalling is critical for cerebellar function in mice and humans, we sought to remove the excess mGluR1 responses with a potent, readily-available pharmacological modulator. Acute treatment with a low dose of the potent and specific activity-dependent mGluR1 negative allosteric modulator JNJ16259685 shortened the prolonged mGluR1 currents in PNs from 12 week old SCA1 mice (*P*<0.005, two-way ANOVA) and also rescued their motor impairments on the rotating rod task and restored their gait (rotarod *P*<0.03, two-way ANOVA, gait analysis *P*<0.02 2-way ANOVA). These results demonstrate a compelling link between excessive mGluR1 signalling and the onset of ataxia and also promote mGluR1-based pharmacology as a realistic new avenue for the treatment of ataxia.

Proteomic identification of an abdominal aortic aneurysm specific 14-3-3 isoform: insights into its potential as a therapeutic target

TD Kabir, C Jin, LV Phillips, AM van Rij, GT Jones

Department of Surgical Sciences, Dunedin School of Medicine, University of Otago.

Abdominal aortic aneurysm (AAA) is a major health issue among men over 65 years of age. Although initiatives, such as national screening programmes, may help reduce the rates of AAA rupture, currently surgical repair is the only available treatment. This study aimed to characterise a novel mechanistic pathway in AAA pathogenesis and determine its potential utility as an AAA specific pharmacological therapeutic target.

Two-dimensional gel electrophoresis was used to determine the differential expression of proteins in AAA specimens and were validated by western blot (AAA, n=82, vs non-aneurysmal, n=12) and ELISA (AAA, n=36, vs non- aneurysmal, n=30). Levels of the most up-regulated protein (14-3-3-θ) was examined further in specific layers of the aortic wall (n=5). 14-3-3-θ and non-targeting control siRNAs were used to selectively knock-down this gene in an immortalized T-lymphocytic (Jurkat) cell line. Its effects on cell proliferation, migration and OKT3-induced activation were measured by CyQuant NF-cell proliferation assay, 2D-chemotaxis assay and ELISA for human IL-2, respectively.

14-3-3-θ was consistently up-regulated (p=0.005) in AAA tissues, particularly in the T-cell-rich lymphoid tissue within the adventitial layer. *In vitro*, transient depletion of 14-3-3-θ in Jurkat cells reduced proliferation (p=0.025), stimulated chemotaxis (p=0.004) and suppressed IL-2 secretion (p<0.05).

The accumulation of lymphocyte aggregate in the adventitia of the aorta was reported to correlate with the expansion and rupture in AAA by mechanisms that are poorly understood. These AAA-associated T-lymphocytes secrete cytokines, contributing to disease progression. We showed that 14-3-3-θ is essential for Jukrat cell activation and exerted a pro-proliferative and an anti-migratory effect, a possible mechanism that would favour their *in situ* activation, aggregation and expansion. Abolishing this aggregation of AAA-associated T-lymphocytes, by targeting 14-3-3-θ, therefore, may hold promise as a disease-specific pharmacological approach to slow the progression of an aneurysmal vascular disease.