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Challenging beliefs about the marketing of food

Robert Quigley, Carolyn Watts

The marketing of high fat-, salt-, sugar- and energy-dense foods is now firmly in the gaze of parents, the community, and health professionals as a vector (cause) of disease.

Maher et al¹ have provided a reminder to us all that the environment in which we live has significant impacts on the obesity epidemic in New Zealand. In a small pilot study, the authors have measured the amount and content of food advertisements, and types of food available nearby to secondary schools. It is a simple and well-conceived study that begins to map out the obesogenic environment of New Zealand.

Maher et al step outside the current paradigm and rightly look upstream at what determines our children's health. We should not be surprised by their results—because a walk around any neighbourhood will show that our environment is sick. Our environment is not normal. We should not be surprised that our children are growing fat when their world is saturated with unhealthy messages. Put simply, we need to focus more on the causes of the causes and less on the individual.

Marketing

The 'marketing' that our children are exposed to is all-encompassing—and Maher et al have only scratched the surface of what children are exposed to. Marketing long ago stepped from the realm of providing information about products so that consumers have information to make healthy choices, into a propaganda mode of selling values, visions, and concepts—to which products are aligned.^{2,3}

The propaganda around fast food and soft drinks is that they are 'healthy choices' which can be part of a 'healthy diet'—directly marketed by our nation's elite athletes.⁴ As *McDonald's* own marketing agency has said, *McDonald's Eat Smart Be Active* campaign has maintained low sales of the *Salads Plus* menu, but has given parents (or 'gatekeepers' as they are called by the marketers) the permission to eat at *McDonalds*—with a subsequent rise in sales of traditional burger-and-fries products.²

Personal choice

In election year, we are again surrounded by the rhetoric of 'individual choice', as though this is a panacea for all ills. However whether an individual chooses a healthy diet is much more influenced by the availability, affordability, and accessibility of food than that individual's knowledge about healthy food choices. Choice is important—but we argue that the right to choose a healthy diet has been all but removed from children today. We live in a world where the saturation marketing/propaganda of unhealthy food is the norm and environments support unhealthy choices—and yet we continue to be surprised that we are in an obesity epidemic.

Parental and personal responsibility are key agents in the propaganda of food companies—if only people were educated about what a healthy diet is and had better food labelling (for example), they'd be empowered to make the healthy choice.

But honestly, what kind of choice are we offering our children when:

- 70% of the advertisements for foods around schools are for junk food;¹
- Two-thirds of the advertisements on children's TV are for junk food;⁵
- *McDonalds* sponsors your child's school dental clinic, the school's road safety programme, and soccer team; and
- *Coke* sponsors their after-school care and 'Christmas in the Park'.

Actually, commercialism directs their life and yours.

Choice has been hijacked by marketers in a similar way with statements such as 'everything is OK in moderation' and 'eat a balanced diet'. However, choice is not positive when the 'choices' being offered are not healthy choices. Interestingly, when you look at the Ministry of Health's recommendations there is no mention of moderation or balance—the Ministry's recommendations actually make sense—'eat less fatty, salty and sugary foods, eat more fruit and vegetables, eat a variety of nutritious foods'.⁶ Providing a choice of unhealthy foods so that the diet is well balanced with junk does not equate to healthy eating.

Evidence

According to the World Health Organization, the heavy marketing of energy-dense foods and fast-food outlets is a probable cause of obesity.⁷ This is backed up by a systematic review of the evidence prepared for the British Office of Communications that states '...the public will never find it credible that an industry that spends huge sums each year advertising food to children on television does so with no actual (or intended) effect on children's food consumption'.⁸

In the most comprehensive systematic review of evidence, prepared for the British Food Standards Agency, the authors concluded that food advertising can influence children's food preferences, their purchase behaviours and what they eat.⁹

Solutions

We need to be much more radical than just banning TV advertising of high fat, high salt, and sugar foods to children. We need to be much more strategic and comprehensive.

Rather than focusing on advertising alone we need to broaden our focus to unhealthy marketing as a whole by limiting the amount of promotion, product development, sponsorship, placement, pricing, and advantageous distribution of unhealthy options. Along with limiting unhealthy marketing, we can also increase 'healthy marketing'.

We acknowledge that banning TV advertising alone would be spectacularly ineffective at reversing the obesity epidemic, but this should in no way be read as a reason not to include it in a suite of interventions to tackle obesity. The question is not whether we should ban TV advertising of junk food to children, but what else should

we do to create a healthy environment for children, where healthy eating is experienced as the norm?

While the Government's approach to preventing childhood obesity is well directed (on paper at least through *Healthy Eating Healthy Action*), it is sadly under-resourced—both in staff and financial commitment. While there are some components of the strategy that target environmental change, these are too few given the significance of this area. We need to re-balance our efforts, so that additional attention focuses on the causes of the causes rather than on the individual. And we should be aware that the food and advertising industries will resist such refocusing.

Health professionals must unite against the dominant paradigms that are prevalent in our society. The health system must be reoriented to focus a more significant proportion of its efforts to targeting upstream determinants

If we do not take on this challenge, no-one else will; if we do not advocate for healthy environments, no-one else will. And if we do not believe that radical change is required, no-one else will!

Competing interests: R Quigley and C Watts have coauthored several scientific papers and published documents on what works to prevent obesity and overweight, risk factors of the disease, and the obesogenic environment. C Watts is chair of Agencies for Nutrition Action (ANA) and a member of the NZ Dietetic Association. R Quigley is a member of the ANA Scientific Advisory Committee and a member of the NZ Dietetic Association. The authors' views are their own and do not represent the organisations to which they are affiliated.

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Circumcision: certain controversy over uncertain origins

Spencer Beasley

It is as hazardous to write about circumcision as it is to venture into the equally controversial areas of abortion, stem cell research, and euthanasia. Few subjects generate as much passion and conflicting opinion. Those who hold views at each end of the spectrum promote them with fanatical fervour. Often they are highly selective in the information they use, and tend to cite 'evidence' that supports their argument while ignoring material (that may be more scientifically sound) that negates it.

In the case of circumcision, extremes of view range from a website that 'blacklists' surgeons believed to perform circumcisions (not referenced here to avoid giving it undeserved credibility or publicity) to authors who claim that it is negligent not to circumcise all boys. Even in peer-reviewed scientific journals, discussion of circumcision frequently contains emotive language and extravagant claims, not necessarily supported by the evidence presented. For these reasons, journals tend to be hesitant about publishing opinion on circumcision.

In this issue, despite the risks, the *Journal* has taken the bold step of publishing an opinion by Robert Darby on the origins of circumcision, and the part that balanitis or infection under the foreskin may (or may not) have had in leading to cultural circumcision, or to the increase in the incidence of the operation in Australasia after the two World Wars.¹

Although Darby was unable to find any reference to recurrent balanitis in the *British History of the Second World War*, the problems created by dust storms in troops who had limited access to facilities for washing were well known to the returning servicemen of the various medical corps. For example, both the eminent Australian surgeon, Russell Howard FRACS and physician Dr Andrew Hutson, of Melbourne, described the significant problems experienced by uncircumcised men in North Africa during the Second World War (personal communication, John Hutson, 2005).

Their colleagues and trainees remember their descriptions of troops suffering from irritation from sand beneath the foreskin—trivial and unspectacular, perhaps, compared with the other illnesses and wounds treated, but not pleasant for the afflicted, nevertheless. The omission of balanitis from tomes primarily focused on the major battles that weaved their destructive courses across continents is not surprising, but does not mean that balanitis was not a cause of significant morbidity for the hapless soldier on the ground. Nor does irritation of the foreskin after a dust storm equate with circumcision on the front line.

It is unlikely many circumcisions would have been performed during the desert campaign itself (and this has not been claimed by those quoted in the accompanying article), but it does seem that the experience of war contributed to surgeons (on their return to Australia and New Zealand) being willing to support routine circumcision of boys in the neonatal period, presumably to reduce the likelihood of balanitis later in life.

The incorrect suggestion by Darby in his Abstract that ‘mass circumcision was necessary’ (or occurred) during the Second World War may have resulted from his misinterpretation of the implications of balanitis, and failure to recognise that it was almost always managed non-operatively.

What Darby has shown us is that the origin of cultural circumcision remains far from clear. Many of the theories promoted to account for ritual circumcisions² may seem fanciful to us, but so too do some of the medical indications for circumcision that have been promulgated by some Western physicians in recent centuries. While they may not have received general acceptance, they are widely quoted and have included epilepsy and for the prevention of masturbation—for the latter, as temporary relief only, we would imagine.

As to whether considerations of hygiene or the discomfort produced by foreign material causing irritation beneath the foreskin played any part in the requirement for circumcision in a variety of religions remains uncertain—and may never be known for sure. There is a lot of conjecture, but not much evidence. While the accompanying article may not shed much light on these aspects, it will prove interesting for those readers not familiar with the controversies surrounding the practise of circumcision in males.

Finally a word of caution: so-called ‘circumcision’ in females is a somewhat misleading term that covers a variety of procedures, some of which are extremely mutilative and extend well beyond the phallus. None of them are performed in this country. These procedures are alluded to in the article, but have little bearing on discussion of male circumcision.

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Advertising and availability of 'obesogenic' foods around New Zealand secondary schools: a pilot study

Anthony Maher, Nick Wilson, Louise Signal

Abstract

Aims To examine the extent and content of outdoor food advertisements and food availability from outlets in the vicinity of secondary schools.

Methods The sample of schools (n=10) was randomly selected from a sample frame of schools in both an urban and rural region (Wellington and Wairarapa regions respectively) and at each extreme of the socioeconomic status (SES) distribution (based on school characteristics). An area of 1-km radius around the schools was examined for food and non-food product advertisements and shops/outlets.

Results Out of 1408 outdoor advertisements for products, 61.5% were for food (i.e. 28 per square kilometre). The major categories were soft drinks (21.6%), frozen confectionary (16.2%), savoury snacks (11.4%), and alcohol (8.1%). Overall, 70.2% of food advertisements were for foods classified as 'unhealthy' (i.e. inconsistent with the national nutritional guidelines for adolescents).

A majority of the 224 outlets sold food (i.e. 56.3%). Those that primarily sold food were (on average) closer than other outlets to the secondary schools ($p=0.03$). Out of those schools that sold meals, the proportion of these that advertised a salad option was significantly lower in the low SES neighbourhoods ($p=0.006$). Other significantly different patterns for food outlet distribution, and category of advertised food were found by SES and rurality.

Conclusion Although only a pilot study, the information obtained suggests that food advertising and food outlets are prevalent in the vicinity of secondary schools and that the advertising is generally not compatible with nutritional guidelines for adolescents. Larger studies into such advertising are needed as well as consideration of policy options to control aspects of the 'obesogenic environment.'

There is growing concern over the prevalence of obesity and related chronic diseases such as diabetes in New Zealand (for example, as shown in the *New Zealand Medical Journal* articles that appeared in the 17 December 2004 'theme' issue. URL: <http://www.nzma.org.nz/journal/117-1207/>).

Over the last 25 years, the prevalence of obesity has doubled in New Zealand adults.¹ Also, the National Children's Nutrition Survey found that 31% of New Zealand children were overweight or obese, with this figure being over half in some population groups.² Childhood obesity is a risk factor for adult obesity but it also has its own adverse psychological and health impacts (e.g. increased risk of insulin resistance syndrome³).

Many factors are likely to be involved in the New Zealand and global obesity epidemic, but a key component is probably the 'obesogenic' environment, which facilitates both overeating of energy dense food and physical inactivity.^{4,5} One

component of this obesogenic environment is the high prevalence of food advertising and the nature of the food advertised (i.e. energy-dense foods that are high in free sugars and fat).

Food advertising has been shown to work on children according to an experimental study of children exposed to a videotape with embedded advertisements.⁶ The exposed children were significantly more likely to choose the advertised items than children who saw the same videotape without advertisements ($p < 0.01$). There has also been one randomised trial that indicates that reducing children's television viewing time is associated with statistically significant decreases in body mass index⁷—but the role of reducing physical inactivity versus advertising exposure were not separated out.

The authors of a recent systematic review also reported that there was good evidence that food advertising influences food preference and purchase behaviour by children.⁸ In addition, compared to lean children, there is also evidence that overweight and obese children demonstrate heightened recognition of food advertisements and consume more food after exposure to such advertisements.⁹

In New Zealand, research has found that food advertising on television during children's viewing hours is predominantly for foods high in sugar, fat, and/or salt.^{10–12} However, there have been no published studies of outdoor food advertising in the New Zealand setting. Furthermore, there has been no work on the 'obesogenic' environment around schools—despite some work on assessing the obesogenic environment inside primary schools.¹³ Therefore, this study was a first attempt to examine the food advertising and food availability environment around secondary schools in New Zealand.

Methods

Region and school selection—The urban and rural regions selected for this pilot study (Wellington, Lower Hutt, Upper Hutt, and Porirua cities as well as the Wairarapa region) were a convenience sample. Rurality was defined as towns with a population of less than 20,000 people, with all towns in the Wairarapa region being within this range.

Only secondary school neighbourhoods were sampled, because:

- Adolescents generally have more spending power than younger children (via pocket money and earnings); and
- They have more freedom to move around the neighbourhood.

The sample of 10 schools (6 urban and 4 rural schools) were randomly selected from a sample frame of secondary schools at each extreme of the socioeconomic distribution (i.e. the top and bottom two deciles for urban schools, and the top and bottom halves for the rural schools—giving five schools, each, in the two socioeconomic categories). This sample frame excluded schools located in central business districts (CBDs) as well as rural schools that were outside towns (i.e. completely rural settings).

The socioeconomic rankings were from the Ministry of Education classification system¹⁴ that is based on the socioeconomic status (SES) of the children who attend the school. It therefore gives a general measure of the SES of the school neighbourhood. Decile 1 is the lowest ranking and represents the most deprived SES grouping, while decile 10 represents the wealthiest grouping.

The urban schools and their decile scores were: Hutt International Boys School (10); Onslow College (10); St Patrick's College (Silverstream) (9); Naenae College, (2); Mana College (2); and Porirua College (1).

The rural schools were: St Matthew's Collegiate (Masterton), (10); Solway College (Masterton), (9); Kuranui College (Greytown), (6); and Makoura College (Masterton), (4).

Neighbourhood search strategy—Using a global positioning system (GPS) device, the main school gates were spatially located and then the 1-km radius area around the schools was systematically searched for food and non-food product advertisements and shops/outlets (during December 2004 and January 2005). A map was used in conjunction with the GPS device to allow a systematic search along every street within the defined area. The 1-km radius was an arbitrary distance but it is within the range for which young people could readily walk to school or to food outlets (e.g. to buy food during the school lunch-hour). It compares to the 1000-foot buffer zone for restrictions on outdoor tobacco advertising used in the USA.¹⁵

Advertisement definition—Outdoor advertisements were defined as stationary objects containing either a recognisable logo and/or an intended message. These included billboards, neon signs, posters, stickers, free-standing signs, banners, painted buildings, bus shelter advertisements, flags, and images in shop windows designed for viewing from outside (i.e. advertisements on buses and delivery vehicles were excluded).

Only advertisements for products or types of products were included—so signs for services and entertainment activities (i.e. airfares/travel, banking, dry cleaning, flybuys, gambling, hairdressing, movies, sports activities, medical and veterinary care, and video rentals advertisements) were excluded.

Outlet definition—Outlets were defined as places primarily offering food or non-food products for sale. This definition excluded those establishments selling primarily non-food services (e.g. lawyer's chambers, doctor's and vet surgeries, land agents, travel agents, hair salons, betting shops, and car repair shops).

Data collection—Data was collected on all product advertisements with regard to the product, brand, size, distance from the secondary school gate (GPS reading), and location. A digital photograph was also taken of all food advertisements, defined as those with food being the major product advertised (and including alcohol, coffee, and water).

At each food outlet, data were collected on its name, description of contents/products sold, distance from the secondary school gate (GPS reading), and a photograph was taken. All the data were entered into a Microsoft Excel spreadsheet and analysed with the EpiInfo (CDC, Atlanta) software package. All rate ratios calculated were adjusted for neighbourhood SES and/or rurality (as appropriate).

Nutritional classification—A system was developed to classify all of the advertised food products. All advertisements were classified as 'healthy' unless they were for:

- A food specifically listed as a high fat, salt and/or sugar food in the *New Zealand Food and Nutrition Guidelines for Adolescents*¹⁶—including chocolate bars, muesli bars, potato chips/crisps, french fries, doughnuts, pies, sweets, fruit leathers, and soft drinks.
- An alcoholic beverage (also specifically not recommended in the above *Guidelines*).
- A frozen confectionary product that was not labelled 'low fat' (since the *Guidelines* recommend 'low fat' versions of dairy products) or was a high sugar product (e.g. an ice block).
- Food from fast food franchise chains, takeaway outlets, or outlets selling pizzas; where the advertisement did not indicate that the outlet sold salad or vegetable options.

As a conservative approach was taken, other foods that had a mix of desirable and undesirable nutritional characteristics were all classified as 'healthy' in this analysis. These foods were: juices; sports drinks; diet soft drinks; milkshakes; flavoured milk; coffee; food from bakeries (unless just pies were advertised), cafés, and restaurants; and all staple foods (including bread made from refined flour and meat regardless of its fat content).

Results

Extent of advertising in the school neighbourhoods—A total of 1408 advertisements for products were documented outdoors in the areas surrounding the 10 schools. Of these advertisements, 61.5% (95% confidence interval (CI)=58.9–64.0) were for food products (Table 1). This equates to an average of 87 outdoor food

advertisements in the 1-km radius surrounding a school (i.e. 28 per square kilometre). In all but one of the school neighbourhoods, food advertisements were more common than non-food product advertisements (Table 1). The proportion of advertising for food was significantly greater in high SES (wealthier) neighbourhoods (rate ratio [RR]=1.18; 95% confidence interval [CI]=1.03–1.34; $p=0.01$). However, food advertisements in low SES neighbourhoods were significantly closer to the secondary schools relative to those in high SES ones ($p<0.0001$).

Characteristics and setting of food advertisements—A majority (68.9%) of food advertisements were in the ‘large’ category (at least the size of an A1 sheet of paper—59 x 84 cm). There were proportionately more food advertisements than non-food advertisements in this large size category (68.9% vs 43.2%; $p<0.00001$).

Most of the food advertisements were associated with dairies/convenience stores (52.2%), and the rest were associated with other outlets (44.3%) or in other settings (3.5%)—i.e. on bus shelters, or stand-alone billboards.

The major categories of advertised food were: soft drinks (21.6%), frozen confectionary (16.2%), savoury snacks (11.4%), and alcohol (8.1%) (Table 2). Some of the food categories comprised a significantly greater proportion of the food advertisements in high SES neighbourhoods than low SES ones (i.e. foods from takeaway outlets [RR=1.54], foods from fast food franchise outlets [RR=1.67], and alcohol [RR=1.50] [$p<0.001$ for each]). However, advertisements for staple foods were relatively more common in low SES neighbourhoods (RR=2.04, 95%CI=1.54, 2.69; $p<0.0001$). The proportion of frozen confectionary foods advertisements was higher in rural than urban neighbourhoods ($p=0.048$).

Out of the top-10 advertisements with branded products, 6 were for food products. Out of all the food products, the top-10 brands were *Coke/Coca Cola* (17.6%), *Tip Top* ice cream (10.4%), *Meadow Fresh* dairy foods (3.9%), *Cookie Time* cookies and snack bars (2.4%), *Burger King* (2.0%), *Streets* ice cream (1.8%), *Tui* beer (1.7%), *Anchor* dairy foods (1.6%), *Mrs Macs* meat pies (1.5%), and *V* (caffeinated) energy drink (1.5%).

Nutritional classification of food advertisements—Overall, 70.2% (95% CI=67.0%–73.2%) of the food advertisements were categorised as ‘unhealthy’—i.e. inconsistent with nutritional guidelines (Table 3). High SES (wealthier) neighbourhoods had significantly more ‘unhealthy’ food advertisements compared to low SES ones (RR=1.46; 95%CI=1.20–1.76; $p<0.001$).

The analysis for just large advertisements also showed this pattern ($p=0.002$). There were no statistically significant differences by rurality. The majority (80%) of the top-10 branded food advertisements were also in the ‘unhealthy’ category.

Outlets in the school neighbourhoods—There were 224 outlets selling products in the sampled neighbourhoods of which 56.3% primarily sold food and 67.9% sold at least some food (e.g. petrol stations selling fuel, food, and other groceries) (Table 4). Rural neighbourhoods had relatively lower proportions of outlets selling primarily food compared to other product outlets (RR=0.57; 95%CI=0.36–0.92; $p=0.01$). This was also the pattern for outlets that sold any food (RR=0.55; 95%CI=0.35–0.86; $p=0.007$). Furthermore, there was a higher proportion of outlets selling alcohol in the high SES neighbourhoods (RR=1.80; 95%CI=1.44–2.25; $p=0.001$).

Table 1. Distribution of outdoor food advertisements in the 10 secondary school neighbourhoods (1-km radius area) studied in the Wellington and Wairarapa regions

School (around which the neighbourhood was defined)	Food and non-food product advertisements*	Food advertisements	Proportion of product advertisements for food	Distance of food advertisements from the school gate (m)		
	n	n	%	Mean	Median	Range
Hutt International Boys School	149	98	65.8	660	791	315–993
Kuranui College	44	24	54.5	377	337	300–718
Makoura College	80	42	52.5	486	471	305–766
Mana College	201	77	38.3	553	593	118–930
Naenae College	351	228	65.0	580	605	37–827
Onslow College	145	96	66.2	777	828	234–990
Porirua College	141	110	78.0	691	684	355–950
Solway College	84	62	73.8	734	746	637–841
St Matthew's Collegiate	80	44	55.0	833	910	684–995
St Patrick's College (Silverstream)	133	85	63.9	337	229	31–937
All schools	1408	866	61.5	613	675	31–995
All high SES schools (deciles 6–10)	591	385	65.1	650	746	31–995
All low SES schools (deciles 1–5)	817	481	58.9	583	640	37–950
All urban schools	1120	694	62.0	604	671	31–993
All rural schools	288	172	59.7	650	684	300–995

*The definition of food in this analysis includes 'alcoholic beverages', water, and coffee; SES=socioeconomic status.

Table 2. Categories of the outdoor food advertisements (N=886) and distribution by socioeconomic status and rurality of the neighbourhood (all 10 school neighbourhoods)

Food category	All areas		High SES areas	Low SES areas	Urban areas	Rural areas
	n	%	%	%	%	%
Soft drinks	187	21.6	18.4	24.1	22.5	18.0
Frozen confectionary (e.g. ice cream)	140	16.2	16.6	15.8	14.8	21.5
Savoury snacks (e.g. pies, potato crisps, bakery goods [excluding bread]).	99	11.4	10.6	12.1	11.4	11.6
Alcohol	70	8.1	11.9	5.0	7.5	10.5
Other staples (e.g. meat, fruit/vegetables)	70	8.1	0.5	14.1	9.2	3.5
Foods from takeaway outlets (e.g. fish & chip shops)	65	7.5	11.2	4.6	7.3	8.1
Milk	61	7.0	7.0	7.1	6.1	11.0
Food from fast-food franchise outlets (e.g. <i>McDonalds</i> , <i>KFC</i> , <i>Burger King</i>)	50	5.8	9.4	2.9	5.6	6.4
Bread	32	3.7	2.6	4.6	3.9	2.9
Other drinks (e.g. coffee, flavoured milk, water, sports drinks)	29	3.3	4.4	2.5	3.5	2.9
Snack confectionary	26	3.0	2.6	3.3	3.3	1.7
Food and meals from restaurants, cafés, or bars	23	2.7	2.1	3.1	3.0	1.2
Juice	14	1.6	2.6	0.8	1.9	0.6
Total	866	100	100	100	100	100
All staples (milk, bread, and 'other staples').	163	18.8	10.1	25.8	18.8	17.4

Note: Percentages do not add up exactly to 100.0% due to rounding; KFC=Kentucky Fried Chicken.

Table 3. Number and proportion of outdoor food advertisements (N=866) classified as 'unhealthy' in relation to socioeconomic status and rurality of neighbourhoods

Size of food advertisement	All areas		High SES areas	Low SES areas	Urban areas	Rural areas
	n	%	%	%	%	%
All sizes	608	70.2	77.9	64.0	68.6	76.7
Only 'large' size*	441	73.9	79.6	68.9	73.7	74.6

*The size of an A1 sheet of paper (59 x 84 cm) or larger; SES=socioeconomic status.

Table 4. Distribution of food and alcohol outlets compared to other product outlets (by socioeconomic status and rurality of neighbourhoods) and by distance from the secondary school (n=224 for all outlets)

Outlet type	All areas			High SES areas		Low SES areas		Urban areas		Rural areas	
	n	%	Mean distance (m)	%	Mean distance (m)	%	Mean distance (m)	%	Mean distance (m)	%	Mean distance (m)
Primarily food	126	56.3	614	57.1	647	55.5	584	60.0	592	42.9	727
All other outlets	98	43.8	684	42.9	764	44.5	617	40.0	641	57.1	792
Any food	152	67.9	618	70.5	653	65.5	585	71.4	596	55.1	723
All other outlets	72	32.1	701	29.5	802	34.5	625	28.6	651	44.9	815
Alcohol	23	10.3	718	18.1	724	3.4	689	9.1	639	14.3	898
All other outlets	201	89.7	636	81.9	691	96.6	595	90.9	609	85.7	742

SES=socioeconomic status.

Table 5. Extent of meal availability from outlets selling primarily food (N=126), and the proportion of advertised meals with salad options*

Outlet characteristic	All areas		High SES areas		Low SES areas		Urban areas		Rural areas	
	n	%	n	%	n	%	n	%	n	%
Meals available	74	33.0	40	38.1	34	28.6	62	35.4	12	24.5
Salad option advertised	52	70.3	33	82.5	19	55.9	43	69.4	9	75.0

*Meals include either lunch or dinner; SES=socioeconomic status.

Outlets that primarily sold food were (on average) 70 metres closer to the secondary schools than other outlets ($p=0.03$). This was also the case for any outlets selling food ($p=0.02$).

Of all the outlets, 33.0% sold (lunch or dinner) meals (Table 5). There were higher proportions of these outlets in the high SES neighbourhoods and in the urban ones (but this difference was not statistically significant). However, the proportion of these outlets that advertised a salad option as part of the meal was significantly lower in the low SES neighbourhoods ($RR=0.52$; 95% $CI=0.28-0.96$; $p=0.006$).

Discussion

Main findings and interpretation—This pilot study found that a majority (61.5%) of outdoor advertisements for products in the neighbourhoods of these schools were for food. The density of these advertisements (28 per square kilometre) is, however, a probable underestimate of the total outdoor food advertisement level—as this study did not include advertisements on vehicles such as buses or delivery vans. The level of outdoor advertising is also only a small part of the total level of food advertising (e.g. when considering television, radio, print media, and advertising within outlets and on product packaging).

Overall, 70.2% of food advertisements were for foods classified as ‘unhealthy’ (i.e. inconsistent with national nutritional guidelines for adolescents). This may also be an underestimate given that the classification system used was conservative (e.g. all bread, all meat, juices, and sports drinks were classified as ‘healthy’). This is despite some of these being high in sugar, some meat products being high in saturated fat, and most bread being made from refined flour. Furthermore, 8 of the top-10 food brands were for foods that did not fit with the nutritional guidelines.

The higher proportion of ‘unhealthy’ food advertisements in the high SES neighbourhoods may reflect the higher levels of disposable income among adults (and probably children) in these areas. This disposable income reason may also explain the advertising patterns for some food categories (e.g. proportionately more advertising for [relatively expensive] alcohol in high SES neighbourhoods, and for [relatively cheap] staple foods in low SES neighbourhoods). Despite such SES patterns, the high prevalence of food advertising is still concerning when considering low SES children—as these children may be at the highest risk of obesity and may be less likely to bring food for lunches from home.²

Our study also found that a majority (56.3%) of the outlets in these neighbourhoods sold food. The reason why food outlets were significantly closer (on average) than non-food outlets to the secondary schools, may be because location near a school provides significant extra sales for these food outlets.

A significantly lower proportion of the meal outlets in low SES neighbourhoods advertised a salad option than those in higher SES ones. This finding, along with that of a greater proportion of the outlets in high SES neighbourhoods selling alcohol, may also be explained in terms of differing levels of disposable income. Work in other countries has also reported that poor social opportunity¹⁷ and rurality¹⁸ can adversely affect access to healthy foods. For example, an Australian study found that people living in low SES categories had higher exposure to fast-food outlets.¹⁹

Our study also found that slightly more food advertisements were in the ‘large’ size category relative to non-food advertisements. This is a similar finding to that from a study of outdoor alcohol advertising in New Zealand.²⁰

Study limitations—As this was a small pilot study in just two regions of New Zealand (Wellington and Wairarapa), the results may not be generalisable to the rest of the country. Furthermore, it was limited to a cross-sectional design that cannot detect temporal patterns. For example, there could be seasonal variation in the level of some forms of advertising (e.g. more soft drinks and ice creams in summer). Nevertheless, frequent changes in the larger advertisements (i.e. the majority of them) would seem unlikely as many of these are probably fairly expensive.

As detailed above, the definition of ‘unhealthy’ food was conservative and so may not fully reflect state-of-the-art nutritional recommendations (e.g. as recently developed for the United States²¹). In addition, the definition for SES for the neighbourhoods was somewhat limited as it was based on the SES of the children attending the school (i.e. if the children tended to come from a wide area then their SES may not correlate well with the area directly around the school).

A more sophisticated analysis could also use small-area measures of deprivation (e.g. NZDep). It could also consider exposure to outdoor advertising arising from:

- School policies around permitting pupils to leave the school grounds to buy their lunch; and
- How pupils travel to school (since students who walk are likely to have higher exposure compared to those who bus or are driven).

Research and policy implications—Given the limitations above and the pilot nature of this study, it is clear that further methodological refinements could be pursued in future studies. The most important of these refinements would probably be to randomly sample school neighbourhoods around the country and to better place outdoor food advertising into a context of total food advertising exposure (such as relative to television). Refining the classification tools (for food nutritional quality and neighbourhood SES) and collecting exposure data are also desirable to establish a better baseline upon which future monitoring can occur.

Despite the pilot nature of this study, it has provided some initial information about the prevalence and the relatively ‘unhealthy’ content of food advertising in secondary school neighbourhoods. These findings provide tentative support for responses by policy-makers to reduce aspects of the ‘obesogenic’ environment.

One of these responses could be restrictions of certain forms of food advertising in the vicinity of schools (as done with tobacco advertising in the USA¹⁵). Moreover, regulations (or even taxes) could be used to shift the balance of advertising towards foods that meet nutritional guidelines. Indeed, advertising has sometimes been a force for improving the New Zealand diet (e.g. industry marketing of low-fat milk, low-salt foods, and olive oil products).

A supplementary approach may be to follow the example of cigarette packet warnings and require such warnings on unhealthy food advertisements along with permitting ‘signposting’ that indicates when a food is compatible with nutritional guidelines.⁵

However, a coordinated approach may be needed so that further controls in one domain, such as outdoor advertising, does not lead to further advertising growth in other media, such as television and Internet advertising.

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Student access to primary health care and preventive health screening at a school-based health centre in South Auckland, New Zealand

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Abstract

Aims. To determine where students usually access primary health care and compare the quality of preventive health services that students who use the school-based health centre (SBHC) receive to those who go elsewhere for health care.

Methods. A convenience sample of 20 classes were selected and surveyed in 2003. Three hundred and forty-three students completed the Young Adult Health Care Survey using a web-based questionnaire.

Results. While most students (79%) access health care from their family doctor, a significant number (40%) of students attended the SBHC in the last 12 months. Overall, health screening and preventive counselling from health care providers was low. Students who used the SBHC were more likely to receive private and confidential health care and preventive screening than students who go elsewhere for health care.

Conclusion. School-based health care provides additional access to health care that does not appear to replace traditional family practice based health care. While the SBHC appears to deliver better quality preventive health services for adolescents compared to traditional primary health care, improvements are needed across all primary health care settings.

Young people face significant threats to their health and wellbeing. Results from Youth2000, a nation-wide survey of New Zealand secondary school students, show that a significant number of students engage in behaviours that threaten their health such as unsafe sexual activity, suicide attempts, and substance use.¹

In New Zealand, *The Primary Health Care Strategy* emphasises that quality primary health care services should be comprehensive and involve health promotion, education, and counselling to help people adopt healthier lives.² Yet international research suggests few primary health care providers discuss or provide preventive health counselling and education to adolescents on important youth health issues³⁻⁶—despite research showing that (among adolescents) preventive counselling in primary care settings reduces health risk behaviours,^{7, 8} reduces teen pregnancy rates,⁹ improves contraceptive use,^{10,11} and reduces tobacco use.¹²

Access to health care has been defined by the Institute of Medicine, as ‘the timely use of personal health services to achieve the best possible health outcome.’¹³ A large body of research has shown that access to health care by adolescents is poor (for reviews see Brindis^{14,15}). Of major concern is the fact that among New Zealand secondary school students, about half have had problems accessing health care in the past year.¹

One of the main reasons students report that they do not access health care is lack of easily accessible health services: including no transport, lack of resources to pay for health care, and/or poor availability of health care. Students also go without health care when they have concerns about the quality of the care they receive, specifically concerns about privacy, confidentiality, and/or not feeling comfortable with the health care provider.^{1,16}

School based health services have been established recently in New Zealand to meet the health needs of young people, recognising that school-based services may offer more accessible and youth-orientated care. Research from overseas has consistently shown that school-based health care enhances access to primary health care, especially for mental health services.^{17,18}

Little is known about the quality of school-based health services and whether school-based health centres (SBHCs) offer more youth-appropriate health care; specifically health care that is private, confidential, and provides preventive screening and anticipatory counselling for the leading causes of adolescent mortality and morbidity.

In New Zealand, SBHCs have been formed in partnership between local primary health care providers and schools. *The Primary Health Care Strategy* emphasises that improvement in quality of health care requires good information. The current study was designed to provide data on the quality of health care students receive at a school with a SBHC using a youth-health-services questionnaire administered to students over the Internet.

The aims of the current study were to:

- Determine where students attending a school that has a SBHC usually access primary health care; and to
- Compare the quality of preventive health services that students who use the SBHC receive to those who go elsewhere for health care.

Methods

Questionnaire development—The questionnaire was adapted from the Young Adult Health Care Survey (YAHCS)¹⁹ which includes measures on adolescent preventive counselling and screening. The YAHCS is designed to assess the quality of health care received by adolescents and adherence to adolescent preventive services guidelines.²⁰

As some of the questions from the YAHCS were inappropriate for the New Zealand context (e.g. chewing tobacco), they were modified for use in New Zealand. Students from health-curriculum classes helped in revising and adapting the questionnaire through cognitive testing of questionnaire items.

Measures in the final questionnaire included:

- Communication and experience of health care
- Private and confidential visits.
- Preventive screening and counselling on substance use, injuries and violence.
- Preventive screening and counselling on sexual health and sexually transmitted infections (STIs).
- Preventive screening and counselling on health issues on weight, nutrition, and physical activity.
- Preventive screening and counselling on emotional wellbeing and relationship issues.

- Helpfulness of counselling provided on specific health issues such as the risks of smoking and alcohol, how and why to use contraception and condoms, and help with quitting use of illegal drugs.
- Overall rating of health care.

The final questionnaire included 132 items that were then incorporated in to a web-based questionnaire. The visual design of the web-based questionnaire included art work from school students to reflect their local school culture. Students also participated in the recording of audio files of the questionnaire. The final questionnaire can be accessed at <http://www.estream.co.nz/surveydemo> (and click the submit button).

Study population—A secondary school, with a recently established SBHC serving predominantly low-income, Pacific, Maori, and Asian students, agreed to participate in the current study. The SBHC was established in 2002 in partnership with a local Pacific health provider. School classes were conveniently selected from each school year based on availability of the computer classroom. Twenty classes were surveyed in October and November, 2003. Participating classes attended the school computer room where the study was conducted. Each student had their own computer to work from and students were seated to maximise privacy and confidentiality. The questions were displayed on the computer screen and at the same time read out over headphones. Answers were made by point-and-click with the mouse. The computer design allowed for branching questions whereby the participant's responses determine the next question. Upon completion of the survey, students were able to continue with school work on the computer. Of the 382 students present on the day of the survey, 343 completed the survey, 33 students declined to participate or quit without completing the questionnaire, and 6 files were corrupted—thus resulting in an overall student response rate of 90%.

Consent—Ethical approval of this study was obtained from the Auckland Health and Disability Ethics Committee. The principal, teachers, and kaumatua (Maori leaders) of the school were involved in the design and implementation of study. The Board of Trustees agreed with the study protocol. Parents were informed of the study through the school newsletter, and any parents who did not want their child to participate were able to withdraw their child from the study. At the beginning of each class, the study was explained and time was given for questions and answers. Students indicated their consent at the beginning of the questionnaire. Students who declined to participate were able to carry on with schoolwork on the computer during the survey.

Analysis—Estimated proportions and cumulative numbers of students accessing health care providers are presented with their standard errors. Bivariate analysis examined the relationship between accessing the SBHC and demographic characteristics of students using chi-square tests of independence. The Statistics New Zealand ethnicity prioritisation method was used to determine ethnicity groupings.²¹ Estimated proportions of respondents who received preventive and/or counselling on YAHCS topics are presented with their standard errors. Internal consistency reliability of derived YAHCS measures was assessed through Cronbach's alpha (Table 3). An overall percentage score is calculated from the mean of each measure's items, except for the private and confidential-care measure which was assessed through the proportion of respondents who received both private and confidential care. The average percentage/score for each YAHCS measure was calculated for students who *usually use* the school health centre for health care, students that *have used* the school health centre, and students who went *elsewhere* for health care. Multivariate linear regression was used to test for significant differences between YAHCS measures and these three groups, controlling for the age, sex, and ethnicity variables. All standard errors, chi-square tests, and multivariate regression estimates were adjusted for the clustering of students within classes using SAS procedures SURVEYFREQ, SURVEYMEANS, and SURVEYREG (release 9.1, 2005; SAS Institute Inc., Cary, NC, USA).

Table 1. YAHCS measures and percentage of students who have discussed specific health topics with a doctor or nurse in the last 12 months

Questionnaire items and YAHCS measures (internal reliability)	n	%	SE
Communication and experience—6 items (0.76)*			
Reception staff at the health clinic or doctor's office helpful as you think they should be	271	73.4%	2.9%
Doctors or nurses listen carefully to you	273	82.4%	1.9%
Doctors or nurses explain things in a way that you can understand	274	75.2%	2.9%
Doctors or nurses show respect for what you have to say	276	83.3%	2.6%
Doctors or nurses spend enough time with you	270	64.8%	2.3%
Overall average percentage of positive experiences of health care	276	65.1%	1.4%
Private and confidential health care—2 items			
Spoke with doctor or nurse in private	271	52.0%	3.0%
Doctor or nurse explained confidentiality	266	54.5%	3.1%
Overall percentage of both private and confidential health care	264	36.4%	3.0%
Preventive screening and counselling on health risk behaviours (substance use, violence, and injuries) —8 items (.86)			
Smoking cigarettes	276	40.6%	3.0%
Alcohol use	275	38.2%	2.9%
Marijuana use	276	35.1%	2.1%
Street drug use(P, speed, ecstasy)	274	30.3%	2.2%
Sexual or physical abuse	275	25.8%	2.9%
Violence prevention	276	18.5%	2.6%
Riding in cars with a drunk driver	276	16.7%	2.3%
Using a helmet for skateboarding or bicycling	275	14.2%	2.8%
Overall average percentage of risk behaviours discussed	276	27.5%	1.9%
Preventive screening and counselling on sexual health and STIs—3 items (.66)			
Condom use	271	31.7%	2.8%
Sexually transmitted infections (STIs)	274	27.7%	2.7%
Birth control	270	15.9%	2.2%
Overall average percentage of sexual health behaviours discussed	275	25.5%	2.1%
Preventive screening and counselling on weight, nutrition and physical activity—3 items (.70)			
Physical activity or exercise	276	47.1%	3.0%
Healthy eating or diet	276	39.5%	2.9%
Your weight	276	26.8%	2.7%

Questionnaire items and YAHCS measures (internal reliability)	n	%	SE
Overall average percentage of nutrition and activity behaviours discussed	276	37.8%	2.3%
Preventive screening and counselling on emotional health and relationship issues—6 items (.74)			
How's school going	276	56.2%	3.0%
How things are at home	276	37.0%	2.9%
Your emotions or moods	276	32.6%	2.8%
Your friends	276	24.6%	2.6%
Suicide	275	10.9%	1.9%
Being gay or straight	275	7.6%	1.6%
Overall average percentage of emotional health issues discussed	276	26.5%	1.6%
Helpfulness of counselling on specific health issues—5 items (N/A[†])			
How to use condoms to prevent pregnancy and sexually transmitted infections	78	89.7%	4.5%
How and why to use birth control	34	85.3%	7.2%
Understanding the risks of cigarettes or smoking to health	79	75.9%	5.2%
Understanding alcohol use and its risk to health	70	81.4%	2.9%
Help on quitting street drugs	21	81.0%	9.1%
Overall average percentage of students who responded their discussions were helpful or very helpful	142	81.9%	3.8%
Overall rating of health care (range 0 to 10)**	273	8.7	0.13

*Always or usually; ** 0=worst health care possible 10=best health care possible; [†] Too few respondents to measure internal reliability; YAHCS=Young Adult Health Care Survey; N/A=not applicable; SE=standard error.

Table 2. Demographic characteristics of students and place of health care (SBHC=school-based health centre)

Characteristic	Total		No health care in last 12 months		Went elsewhere for health care in the last 12 months		Have used the SBHC for health care in the last 12 months		Use SBHC for usual health care in the last 12 months	
	n	%	n	%	n	%	n	%	n	%
Gender	343	100	62	18.4	155	46.0	95	28.2	25	7.4
Male	156	45.9	26	16.9	84	54.5	37	24.0	7	4.5
Female	184	54.1	36	19.8	71	39.0	57	31.3	18	9.9
									p=0.003	
Age (years)										
13	45	13.2	10	22.2	22	48.9	12	26.7	1	2.2
14	96	28.1	16	17.2	48	51.6	25	26.9	4	4.3
15	78	22.8	13	16.7	37	47.4	20	25.6	8	10.3
16	61	17.8	11	18.3	28	46.7	18	30.0	3	5.0
17	62	18.1	12	19.7	20	32.8	20	32.8	9	14.8
									p=0.3	
Ethnicity										
Asian	45	13.3	6	14.0	26	60.5	8	18.6	3	7.0
Maori	40	11.8	6	15.0	18	45.0	12	30.0	4	10.0
European/ Other	23	6.8	7	30.4	10	43.5	4	17.4	2	8.7
Pacific	231	68.1	42	18.3	100	43.7	71	31.0	16	7.0
									p=0.5	

Table 3. Place of health care utilisation

Variable	Usual place of health care			Health service/provider accessed by students in the past 12 months		
	n	%	SE	n	%	SE
Family doctor's office	266	79.4	3.0	276	82.1	2.5
School health centre	31	9.3	2.2	132	39.3	3.0
Hospital clinic	16	4.8	1.4	32	9.5	1.8
After-hours accident and emergency	6	1.8	1.0	17	5.1	1.1
Hospital emergency department	4	1.2	0.7	27	8.0	1.7
Nowhere	5	1.5	0.6	14	4.2	1.0
No one particular place	5	1.5	0.6	–	–	–
Family planning	1	0.3	0.3	4	1.2	0.6
Other health provider (e.g. alternative therapist, traditional healer)	1	0.3	0.3	19	5.7	1.3
Total	335			521		

SE=standard error.

Table 4. Mean score of YAHCS measures by place of health care

YAHCS Measure	Went elsewhere for health care in the last 12 months			Have used the SBHC for health care in the last 12 months			Use SBHC for usual health care in the last 12 months			
	n	Average	SE	n	Average	SE	n	Average	SE	p value
Communication and experience of health care	155	63.0%	1.8%	95	68.4%	2.1%	25	66.7%	5.8%	ns
Both private and confidential health care	147	23.8%	4.6%	91	47.3%	4.7%	25	72.0%	8.8%	<0.0001
Preventive screening and counselling on risk behaviours (substance use and injury)	155	27.4%	2.8%	94	26.7%	4.8%	25	31.0%	8.2%	ns
Preventive screening and counselling on sexual health and sexually transmitted infections	155	21.9%	3.4%	93	26.5%	4.8%	25	45.3%	8.5%	0.03
Preventive screening and counselling on weight, nutrition and physical activity	155	31.4%	4.2%	94	45.4%	4.4%	25	49.3%	9.4%	0.03
Preventive screening and counselling on emotional health and relationship issues	155	25.5%	2.2%	94	24.9%	2.6%	25	36.0%	7.1%	ns
Helpfulness of counselling on specific health issues	63	74.4%	6.9%	37	89.6%	3.2%	15	85.0%	6.6%	ns
Overall rating of health care (range 0 to 10)	154	8.7	0.2	94	8.6	0.1	23	9.3	0.6	ns

YAHCS=Young Adult Health Care Survey; SBHC=school-based health centre; ns=not significant; SE=standard error.

Results

Demographics—Table 2 shows (in the previous 12 months) the demographic characteristics of the students who completed the health care survey and the demographic characteristics of students by place of health care. The demographic characteristics of surveyed students were similar to the demographic composition of the school. Most of the students were under 16 (64%) years of age, and most of the students were of Pacific ethnicity (68%).

To examine the demographic characteristics of students who use the SBHC, respondents were grouped into four groups: those that hadn't accessed health care in the previous 12 months; students who had used the SBHC in the previous 12 months, but not regularly; students who used the SBHC for their usual health care in the previous 12 months; and students who had gone elsewhere for health care in the previous 12 months.

Female students were more likely to use the SBHC than male students ($p=0.003$). There were no significant differences by age or ethnicity between these groups of students.

Health care utilisation—Most students (79%) used their family doctor as their usual place of health care, and most students (82%) had been to their family doctor in the previous 12 months (Table 3). The SBHC was the next most common place students accessed health care. Almost 40% of students had attended the SBHC in the previous 12 months, and 9% of students used the SBHC for their usual place of health care. After-hours accident & emergency and hospital emergency departments were infrequently used for health care, with fewer than 10% of students having attended these services in the previous 12 months.

Quality of health care—Only those students (82%) who had received health care in the previous 12 months were asked questions about health care quality. Table 1 shows the eight YAHCS measures and questionnaire items that assessed the quality of health care students received in the last 12 months.

The first measure assessed quality of communication and experience of health care. Most students (82%) said that their doctor or nurse always or usually listened carefully to them, and most students (83%) said that their doctor or nurse showed respect for what they had to say. Fewer students (65%) said that a doctor or nurse always or usually spent enough time with them.

The second measure asked about private and confidential health care. Fifty-two percent of students had spoken with a doctor or nurse in private, and 55% of students had had confidentiality explained to them by a doctor or nurse. Overall, the number of students who had received both private and confidential care was low. Only 36% of students reported that they had spoken with a doctor or nurse in private and that a doctor or nurse had explained confidentiality to them.

Preventive health screening and counselling was assessed by four measures: health-risk behaviours; sexual health; weight, nutrition, and physical activity; and emotional health and relationship issues. Overall, the number of students who had discussed any of these health issues with their health provider was low. Forty percent of students had discussed smoking cigarettes with a doctor or nurse in the previous 12 months,

and 38% had discussed alcohol with their doctor or nurse. Only one in six students reported that a doctor or nurse had discussed riding in cars with a drunk driver.

Fewer than 40% of students have discussed healthy eating and exercise with their health care provider, and only 25% of students had discussions about condom use, sexually transmitted infections, or birth control. About one-quarter of students had discussions about emotional health and relationship issues.

When doctors counselled students on these health issues, most students reported that their discussions were helpful. Students were only asked about the helpfulness of these conversations if they had engaged in the health-risk behaviour and had discussed that behaviour with their doctor or nurse. Among students who discussed with their doctor or nurse how to use condoms to prevent pregnancy and sexually transmitted infections, 90% reported these discussions were helpful or very helpful. Over three-quarters of students who had conversations with their doctor or nurse about substance-use, found these conversations helpful or very helpful.

Health care quality by place of health care—Table 4 compares the eight YAHCS measures on ‘quality of health care received among students who use the SBHC for their usual health care,’ ‘students who have used the SBHC in the past 12 months,’ and ‘students who have gone elsewhere for health care in the past 12 months.’

Students who use the SBHC were more likely to have received private and confidential health care than students who go elsewhere for health care. Table 5 shows that about 70% of students who use the SBHC for their usual care, and 47% who have used the SBHC in the past 12 months, had received both private and confidential health care—compared to 25% of students who go elsewhere for health care.

Students who use the SBHC were also more likely to have had preventive health screening about nutrition, physical activity, sexual health, and sexually transmitted infections than students who go elsewhere for health care. Almost half the students who used the SBHC had discussed sexual health and sexually transmitted infections with their doctor or nurse compared to only 20% of students who go elsewhere for health care.

There were no statistically significant differences between students who accessed health care from different settings on preventive screening for emotional health issues or risk behaviours. There were also no significant differences by source of health care in how positively students rated their experience when receiving health care or in how helpful they found the counselling on specific health topics.

Discussion

The purpose of the current study was to describe where students access primary health care and to assess the quality of health care those students receive comparing students who use the SBHC to those who go elsewhere for health care.

Most students (79%) access health care from their family doctor, and the majority (82%) have seen their family doctor in the previous 12 months. These results are similar to findings from a national survey of youth where 80% of students reported that they had seen a family doctor in the past 12 months.¹ These findings suggest that access to school-based health care doesn’t displace health care from traditional family

doctor settings as the percentage of students who have seen a family doctor in the past 12 months is similar to the national average.

In the current study, 40% of students used the SBHC in the past 12 months, and almost 10% of students use the SBHC for their usual place of health care. These findings are similar to other studies of students with access to SBHCs. Kisker and Brown found that, among students from 19 schools with SBHCs, 44% had used the SBHC in the past year and 15% of students use the SBHC for their usual place of health care.²²

In the current study, rates of health care utilisation at the SBHC are significant, and appear to be in addition to students accessing their family doctor for health care. The additional health care access through the SBHC may reflect previously unmet health needs of students at the school.

Significantly, 18.6% of students reported that they received no health care in the previous 12 months—this highlights the need for SBHCs to liaise with schools about students who may not be presenting to health services but who may have significant health needs (and who often come to attention of the school through truancy or behavioural problems). For example, it is estimated that for students who do not complete high school, over 20% prematurely end their education because of early-onset psychiatric disorders.²³

Overall, the percentage of students who received screening and preventive counselling from health care providers was low, regardless of the setting of care. Fewer than 50% of students had received any health counselling on any one health topic, and (on average) only 25% of students had received counselling on substance-use, violence, injuries, emotional wellbeing, or sexual health—which is alarming as these behaviours are the major contributors to adolescent morbidity and mortality in New Zealand.¹

When students did receive counselling on specific health issues, the majority of students felt that this was helpful. Previous research has shown that young people trust doctors as credible sources of health information and that they want to talk to their doctors about sensitive health issues.²⁴ The low rates of preventive health counselling found in the current study may reflect existing time constraints around the delivery of primary health care in family-practice settings.

In traditional primary-care settings, average consultation times for adolescents are 10 minutes or less, and are shorter than average consultation times for adults.²⁵ However, it has been estimated that the time required for comprehensive adolescent health care, including preventive health counselling, is about 20 to 25 minutes for low-risk adolescents and may be substantially longer if multiple problems are identified.²⁶

In this study, students who used the SBHC were more likely to receive counselling and preventive screening on sexual health, sexually transmitted infections, nutrition, physical activity, and weight issues than students who go elsewhere for health care. Kaplan et al showed that students enrolled in a large managed care organisation in Colorado with access to a SBHC were more likely to have had a comprehensive health supervision visit, and to have received preventive health screening and anticipatory guidance, than students without access to a SBHC.¹⁸

Blum et al compared the quality of adolescent health screening among five different practice settings and found that teen-orientated community clinics screened for substantially more health-risk behaviours than private practice settings.⁴ Furthermore, they suggest that the variation among practice settings may be due to provider characteristics such as variation in training, prior experience, and attitudes towards youth. Indeed, evidence suggests that (in many traditional health care settings) providers are uncomfortable providing health care to adolescents and feel inadequately trained in youth health issues.^{27,28}

In comparison with traditional primary care, youth-orientated health care settings (such as SBHCs) often attract providers interested and/or experienced in youth health. And these youth health providers are more likely to have had received training in youth health. Sanci et al has shown that training in adolescent health issues significantly increases the likelihood that providers screen for adolescent health issues and feel more comfortable providing anticipatory guidance.²⁹ In New Zealand, there are very few opportunities for youth-health training and most health professionals who work with young people have not had training in youth health either at undergraduate or postgraduate levels.

In the current study, students who used the SBHC (compared to students who go elsewhere for health care) were more likely to report that they had spoken with a doctor or nurse in private, and that a doctor or nurse had explained confidentiality to them. It may be that students use the SBHC for health issues that are private and confidential such as STIs and contraception, rather than using traditional primary care settings.³⁰

School-based health care also has the advantage of seeing students without their parents. This would enable SBHCs to more easily provide confidential and private health care. Nevertheless, the percentage of students receiving confidential health care from health settings outside the school was less than one-quarter.

Confidential health care is an important component of high-quality health care for adolescents.³¹⁻³³ Indeed, there is evidence that lack of private and confidential health care is a significant barrier for young people accessing health care, especially for sensitive or potentially embarrassing behaviours.³⁴⁻³⁶ Ford et al showed that when adolescents are assured of confidentiality they are more likely to disclose information, be honest in their discussions with their doctor, and are more likely to return for future visits.¹⁶

One of the strengths of the current study is that it is situated in a school with a high proportion of students from low socioeconomic backgrounds and a high proportion of students identifying with Pacific ethnicities. Pacific peoples (i.e. from Pacific islands such as Samoa and Tonga) are known to have more unmet health needs and more barriers to health care access.³⁷ This study provides important information to address health disparities and improve health care access for Pacific and low-income youth populations. However, the findings from this study are limited by several factors.

Firstly, the cross-sectional nature of the study design limits any conclusions about the causality of the findings. For example, it is possible that students with greater health need self-select to use the SBHC and are therefore more likely to receive anticipatory health counselling.

Secondly, the study was based in only one school and the numbers of students using the SBHC for usual health care was small; there may have been additional differences between health care providers, but this study did not have the statistical power to detect them.

Lastly, the study is also limited by the nature of the self-reported YAHCS questionnaire which may not capture all the preventive health counselling that takes place in primary care settings. At present, adolescent self-report has been shown to be the most valid source of data about the provision of preventive counselling and screening services in primary care.²⁰

Conclusion

This study highlights that school-based health care can provide accessible and appropriate health care that may be meeting previously un-met health need among adolescents. It appears that school-based health care provides additional access to health care that does not replace traditional family practice based health care. While school-based health care in New Zealand is in its infancy, it is gaining significant attention and the Ministry of Health has provided guidelines for schools and health providers interested in setting up SBHCs.³⁸

Training of health providers, and especially primary care physicians interested in working in the school environment, is critical. There are unique qualities of the school environment and working in partnerships with schools is vital to the success of school-based services.³⁹

This study also highlights that improvements are needed in the delivery of high-quality preventive health services for adolescents in primary health care settings in New Zealand. There is a need for both structural changes among primary care settings, with the ability for services to provide longer consultation times and training for health care providers who work with young people on youth health issues, particularly around consent and confidentiality.

Traditionally, primary health care has focused on acute medical care with short consultations and episodic and problem focused care.⁴⁰ The *Primary Health Care Strategy* recognises that for health services to provide preventive health care a paradigm shift towards health promotion and disease prevention is required.² With recent changes in the delivery of primary care in New Zealand and population based funding, clinicians have the opportunity to provide ongoing comprehensive, preventive focused health care that recognises the influence of social behaviours on the health and wellbeing of young people.

The potential impact of better preventive health counselling by primary care providers on the health and wellbeing of adolescents is considerable²⁴ and has been shown to be cost-effective.⁴¹

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Assessing and developing community participation in primary health care in Aotearoa New Zealand: a national study

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Abstract

Aims. This study aimed to develop and test a framework and tool for assessing and developing community participation in Primary Health Organisations (PHOs) in New Zealand.

Methods. A qualitative study completed in three phases: semi-structured interviews with 42 key stakeholders in the primary care sector; development of and consultation on a draft toolkit, which included a PHO review process; and piloting the toolkit in four different types of PHOs.

Results. A toolkit entitled *Community Participation: A Resource Kit and Self-Assessment Tool for PHOs* (CP Toolkit for PHOs) was developed, which contains a set of resources for organisational self-review and a framework for community participation under the six headings: Organisational Structure, Maori Responsiveness, Governance and Management Processes, Use of Resources, Links to the Wider Community, and Consultation & Decision-Making. The pilot PHO sites found the CP Toolkit, and the review process contained within it, to be very relevant to the implementation of the Primary Health Care Strategy at a PHO level.

Conclusions. The Community Participation Toolkit for PHOs complements existing quality tools available for clinical general practice and nursing, and supports the further development of primary health care in New Zealand.

Consumer and community involvement in the planning and delivery of health care is core to the original concept of primary health care, as defined in the Alma Ata Declaration.¹

Primary health care is essential health care based on practical, scientifically sound and acceptable methods and technology made universally accessible to individuals and families in the community through their full participation and at a cost that the community can afford to maintain at every stage of their development in the spirit of self-reliance and self-determination.

The Declaration was the response of the World Health Organization, and its international delegates, to the failure of hospital-centred health care to provide for the basic health care needs of the rural poor in developing countries. It recognised the success of early grassroots primary health care initiatives in South Africa and other developing nations.^{2,3} These initiatives had demonstrated the positive impacts on access, appropriateness, and affordability of involving people in planning their own local health services.²⁻⁴

Many rationales underlie the involvement of consumers and communities in the planning and delivery of health care—although in health policy, these rationales are often implicit. Arguments for participation include the legitimisation of policy, the improvement of service appropriateness, the redistribution of resources, and the reduction of inequalities in health.^{5–7} A fundamental justification in New Zealand is found in the Treaty of Waitangi, which defined political participation as a right of the Treaty partners, Maori as tangata whenua, and the Crown.^{8,9} There is evidence that community participation in health organisations is ineffectual, unless the organisation has individuals within it who are committed to learning and open to change.^{10,11}

There are few rigorous studies that have measured the effects of community participation in terms of health outcomes; however there is evidence of service improvement. A systematic review of the evidence related to involving patients in the planning and development of health care concluded that involving patients contributes to changes in service delivery in several ways across a range of settings.¹² Changes include an improvement of patient information sources and access to services (such as simplified appointment procedures, extended opening times, and improved physical access for people with disabilities) as well as the development of new services (such as advocacy, employment initiatives, and crisis services).

Third-sector (non-government and non-profit) primary care organisations in New Zealand and USA are more likely than private general practices to have community involvement in their governance.^{13–15} Research into third-sector services provides indirect evidence concerning the effects of community involvement.

In New Zealand, community-governed third-sector primary care organisations have been found to serve largely non-European and low-income populations,¹⁴ to have comparatively low user-charges, and to employ large numbers of Maori and Pacific staff.¹⁶ Similarly, the achievements of community-governed third-sector community health centres (CHCs) in the USA in improving access to care for vulnerable populations, providing high-quality primary care, improving health outcomes, and reducing health inequalities have been well documented.^{15,17–23}

Until recently, consumer and community participation in primary care organisations had been a reality for only a small proportion of providers in New Zealand.²⁴ It became an explicit part of government policy with the advent of New Zealand's Primary Health Care Strategy²⁵ and the emergence of Primary Health Organisations (PHOs) in 2002, as demonstrated in the following statements:

PHOs must demonstrate that their communities, iwi, and consumers are involved in their governing processes and that the PHO is responsive to its community.²⁵

The DHB must be satisfied that community participation in PHO governance is genuine and gives the communities a meaningful voice. In addition, DHBs will require PHOs to show how they respond to their communities.²⁶

The implementation of this aspect of the Primary Health Care Strategy has proved challenging not only for many PHOs but also for District Health Boards (DHBs), charged with PHO contracting. While many third-sector primary care organisations have experience at successfully implementing community governance models, community involvement is largely foreign to the for-profit model of general practice, which has been mainstream in New Zealand.

In an environment of privately owned general practice, it is not easy for primary care providers, or even contractors, to adopt a culture that acknowledges the contributions of consumers and communities to health service planning and delivery. From both theoretical and practical perspectives, community involvement in governance challenges the ownership boundaries inherent in a business model.²⁷

Despite clear government policy aimed at introducing community participation in the governance of PHOs, there has been a notable absence of frameworks and tools to aid PHOs in engaging with the communities they serve. This project aimed to develop a framework for assessing and developing community participation in PHOs, and to produce a toolkit for PHOs which would be a practical resource for primary care providers.

Methods

The research project was undertaken using qualitative methodology. Sampling was purposive, with the aim of collecting a diverse sample of key stakeholders from different levels of the primary care sector throughout New Zealand. Initially a snowballing technique was used, beginning with people known to the researcher (PN), followed by heterogeneity sampling for assumed diversity of views. The three key factors sampled for were role in the sector, ethnicity and geography.

In Part 1, participants were asked their views on, and experience of, community involvement in primary care organisations and how one might assess it. In Part 2 and Part 3, the role of participants was principally to offer feedback on the draft toolkit for PHOs.

Data collection was by in-depth semi-structured individual and group interviews; however a written feedback form was used in Part 2 for the toolkit consultation. Nearly all participants were interviewed face-to-face, with a few by telephone. All interviews were audiotaped (with the consent of participants) and subsequently transcribed. All Part 1 participants were sent their own transcripts for correction or comment.

Transcripts of the audiotapes were analysed with the help of NUD*IST qualitative analysis software (N6 version) to manage and code the data. Data analysis involved using a constant comparative method, in which transcripts are repeatedly reviewed to find emergent themes, consistent with a general inductive approach.²⁸ The framework for the community participation toolkit (made up of a series of process indicators) was developed from these themes.

The research was organised around three parts, each with its own method and sample. These three parts are described below.

Part 1—Interviewing key stakeholders in the primary care sector:

- Completion of individual interviews (N=26) and focus groups (N=3) with key stakeholders in the primary care sector (Table 1).
- Development of a draft toolkit for assessing community participation in primary health care organisations, based on the literature and interview data.

Table 1. Part 1 participants by role, ethnicity, and geography (N=42)

Role in the sector	Ministry of Health, district health boards, general practice, primary care provider organisations, primary health organisations, doctors' organisations (IPAC, Royal NZ College of GPs, one IPA), third-sector organisations, other opinion leaders (such as community activists, health sector consultants, and board members of third sector organisations)
Ethnicity	Maori (20), Pacific (1), Non-Maori, non-Pacific (21)
Geography	Urban metropolitan (18), urban provincial (7), rural (17); North Island (41), South Island (1)

IPAC=Independent Practitioners Associations Council: <http://www.ipac.org.nz/index.aspx>

Part 2—Consultation on the draft toolkit:

The aim of Part 2 was to consult on the toolkit with Part 1 participants, and with an even broader group of key stakeholders in the sector. Invitations were sent by email to representatives of key stakeholder groups which had not yet participated in the research. In particular, the medical directors of eight Independent Practitioners Associations (IPAs) were contacted, and all but two of those organisations agreed to participate. Several primary care nursing leaders were also contacted, along with DHB employees working with PHOs.

A feedback form was developed, which invited participants to rate the degree to which they agreed with a series of statements about the toolkit's form, content and potential usefulness to general practice and to PHOs.

Seventy-eight copies of the draft toolkit were mailed with feedback forms to the Part 1 participants and the other stakeholders who had responded positively to the invitation. The consultation period was extended from 1 to 3 months, due to the limited number of early responses. A total of 32 written responses were received from the sector over the 3-month period. Half of the respondents were Part 1 participants. None of the IPAs returned feedback forms, but feedback was received from five primary care nursing leaders, from seven PHOs, and from six different DHBs. Despite the limited response from GP organisations, there was a large amount of useful feedback received from a wide variety of stakeholders, including three GP leaders.

Part 3—Piloting the CP Toolkit in Primary Health Organisations

Part 3 consisted of the following activities:

- Informal invitation to several PHOs to participate in the pilot process.
- Selection of a sample of five PHOs, diverse by size, type and location.
- Negotiation of a realistic pilot process with the interested PHOs.
- Production of an information package and sending out of formal invitations to five PHO boards.
- One or two day visit to pilot sites to facilitate the organisational review process with each PHO.

From a research perspective, it was important to have a diverse group of PHOs involved in piloting the toolkit; diverse by location, size, and history/type. Of the eight PHOs which expressed an interest in participating, five were formally approached. These five included two rural PHOs (one in the North Island and one in the South Island), two urban PHOs (one moderately large, one small) and one very small PHO located in a small town. Two of the PHOs served a high proportion of Maori and one served many Maori and Pacific peoples. Three of the PHOs were funded on the Access formula, one on the Interim, and one on Interim with Access practices. Two of the PHOs grew out of community-based organisations, while the other three grew out of partnerships between IPAs and community providers. Despite initial consent by its board, the South Island PHO subsequently withdrew from the research before the pilot site visit, leaving four PHOs in the pilot phase.

The researcher negotiated with the PHO contact people (usually managers) to arrange meetings with key individuals to trial the toolkit's review process. For the purposes of the pilot process, the full review was adapted by combining the self-review and external reviews. Different groups within each PHO tested different sections of the toolkit. In three PHOs, all six sections of the CP Toolkit were tested. Meetings were held with PHO boards, iwi representatives, community representatives, PHO management staff, and other groups (such as clinical advisory groups).

All meetings were audiotaped and transcribed, and data analysed. In one PHO, the researcher was only able to facilitate a limited review process with one staff and one board member, due to staff time constraints at the time of the visit.

Subsequent to the pilot site visits, each PHO manager responded to an email questionnaire which sought feedback on the usefulness to their PHO of the *CP Toolkit for PHOs*. Data gathered during the pilot process contributed to the final editing of the toolkit.

Results

Part 1—The primary output of this study has been the Community Participation Toolkit for PHOs, which will be published in 2005. For the scope of this paper, it is not feasible to do more than give an overview of the toolkit contents.

The toolkit consists of a set of resources on community participation in primary health care, including defining communities, engaging with Maori communities, and a ladder of participation. It also includes a review process, involving process indicators, that a PHO can use as part of its strategic planning or continuous quality improvement processes. Finally, the toolkit contains a list of references for further exploration of the topic.

Part 2—The feedback from the consultation was mostly very positive and a large majority of respondents could see the relevance and importance of the toolkit for PHOs. A summary of the rating of individual items on the feedback form is shown in Table 2.

Table 2. Part 2 feedback form responses (N=26)

Question	Agree	Neutral	Disagree
User-friendly?	13 (50%)	8 (31%)	5 (19%)
Manageable size?	12 (46%)	8 (31%)	6 (23%)
Clear language?	23 (92%)	1 (4%)	1 (4%)
Inclusive of relevant communities?	7 (29%)	11 (46%)	6 (25%)
Appropriate name?	9 (35%)	9 (35%)	8 (30%)
Important dimensions of CP?	20 (80%)	0 (0%)	5 (20%)
Appropriate for kaupapa Maori organisations?*	2	1	0
Appropriate for Pacific organisations?*	0	1	0
Appropriate for general practice?*	3 (30%)	2 (20%)	5 (50%)
Appropriate for other primary care organisations?*	4	3	1
Use a modified version?	2	4	2
Relevant to PHOs more than individual providers?***	14 (64%)	1 (4%)	7 (32%)
Relevant to individual providers more than PHOs?***	2 (10%)	1 (5%)	17 (85%)
Relevant to both PHOs and providers?***	10 (50%)	5 (25%)	5 (25%)

*Those items which only selected participants were asked to complete, and therefore the number of responses is low; **Confusion over questions 13–15 (which were interrelated) meant that the written feedback was given more weight than the ratings for these items.

There was concern expressed by some that PHOs were in need of a set of resources, but perhaps were not ready to seriously engage in the review process outlined in the *CP Toolkit for PHOs*. Many stated a view that the toolkit was relevant to general practice as well as PHOs, while others stated that it had no relevance to general practice. Many respondents offered suggestions for decreasing the size of the toolkit and making it more user-friendly for PHOs. As a result of the feedback, many changes were made to the toolkit before the second draft was completed for the pilot process.

Part 3—The three pilot PHOs that completed the full adapted review process expressed satisfaction with the process. They felt that the discussion of items in the toolkit's workbook gave them the opportunity to reflect on what their PHO had achieved, to discuss future goals, and to further develop relationships within the PHO

during the process. There was evidence of particular benefit for iwi and community representatives on boards and community advisory committees. They felt that they learned new information about their local PHO through the review process, and gained confidence in their role in the organisation. The fourth pilot PHO expressed regret that they were unable to complete the review due to time limitations, as they felt it would have been beneficial to their organisation.

Following the pilot site visits, the PHO managers' feedback on the toolkit and its review process was very positive, with suggestions that they would use the toolkit in a number of ways, such as for strategic planning, board training, or as performance indicators for PHO management.

Despite the difference in the provider make-up of the four pilot PHOs, in each PHO there was evidence of a genuine commitment to engaging with communities, and each was taking a unique approach to it. In three of the four PHOs, the PHO manager demonstrated strong leadership for community participation. Health promotion coordinators and some board members were also notable advocates. In the fourth PHO, the leadership for community engagement came from a practice manager and a board member. Much of the leadership for community participation in the pilot PHOs came from people with nursing backgrounds.

In this study, there appeared to be some relationship between the level of buy-in that providers had in the PHO and the size of the PHO. In the larger organisations, the PHO was perceived by providers to be external and separate to them. This observation held true not only for GP providers but also community health providers, and in both rural and urban locations. The smaller PHOs demonstrated a stronger identity, regardless of their provider make-up.

There was evidence in the pilot PHOs of tension between the business imperative of running a viable not-for-profit organisation and involving communities in governance and decision-making processes. There were two distinct issues described. Firstly, managers described a lack of adequate funding for PHOs to spend the time necessary to meaningfully engage with communities. Even those PHO managers who were clearly passionate about working more closely with the communities they served stated that PHO management funding in their contract was inadequate to cover the actual cost of community participation.

Secondly, some GPs expressed concern that community representatives on PHO boards could have the potential to make decisions that impacted negatively on their constituent practices. In the three pilot PHOs that had a private GP provider base, the GPs were strongly represented at board level.

Discussion

This research has led to the publication of the *Community Participation Toolkit for PHOs*, which has been preliminarily tested in a small set of PHOs. Consistent with the literature,^{29–32} this research offers further evidence that it is possible to define the processes which health organisations can set in place in attempting to ensure that community involvement benefits communities and health organisations alike. Detailed discussion of the toolkit is the subject of another paper.

While the toolkit is intended for use by PHOs, it may also be a useful resource for District Health Board (DHB) and Ministry of Health personnel responsible for

primary health care funding and planning. The toolkit relies on the commitment of a PHO, its staff, and its board to involving communities in decision-making. The existence of visionary leaders in PHO management and governance, who are committed to working with communities and to developing innovative primary health care services, is an essential ingredient to meaningful community engagement in PHOs. This finding is consistent with previous research.^{10–11} In particular, the UK research on Primary Care Groups demonstrated that leadership for public involvement and openness to organisational change were key factors in public involvement leading to positive change.¹¹

Even with the emergence of the ‘PHO model’, there is clear evidence that different types of PHO lead to different expressions of community engagement. As each PHO has a unique history of local relationships, there can be no ‘gold standard’ for engaging communities in PHOs. There is a particular challenge ahead for large PHOs in which both providers and communities may feel less engaged in the PHO. For them, the first task may be to develop the ‘internal PHO community’ to the point where providers share a vision for the PHO and are open to developing innovative health promotion and health services.

The cost of community participation is a challenge for PHOs, since engaging with communities can be a time-consuming process. Without a doubt, the business imperative of both PHOs and constituent provider organisations can act as a barrier to meaningful community engagement. Furthermore, there are ongoing tensions in the sector due to the issue of ownership boundaries in primary care still remaining unresolved from a policy perspective, as discussed elsewhere in the literature.^{14,27,33}

During the period of this research (2003–2005), there was a notable shift in the views of many key individuals in the primary care sector about the place of community involvement in the planning of primary health care at a local level. At the outset of the project, many participants were uncertain about the relevance of community involvement to primary health care. By the time of the pilot site visits, the leadership for community engagement in PHOs was coming not only from communities, but also from nurses and GPs.

This study had some limitations. Participants were limited in number for practical reasons. There are many advocates of consumer and community involvement who work outside the formal health sector, and their views were not incorporated into this study. There were some key stakeholder groups in the sector that the researcher had difficulty engaging in the study. The pilot process was limited by the timing of the research. Many PHOs were in the early stages of development and managers were coping with the new reporting requirements. This administrative load took precedence, for some, over participating in the research.

As a result, one pilot site pulled out and another did not complete the full review process. Clearly, the pilot process was not a comprehensive testing of the toolkit. To validate the observations made during this research, a much larger evaluation of the toolkit, involving many more PHOs in New Zealand, would need to be carried out. The *CP Toolkit for PHOs* has, however, been reviewed in-depth by three diverse PHOs, and was found to be a useful resource.

As the research employed qualitative methods, it is not possible to generalise the findings described here to all PHOs. Instead, the findings offer insights into some of

the issues facing PHOs with regard to their engagement with the communities they serve. By its very nature, each PHO is unique as there is no 'one size fits all' for primary health care services if they are responsive to their communities.

Engaging with communities is core to the development of innovative services and health promotion in primary health care. The *Community Participation Toolkit for PHOs* has been developed to complement existing quality tools available for clinical general practice and nursing, and is intended to support the ongoing development of primary health care in Aotearoa New Zealand.

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Prevalence of Type 1 diabetes in New Zealanders aged 0–24 years

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Abstract

Aims. The incidence and prevalence of Type 1 diabetes is increasing internationally. There is, however, no current estimate of the prevalence of Type 1 diabetes in young New Zealanders. We therefore aimed to estimate prevalence in 0 to 24 year olds.

Methods. The point prevalence of Type 1 diabetes was determined in a geographically defined area, namely the Canterbury District Health Board catchment area, by comparing data from multiple clinical and research sources. The New Zealand prevalence, stratified by age and ethnicity, was then estimated using 2001 population census data.

Results. There were 353 people with diabetes aged 24 years and less, residing within the catchment area at the time of study. Of these 353 people, 330 had Type 1 diabetes, giving a prevalence of 227 per 100,000 children and young adults. The estimated number of New Zealanders with Type 1 diabetes in this defined age group, adjusted for ethnicity, was 2,540. An estimated 2,158 were of European descent.

Conclusions. Although the prevalence of Type 1 diabetes is lower in non European New Zealanders compared to European New Zealanders, the changing demographics of children and youth in New Zealand means that there are increasing numbers of Maori, Asian, and Pacific peoples with Type 1 diabetes.

For an optimal long-term outcome, Type 1 diabetes in children and young adults requires intensive management by the patient, their family, and also their health professional team. The incidence of Type 1 diabetes has doubled in New Zealand in the last 15 years, mirroring the international trend towards an increasing incidence of Type 1 diabetes.^{1,2} Prevalence of Type 1 diabetes will also therefore be increasing, which has implications for service delivery and planning. There has been a recent estimate of diabetes prevalence in New Zealanders aged 25 and more,³ but there are no current estimates of the prevalence of Type 1 diabetes in New Zealand in children and young adults.

Methods

The number of children and young adults with diabetes, aged 24 years or less and residing in the CDHB (Canterbury District Health Board) catchment area on 1 November 2003, was estimated through review of current databases and medical records.

Diabetes type (i.e. Type 1, Type 2, and other types) was determined by the attending clinician, using immunogenetic testing where appropriate.⁴ Currently, young patients with newly diagnosed diabetes are routinely tested for the diabetes-related antibodies IA2 and GAD, as an aid to diagnostic classification. Selected families may also undergo genetic testing for monogenic forms of diabetes. Patients with transient steroid-induced diabetes were excluded from analysis. Ethnicity was either self-defined or, for younger children, defined by the main carer(s). Locally, younger patients who regularly

attend secondary care diabetes services are not routinely included in the free Ministry of Health primary care diabetes annual check, thus no attempt was made to use primary care records as a method of case identification.

Christchurch Diabetes Services have a database of children and young adults with Type 1 diabetes, assembled from both clinical and research records, as previously described.⁵ The database appears to be near complete, and includes patients not currently attending secondary care services. As a further check of completeness of clinical records, all Christchurch hospital diabetes discharges were reviewed for the 2½ year period prior to undertaking this study.

Community-focussed diabetes healthcare workers were also asked if they knew of anyone with diabetes who was not currently in contact with any hospital service. Some patients attend both Christchurch Hospital and also the Ashburton Diabetes Clinic for their specialist diabetes care, and Ashburton clinical records were also reviewed.

The domicile of people who attended school or university in one location but were resident during holiday periods in one or more different locations, was arbitrarily defined by the location which delivered the majority of their diabetes healthcare.

The prevalence of diabetes in the CDHB catchment area, which includes the city of Christchurch, the town of Ashburton, and rural areas around North Canterbury, was estimated using the latest available census statistics, from 2001.⁶ There are known ethnic differences in the incidence and prevalence of Type 1 diabetes—with Maori, Pacific peoples, and Asians having a lower incidence than those of European descent.^{2,7,8} Also, the age distribution of the CDHB population is skewed towards the elderly when compared to New Zealand as a whole.⁶ When extrapolating local results to the whole of New Zealand, data was therefore stratified by ethnic group and by age bands 0–14 and 15–24 years of age.

The confidence interval on this estimate was derived from the pooled error from each of the age-ethnic group estimates. This audit had local ethics committee approval.

Results

There were 353 children and young people residing in the CDHB catchment area in 2003 with known diabetes. Of these, 330 had Type 1 diabetes and 51% were male. Seventeen of the remaining 23 people had Type 2 diabetes. At the latest census, there was an estimated 145,164 people aged 24 years or younger, residing in the CDHB catchment area. The prevalence of Type 1 diabetes in children and young people was therefore 227 per 100,000 (95% confidence interval 203 to 252). In the age range 0–9 years, prevalence was 99 per 100,000. In the 10–19 age range, it was 261 per 100,000.

Of the 330 people with Type 1 diabetes, 307 were European New Zealanders, 13 were Maori, 3 were Pacific peoples, and 7 were Asian. The estimated prevalence per 100,000 population was therefore 274 for Europeans, 81 for Maori, 77 for Pacific peoples, and 52 for Asians. There were sufficient numbers of Europeans to stratify prevalence data by 5-year age bands. Results are shown in Table 1.

Table 1. Number and prevalence of European New Zealanders with Type 1 diabetes (stratified by age) residing in the Canterbury District Health Board Catchment Area in 2003

Age range (years)	0–4	5–9	10–14	15–19	20–24
Number of patients	7	49	64	92	95
Prevalence per 100,000 population*	33	217	275	403	427

*Age stratified population data obtained from 2001 census.

Extrapolating the above (Christchurch) figures to the whole of the New Zealand population thus produced an estimate of 2,158 Europeans, 231 Maori, 74 Pacific peoples, and 77 Asians with Type 1 diabetes—giving a total number with Type 1 diabetes of 2,540 (95% confidence interval 2250 to 2830).

There were an estimated 1,118 people with Type 1 diabetes in the age range 5 to 14 years, thus approximately 1:516 New Zealand schoolchildren in this age range have Type 1 diabetes. An estimate of the number of young people with Type 1 diabetes in each District Health Board is given in Table 2.

Table 2. Estimated number of young people with Type 1 diabetes by DHB (District Health Board)

DHB	Total population aged <25 years*	Number with diabetes 0–14 years	Number with diabetes 0–24 years
Canterbury**	145,164	129	330
Northland	50,589	39	79
Waitemata	154,515	123	287
Auckland	128,625	74	208
Counties Manukau	153,714	92	214
Waikato	121,392	98	231
Lakes	36,639	26	58
Bay of Plenty	62,586	52	111
Tairāwhiti	17,568	12	25
Taranaki	36,828	34	76
Hawke's Bay	52,158	44	95
Whanganui	23,151	19	42
Midcentral	57,705	47	120
Hutt	48,729	39	90
Capital & Coast	87,186	63	174
Wairarapa	12,942	12	25
Nelson Marlborough	40,140	40	89
West Coast	9,903	11	21
South Canterbury	16,599	18	38
Otago	60,744	50	148
Southland	35,772	34	79
Total	1,352,649	1056	2540

*Population data from 2001 Census; **Measured rather than estimated number with Type 1 diabetes.

Discussion

The incidence and prevalence of Type 1 diabetes in young people is increasing. The prevalence of Type 1 diabetes in Canterbury in 1988 was measured as 30 per 100,000 in the age range 0–9 years and 180 per 100,000 in the age range 10–19 years.⁹ In contrast, the corresponding 2003 estimates (which included Ashburton cases) are 99 and 261 per 100,000, in the respective age ranges. The reason(s) for this temporal increase in prevalence in the younger age group in particular is unclear.

It has been suggested that the age of presentation of Type 1 diabetes may be falling, in association with increased childhood obesity.¹⁰ Between 1970 and 1999, there were 474 incident cases presenting in the region but the age of presentation did not change significantly with time.¹ The absolute numbers of children developing diabetes in

Christchurch is small however. Also, a formal patient tracking study would be required to determine whether temporal change in prevalence data was in part due to disproportionate movement (geographical relocation) by a subgroup of patients, into or out of the CDHB catchment area.

Currently, there are approximately 2,540 children and young people with Type 1 diabetes in New Zealand. These findings have service delivery implications. For example, if around 1:500 schoolchildren have Type 1 diabetes then this has implications for health policy planning in schools, as most secondary schools are likely to have at least one pupil with diabetes. If the rate of change in prevalence continues at its current pace, it is likely to place sufficient burden on both paediatric and adult secondary care services to require new models of service delivery.

The method used to estimate of the number of young people with Type 1 diabetes has several limitations, but the magnitude of any associated bias is likely to be small. For example, the local estimate of the number of people with Type 1 diabetes may not have captured every individual with this condition, but no additional patients were identified either from patient discharge data or by diabetes community workers, who were not already on the clinic database. This suggests that the database was near complete.

It can be hypothesised that people with Type 1 diabetes may preferentially choose to reside in areas where they have ready access to emergency and specialist services, thus they may choose to move from a rural to an urban area, following the diagnosis of diabetes. Undertaking an estimate of Type 1 diabetes prevalence in a DHB with a large rural population may in theory result in an underestimation of the number of people with Type 1 diabetes. The urban:rural split in the CDHB catchment area population is, however, similar to that of New Zealand as a whole. For example, one in seven Cantabrians live rurally, a figure that is identical to that of the total New Zealand population.⁶

There has been a small (3%) increase in the New Zealand population between the last census in 2001 and the time of the study in 2003,⁶ thus the current study is likely to result in a minor overestimate of prevalence per 100,000 population. This slight shift in demographics will not, however, affect the estimate of the absolute number of people with Type 1 diabetes in New Zealand.

Internationally, there are large geographic variations in the incidence and prevalence of Type 1 diabetes.² New Zealand data from the previous three decades suggests that the incidence of Type 1 diabetes is higher in the South Island compared to the North Island,^{2,7,11} but these studies made no or limited attempts to stratify by ethnicity. In contrast, a comparison of incidence data over a 20-year period (1977 to 1996) from Christchurch and Auckland showed mean incidence rates for childhood onset Type 1 diabetes of 16.44 and 11.61 per 100 000, per annum, for each respective city. However this study did not detect any difference in incidence between these South and North Island cities, when results were stratified by ethnicity (Jinny Willis, personal communication, 2004).¹²

Our study found the prevalence of Type 1 diabetes in Europeans to be three to four times higher than non-European New Zealanders. This ratio is similar to the ethnic differences reported in the more recent incidence study.¹² Extrapolation of local prevalence data, stratified by ethnicity, to the North Island population would therefore

seem reasonable. The ideal approach to estimating the prevalence of Type 1 diabetes in New Zealand would of course be for each DHB to undertake its own local study of prevalence, using predefined methodology, then collate results with those from other centres.

This study estimated the prevalence of Type 1 diabetes only, in part because the number of young people with Type 2 diabetes in our local population was low. In regions where there is a high percentage of Maori in the population, Type 2 diabetes in young adults is of increasing importance,¹³ and may represent a major clinical burden in that area.¹⁴ Estimating Type 2 prevalence in young people is, however, likely to be more difficult than estimating the prevalence of Type 1 diabetes. Onset of Type 2 diabetes is usually slower, with a gradual progression from impaired glucose tolerance to frank diabetes, thus there is no clearly defined symptomatic onset of disease requiring the patient to seek medical attention. Also, at least in our local area, not all young people identified as having Type 2 diabetes are referred to specialist services, so case identification for research and audit purposes is more difficult.

In summary, this report gives an up-to-date population-based estimate of the number of children and young people with Type 1 diabetes in New Zealand. Young New Zealanders are from an increasingly multiethnic background and this is reflected in the increasing numbers of non-European New Zealanders estimated to have Type 1 diabetes. Specialist diabetes services, in particular, therefore need to address cultural issues in relation to Type 1 diabetes in ethnic groups, whilst at the same time accommodating the health needs of an increasing number of young people with Type 2 diabetes.

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Teenage use of GP care for moderate to severe asthma in Auckland, New Zealand

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Abstract

Objectives. To describe and understand teenagers' frequency of attendance for General Practitioner (GP) care of moderate to severe asthma in the Auckland region.

Methods. Ten Auckland schools identified 510 children aged 13–14 years with breathing problems, who were invited to complete a screening questionnaire. 271 children participated, of whom 114 had moderate to severe asthma.

Results. 39% of the 114 had made 0–1 GP visit for asthma, and 17% made ≥ 5 visits. Low attendees (0–1 visit) were disproportionately New Zealand European. High attendees (≥ 5 visits) tended to be Maori and/or Pacific Islanders. Half of the teenagers attended GP asthma care as often as it wanted, independently of ethnicity; 62% tell their parents when they cannot manage their asthma; and 29% must pay for GP care. Expected attendance was increased for Maori and Pacific students versus others by 77% ($p=0.002$), and by asthma of increased severity ($p < 0.001$). Teenager resistance to accessing GP asthma care reduced expected attendance by 24% ($p=0.003$).

Conclusions. Maori and Pacific peoples have traditionally faced barriers to accessing GP care, but their more frequent attendance (than New Zealand Europeans) in this case, challenges whether such barriers persist, at least for acute care of moderate to severe asthma.

As children move into their early and middle teenage years, they begin to acquire the ability and permission to help make medical care-seeking decisions. Enhancing the ability of adults to support teenagers is the capacity and willingness of the teenager to recognise a particular health problem or need, and disclose it to an adult.

Problem non-recognition by teenagers may reflect a lack of both functional limitation and sensed danger.¹ Non-disclosure, when there is recognition of health need, may result from believing that continuing functional limitation is inevitable² or an expectation that the problem will not be managed as the teenager wants. Negative teenager perceptions of the organisation and delivery of general practitioner (GP) services^{3–6} may help to account, in turn, for teenagers' under-use of these services.⁷

Asthma is a common condition of rising prevalence,⁸ which affects teenagers' health status⁹ and can impair development into independent, functional adulthood.¹⁰ In New Zealand (NZ), GPs are responsible for the diagnosis and clinical management of most asthma. Attendance patterns for GP asthma care vary widely in NZ¹¹ but are poorly understood for teenagers.

This paper focuses on 13–14 year olds with moderate to severe asthma in the Auckland region. It aims to describe and understand from their perspective the frequency with which they access GP asthma care. Reference is made to teenagers'

perspectives on their need for this care; their communication with parents and other guardians; and GP care.

Methods

Sampling—A sample of 13–14 year olds with moderate to severe asthma was produced in three stages. Stage 1 involved the random selection (in mid-2002) of State secondary schools in Auckland City and Manukau City (south of Auckland City). Schools were ineligible for selection if they had been invited to participate in the concurrent, third phase of the International Study for Asthma and Allergies in Childhood (ISAAC).¹² Participation in our research was sought from 15 schools, first in writing to the school principal and school nurse, and then through a follow-up phone call to both. Site visits to interested schools established a working relationship as well as the roles and responsibilities of each party.

In Stage 2, each participating school used its records to identify all known 13–14 year-olds with breathing problems, and allowed us to speak with this group about breathing, asthma, and our research. The teenagers were invited to obtain from an adult guardian written informed consent to self-complete a short questionnaire at a return visit. They were given a supporting letter from the school principal; a coloured flyer about the project; an information sheet; and a consent form. Our materials were available in English, Maori, Mandarin, Samoan, and Tongan. The teenagers were told that all participants would enter a draw for petrol vouchers. After approximately 2 weeks, a follow-up phone call was made to guardians of the teenagers who had not returned consent forms. In late 2002, we administered the self-complete questionnaire to the teenagers for whom written consent had been received.

Stage 3 identified the teenagers with moderate to severe asthma from responses to the questionnaire (see below).

Sample size—Power calculations had been performed for different sample sizes. Without clustering by school, a sample of 107 students with moderate to severe asthma was sufficient to estimate with 95% confidence any item response reported with a prevalence of $75\% \pm 8\%$. For a regression model estimated by maximum likelihood, we planned to allow approximately one parameter for each 10 observations.

Data collection—The questionnaire had two parts. Part A focused on the presence and severity of breathing problems during the previous 12 months. For the teenagers reporting ‘wheezing or whistling in the chest’, *moderate to severe asthma* was defined by reports of at least one of the following: ≥ 4 wheezing attacks, asthma-associated sleep disturbance on ≥ 1 night per week, and wheezing severe enough to limit speech to one or two words at a time between breaths. These definitions of asthma and severity of asthma were used by ISAAC.¹²

Among other questions was one asking how many GP visits the teenagers had made for their wheezing in the past year. Part B covered issues not discussed at our oral presentation and was required only if the asthma was moderate to severe. The questions covered potential influences on the use of GP care for wheezing. They were developed from published literature and our prior qualitative interviews with families, including some teenagers, on child access to GP care for moderate to severe asthma.^{13–15} The questions were reduced to six variables through additive scales. Two of the variables describe perceived need. Three describe teenager communication and one describes beliefs about GP care. Teenagers were assigned to each of the one or more ethnic groups they specified.¹⁶

Data analysis—Simple descriptive and inferential statistics were produced to explore the dataset for teenagers with moderate to severe asthma. The outcome of primary interest was a count of GP visits for asthma in the year before completion of the questionnaire. A negative binomial regression model (NBRM) was fitted using the software package, Stata¹⁷ to account for this outcome. An intraclass correlation of 0.000 suggested that no variation in GP attendance was attributable to the clustering of students within the same schools.

Ethical approval for the study was obtained from the Auckland Ethics Committee.

Results

Ten of the 15 invited schools took part. Their mean decile was 5.1 ($s=3.1$). Their mean total roll was 1386 students ($s=935$) with, on average, 12.9% Maori and 22.3%

Pacific Islanders. Of the five other schools, all cited workload as their reason for not participating. Their mean decile was 3.8 ($s=3.8$) and they averaged 1312 students ($s=654$) of whom 15.1% and 42.5% (on average) were Maori and Pacific Island students respectively.

Participant schools identified 510 13–14 year-olds as having breathing problems or asthma, of whom 271 (53.1%) returned written consent from a guardian to complete our questionnaire. Of the 85.2% (231/271) students reporting ‘wheezing or whistling in the chest in the last 12 months,’ almost half (114/231) fulfilled the criteria for moderate to severe asthma.

The students’ mean age was 13.5 ($s=0.5$) with a sex ratio of 40.7 (33 boys and 81 girls). Almost three-quarters (84/114) identified as NZ European. Maori and Pacific teenagers numbered 15.8% (18/114) and 13.6% (15/114) respectively; 22.8% (26/114) identified as Maori and/or Pacific Islander. Fourteen teenagers (12.3%) identified as Asian.

Approximately four in every ten (39/100) of the teenagers had made 0 or 1 visit to a GP for asthma in the year before completing the questionnaire; 17% (17/100) had made ≥ 5 visits. Low attendees (0 or 1 visit) were disproportionately NZ European, and high attendees (≥ 5 visits) tended to be Maori and/or Pacific Islanders.

However, no statistically significant differences were detected between the mean numbers of visits reported by Maori and Pacific teenagers (4.5 visits) compared with other teenagers (2.5 visits) ($t = -1.673$, $p > |t| = 0.108$), or by the boys (3.9 visits) and girls (2.5 visits) respectively ($t = 1.4$, $p > |t| = 0.189$). A statistically significant relationship was found between the number of GP visits for asthma and the severity of moderate to severe asthma ($F = 19.6$, $p < F = 0.000$).

Table 1 reports the distribution of responses to statements about need, communication, and GP care. Among key findings was that only half of the teenagers received GP care as often as these teenagers wished. Almost one-third reported having to pay for their GP care. Table 2 summarises findings from the NBRM. Expected attendance was increased for Maori and Pacific students compared with other students, and by asthma of increased severity. No interactions were detected.

Table 1. Frequency of agreement with statements about need, communication and General Practitioner (GP) care among 13–14 year-olds with moderate to severe asthma

Variable	Yes (%)	No (%)	Neither Yes nor No (%)	n
Wheeze-related need				
Wheezing is a problem for me	49.1	15.7	35.2	108
My wheezing can be controlled	83.3	4.6	12.0	108
I need help with wheezing	19.4	55.6	25.0	108
I don’t want to bother parents with my wheeze	15.9	53.3	30.8	107
Need for GP care of wheeze				
I need GP care for wheeze	52.9	24.0	20.2	104
I have seen a GP about my wheeze as often as I wanted	49.5	28.0	22.4	107
Going to GP about wheeze is important to me	36.2	41.9	21.9	105
I might waste GP’s time with my wheeze	15.4	68.3	16.4	104

Resistance to GP care of wheeze				
I sometimes resist seeing doctor for wheeze	24.3	62.6	13.1	107
Direct request for GP care of wheeze				
I ask to see doctor about my wheeze	51.4	21.5	27.1	107
Information-telling about wheeze and its management				
I tell parents when I wheeze	84.1	11.2	4.7	107
I tell staff member when I wheeze at school	33.3	39.8	26.9	108
I tell parents how often I wheeze	52.3	32.7	15.0	107
I tell parents how often I use inhaler	50.5	37.4	12.2	107
I tell parents when inhaler gets low	78.5	14.0	7.5	107
I tell parents when inhaler runs out	86.9	5.6	7.5	107
I tell parents when I can't manage wheeze myself	61.7	21.5	16.8	107
GP care				
I find it difficult to talk to GP	8.5	82.1	9.4	106
I have difficulty understanding GP	10.4	76.4	13.2	106
GP shows me respect	87.7	3.8	8.5	106
I have to pay for my GP care	28.9	55.8	15.4	104
I trust practice staff to respect my privacy	86.5	3.9	9.6	104
I have to go to family GP	63.5	22.1	14.4	104
GP is available at times that suit me	58.7	20.2	21.2	104

Table 2. General Practitioner (GP) visits by 13–14 year olds with moderate to severe asthma: negative binomial regression model (N=92)

Dependent variable: GP visits	B	z	P > z 	%
Independent variables (x)				
Need related to wheeze	0.120	1.693	0.091	12.8
Need for GP care of wheezing	0.225	4.259	0.000	25.2
Resistance to GP care	-0.277	-2.927	0.003	-24.2
Direct request for GP care	-0.005	-0.050	0.960	-0.5
Information telling about wheeze and its management	-0.068	-1.969	0.049	-6.5
GP care	0.014	0.315	0.753	1.4
Asthma duration	-0.519	0.090	0.090	-40.5
Asthma severity	0.231	4.054	0.000	26.0
Asthma severity	0.568	3.080	0.002	76.5
Maori or Pacific person	-0.704	-3.762	0.000	-50.5
Girls				
a	0.17			
Pseudo-R²				16.3

- X = independent variable
 B = raw coefficient
 Z = z-score for test of $b = 0$
 $P > |z|$ = p value for z-test
 $\%$ = percent change in expected visit count for unit increase in x
 a = overdispersion parameter

Discussion

Auckland 13–14 year-old students with moderate to severe asthma described a variable frequency for accessing GP asthma care. Low attendees were disproportionately NZ European. High attendees tended to be Maori and/or Pacific Islanders (groups that have traditionally experienced barriers to accessing GP care^{12,18–20}). Our survey of the parents of 6–9 year olds with moderate to severe asthma replicated this finding.²¹

Teenage beliefs influencing this utilisation pattern were identified. About half the sample reported 'needing' GP asthma care. A similar proportion receives such care as often as it wants, independently of ethnicity. With only 36% agreeing that this care is 'important,' these results indicate an unmet desire for GP asthma care that teenagers consider appropriate rather than necessary.

Most teenagers reported telling their parents when they wheeze (or much less frequently, school staff) and when their inhaler runs out. Only three in every five said they tell their parents when they cannot manage their wheeze themselves, and only half ask to see a GP about their wheeze. This highlights a need to explain to teenagers the circumstances under which growing autonomy does not preclude asking for help.

Almost one in every four teenagers indicated sometimes resisting GP attendance for asthma care. That 29% reported having to pay for their GP care indicates that GP use has frequently depended on the financial resources of teenagers, which presumably are low, and not merely the motivation to attend, which our results suggest is generally also not high.

The NBRM estimated that perceived need for GP asthma care increases the expected number of visits for asthma by 25%. Teenagers' resistance to this care reduces the expected number of visits by a similar proportion. So too, to a lesser extent, does teenagers telling a guardian about their asthma and its management. However, low attendance could promote information giving-rather than *vice versa*, and attendance could be reduced less by information-giving *per se* than unwillingness by teenagers to advocate for attendance requiring dependence on parents. Also, GP care is frequently considered unimportant by teenagers, who may see it to interfere with their ability to minimise differences from peers.

Expected attendance was increased for the Maori and/or Pacific teenagers by 77% and by asthma of increased severity by 26%, given all the values in the model. This challenges the persistence of barriers to Maori and Pacific peoples accessing GP services, at least for acute asthma.^{12,18–20} None of the six predictors relating to need, communication, and GP care characterised disproportionately the Maori and Pacific teenagers. However, although we asked solely about total visits, our findings could be explained by barriers to these teenagers accessing only routine preventative care. Asthma exacerbations could then have prompted GP visits for acute care, explaining the higher total number of Maori and Pacific visits for asthma.

Other reasons are needed for why almost 40% of the teenagers, who were mainly NZ European, under-used GP care for their moderate to severe asthma. Compared with Maori and Pacific teenagers, they were perhaps more likely to have previously received and redeemed repeat prescriptions for asthma, and to receive acute care out-of-hours from relatively high-cost, community-based Accident and Medical Services.

It is also unclear why boys were more likely than girls to report obtaining GP asthma care. The finding is not due to differences in asthma severity.

Strengths and limitations—Complementing our other quantitative²¹ and qualitative research^{13–15,22} on factors influencing child access to GP care for moderate to severe asthma, this study responds to lack of knowledge regarding how frequently 13–14 year olds attend for GP care of moderate to severe asthma, and why. The high attendance by Maori and Pacific teenagers highlights a need to question whether

barriers to GP attendance persist for this group (at least for acute asthma), compared with NZ Europeans. The study raises the possibility that barriers to accessing preventative care can account for increased total numbers of visits among Maori and Pacific teenagers.

Nevertheless, the findings have limitations. One-third of the schools did not participate, but characteristics of these schools did not differ from those that did. A more major limitation is that the response rate by guardians of children with breathing problems was only 53%, which may limit the generalisability of the findings to all asthmatic teenagers. We do not know how non-participating teenagers differed from the teenage participants. A further limitation is that the final sample size for the teenagers was also small, reflecting difficulty in accessing this group.

Use of a school-wide screening questionnaire was unacceptable to the schools, thus requiring school nurse records to identify asthmatic teenagers. This is unlikely to have produced a large selection bias because nurses were most likely to know the teenagers with moderate to severe asthma. In our companion study,²¹ prevalences of moderate to severe asthma among 6–9 year-olds (based on parents' questionnaire responses) were independent of how schools identified children with breathing problems.

Over-representation of girls in the study most likely reflects our sample, which included two girls-only schools but no boys-only schools. Self-enumeration of GP visits for asthma in the previous year was subject to misclassification. It was not validated against GP claims for patient subsidies because this would have breached participants' anonymity. GP records of visits could not be checked because of the large number of GPs and the potential for each patient to attend more than one practice. However, self-reports have been shown not to impact systematically on estimates of ethnic differences in health care use,²³ and these differences show the same pattern as reported by the parents of similar 6–9 year-olds.²¹ Furthermore, non-systematic misclassification would tend to reduce our ability to find significant associations.

No distinction was made between routine visits and visits for acute care. Exposure time was not measured at the interval level, and use of school records might not have identified some eligible students. Reports of wheezing were not validated against a diagnosis from a doctor, although our focus on moderate to severe asthma most likely minimised this problem. The questions defining moderate to severe asthma came from ISAAC,¹² but we developed our own questions on factors influencing access to GP care in the absence of any standardised and validated tool. Qualitative interviews with teenagers might have yielded different insights and concerns.

Implications—There is a need to respond to an unmet preference by teenagers for improved access to GP care of moderate to severe asthma—despite a high level of self-reported attendance for GP asthma care by Maori and Pacific teenagers. Research is needed to test whether or not this utilisation pattern is specifically for acute care of poorly controlled asthma in the face of barriers to accessing routine, preventative care.

If barriers to accessing preventative care persist and increase both acute visits and hence total visits among Maori and Pacific teenagers, this challenges the assumption that access can be defined simply in terms of barriers that must be overcome to obtain healthcare.

A concurrent need exists to understand the widespread under-use of GP care for moderate to severe asthma, especially among NZ European teenagers. In the meantime, health policy should educate teenagers with moderate to severe asthma, and their guardians, on the importance of preventative care for asthma (as part of integrated strategies for addressing known barriers to teenage use of GP services). This would complement previous research highlighting a need to improve knowledge about asthma and its management among parents, teachers and teenagers.^{12,18–20}

Health policy must also respond to concerns that teenagers and guardians express about the acceptability of GP services, while promoting teenage-guardian communication about health issues associated with teenage asthma. An indicator of success will be whether teenagers are taken to a doctor when requested.

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Extended-spectrum beta-lactamase-producing *Enterobacteriaceae* at Middlemore Hospital

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Abstract

Aims. To review patients colonised or infected with extended-spectrum beta-lactamase-producing *Enterobacteriaceae* (ESBLPE) at Middlemore Hospital, Auckland, New Zealand.

Methods. All patients who had an ESBLPE isolated at the Middlemore Hospital Microbiology Laboratory from January 2001 to June 2004 were included in this review.

Results. ESBLPE were isolated from 132 patients during the review period. There were 12 patients colonised or infected with an ESBLPE in 2001, 34 in 2002, 43 in 2003, and 43 in the first 6 months of 2004. The isolates were *Escherichia coli* (n=56), *Enterobacter* spp. (n=55), and *Klebsiella pneumoniae* (n=21). ESBLPE were isolated from a wide range of specimens including peripheral blood in 18 patients. Thirty-three (25%) patients had an ESBLPE isolated within 48 hours of admission; seven of these patients were neither long-term care facility (LTCF) residents nor had hospital admissions in the previous 6 months. Thirty-one patients (23%) resided in a LTCF before their admission; four patients from the same LTCF had indistinguishable isolates. All isolates tested were susceptible to meropenem and imipenem. All but one isolate tested was susceptible to ertapenem, and all but two were susceptible to amikacin.

Conclusions. Colonisation and infection due to ESBLPE are increasing at Middlemore Hospital and in the Auckland community. We expect this trend to continue. There is evidence to suggest transmission of ESBLPE both in the Auckland community and LTCFs. Antibiotics useful for treatment of patients with proven ESBLPE infection at Middlemore Hospital include amikacin or a carbapenem. Careful infection control practices and antibiotic prescribing will be necessary to reduce the rate of increase of ESBLPE colonisation and infection at Middlemore Hospital and in the Auckland community.

Extended-spectrum beta-lactamase-producing *Enterobacteriaceae* (ESBLPE) were first described in Europe in 1983.¹ In the subsequent 20 years, bacteria with this resistance mechanism have become increasingly important. ESBLPE are not only resistant to penicillins and cephalosporins but are often also resistant to a wide range of other antibiotic classes (including fluoroquinolones, aminoglycosides, and trimethoprim/sulfamethoxazole) due to accumulation of other resistance genes. This limits effective treatment options.

ESBLPE were first identified in New Zealand in 1994² but were only isolated in low numbers nationwide (less than 30 per year) until 2001.³ Since then, there has been a significant increase in the number of isolates received by the Institute of Environmental Science and Research Limited (ESR), with 83 in 2001, 230 in 2002,

305 in 2003,³ and 182 in the first 6 months of 2004 (personal communication, H Heffernan, Senior Scientist, ESR, 2004).

This review was undertaken to assess the demographics and microbiology of patients colonised or infected with ESBLPE at Middlemore Hospital and the treatment and outcome of patients with ESBLPE bacteraemia.

Methods

All patients who had an ESBLPE isolated at the Middlemore Hospital Microbiology Laboratory from January 2001 to June 2004 were included in this review. Potential ESBLPE were identified by any of the following: reduced susceptibility to a third-generation cephalosporin; resistance to two or more classes of antibiotics excluding beta-lactams; synergy between ceftriaxone and amoxycillin/clavulanic acid on routine disc susceptibility testing, or resistance to cefaclor or cefuroxime while retaining susceptibility to amoxycillin/clavulanic acid.

Isolates were confirmed as ESBL-producers by either the National Committee on Clinical Laboratory Standards (NCCLS) method⁴ (modified to include cefpodoxime and ceftiofime discs with and without clavulanic acid)⁵ and/or the double disc synergy test.⁶ It is common practice for ESBL-producing isolates in New Zealand to be referred to ESR for surveillance purposes. All isolates included in this review were also confirmed as ESBL-producing by ESR.

Data were retrospectively collected on patient demographics, organism isolated, antibiotic susceptibility profiles, specimen type, day of admission when ESBLPE was first isolated, and potential risk factors for ESBLPE acquisition (a hospital admission in the previous 6 months, residence in a long-term care facility [LTCF], and admission to an intensive care unit [ICU]). Antibiotic susceptibilities were performed by either the disc diffusion method or by the Vitek 2 system (AST-NO19 card, bioMérieux), and interpreted using NCCLS breakpoints.⁴

For patients in whom an ESBLPE was isolated from peripheral blood culture, data were also collected on source of bacteraemia, day post admission when ESBLPE bacteraemia was first detected, antibiotics received, and treatment outcome ('cure' defined as no clinical deterioration following completion of antibiotic treatment; 'relapse' defined as clinical deterioration following completion of treatment and/or isolation of the same organism from blood following treatment; or 'death').

In addition to the antibiotic susceptibility testing methods described above, the minimum inhibitory concentrations (MIC) of ciprofloxacin, meropenem, ertapenem, imipenem, and amikacin were determined (E-test, AB Biodisk, Solna, Sweden).

Results

During the 3½-year period of this review, ESBLPE were isolated from 132 patients admitted to Middlemore Hospital. The median age was 67 (range 1 to 93) years, and 72 (55%) were female. The ethnicities of these patients were recorded as European (n=85), Maori (n=14), Samoan (n=5), Chinese (n=3), Fijian (n=3), Fijian Indian (n=3), Indian (n=3), Rarotongan (n=3), Tongan (n=3), and Other (n=10).

There were 12 patients colonised or infected with an ESBLPE in 2001, 34 in 2002, 43 in 2003, and 43 in the first 6 months of 2004. The Service primarily responsible for patient care before the isolation of an ESBLPE was Medicine (n=27), Surgery (n=25), Orthopaedics (n=23), Plastics/Burns (n=18), Geriatrics (n=11), Intensive Care Unit [ICU] (n=9), Paediatrics (n=3), and Other (n=13).

Of the 132 patients, 129 had an ESBLPE first isolated as an inpatient and 3 as an outpatient. The median number of days after admission when an ESBLPE was first isolated was 11 (range 0 to 94) days. Thirty-three (25%) patients had an ESBLPE isolated in the first 48 hours of admission to hospital. Of these 33 patients, 21 had one or more hospital admissions in the previous 6 months; 7 of these 21 patients were also LTCF residents. Of the remaining 12 patients, 5 were LTCF residents and 7 were

neither LTCF residents nor had hospital admissions in the previous 6 months. Thirty-six of the 132 patients (27%) had an ICU admission before the first isolation of an ESBLPE.

Overall, during the review period, 31 patients (23%) resided in a LTCF before the admission when an ESBLPE was isolated. Eleven LTCFs had one patient, six LTCFs had two patients, one LTCF had three patients, and one LTCF had five patients colonised or infected with an ESBLPE.

The isolates from the five patients residing in the same LTCF were all *Escherichia coli*. These five patients were admitted to hospital between June 2003 and March 2004. DNA analysis using pulsed-field gel electrophoresis (PFGE) after restriction digestion with *Xba I* was performed on these isolates by ESR. Four of the five isolates had an indistinguishable pattern and the remaining isolate had a very closely related pattern with one band difference only. An ESBLPE was first isolated from two of these patients on the day of admission and from the others on days 7, 8, and 16 after admission. Four of these five patients had a hospital admission in the previous 6 months.

The species producing an ESBL included *E. coli* (n=56), *Enterobacter cloacae* (n=48), *Klebsiella pneumoniae* (n=21) and *Enterobacter* spp. (n=7). During the review period, the Middlemore Hospital Microbiology Laboratory isolated *E. coli* from approximately 7370 patients, *Enterobacter* spp. from approximately 790 patients, and *K. pneumoniae* from approximately 810 patients.

Thus ESBL-production was detected in 0.8% of *E. coli*, 7% of *Enterobacter* spp., and 3% of *K. pneumoniae* at Middlemore Hospital. Many patients had an ESBLPE isolated from more than one specimen type.

The most clinically significant isolate came from peripheral blood culture (n=18), catheter blood culture (n=2), central venous catheter tip (n=2), tissue/abscess (n=6), drainage fluid (n=2), epidural catheter tip (n=1), joint aspirate (n=1), pleural aspirate (n=1), sputum/tracheal aspirate (n=5), wound swab (n=25), midstream urine/catheter urine (n=63), and faeces (n=6).

The antibiotic susceptibilities for all isolates are shown in Table 1. Seventy-eight of 80 (98%) isolates tested were susceptible to amikacin. All 115 isolates that were tested were susceptible to imipenem. Sixty-seven of 68 (99%) isolates tested were susceptible to ertapenem.

Table 1. Antibiotic susceptibilities for all isolates

Antibiotic	Susceptible	Intermediate susceptibility	Resistant
Imipenem	115 (100%)	—	—
Ertapenem	67 (99%)	—	1 (1%)
Piperacillin/tazobactam	66 (92%)	3 (4%)	3 (4%)
Gentamicin	18 (14%)	1 (1%)	108 (85%)
Amikacin	78 (98%)	—	2 (2%)
Ciprofloxacin or norfloxacin	55 (44%)	11 (9%)	60 (47%)
Trimethoprim/sulfamethoxazole	3 (5%)	—	57 (95%)

Table 2. Gender; age; organism isolated; source of bacteraemia; day post admission of first positive blood culture; ciprofloxacin susceptibility by breakpoint; ciprofloxacin, carbapenem, and amikacin MICs; antibiotics received from day of first positive blood culture; and outcome for the 16 patients treated for ESBLPE bacteraemia

Patient	Gender, Age (years)	Organism	Source of bacteraemia	Day post admission of first positive blood culture	Ciprofloxacin susceptibility	Ciprofloxacin MIC (mg/L)	Meropenem MIC (mg/L)	Ertapenem MIC (mg/L)	Imipenem MIC (mg/L)	Amikacin MIC (mg/L)	Antibiotics received from day of first positive blood culture	Outcome
1	M, 91	<i>E. coli</i>	Biliary tract	0	R	>32	0.032	0.064	0.25	8	CTO d0 to 1, IMP d2 to 5, treatment withdrawn d5	Died d8
2	F, 89	<i>E. coli</i>	Biliary tract	0	R	>32	0.064	0.064	0.25	8	CTO d0 to 2, GEN d0 to 1, treatment withdrawn d2	Died d3
3	M, 86	<i>E. cloacae</i>	Urinary tract	0	S	1	0.125	0.125	0.5	1	CFX d0 to 2, GEN d0, treatment withdrawn d2	Died d9
4	F, 85	<i>K. pneumoniae</i>	Urinary tract	0	S	0.032	0.064	0.016	0.125	2	CFX d0 to 2, ROX d0 to 1, GEN d1 to 2, NOR d2 to 3, CIP d3 to 10	Cured
5	M, 64	<i>E. coli</i>	Uncertain	2	R	>32	0.064	0.125	0.25	8	MOX d0 to 3	Died d3
6	M, 30	<i>E. cloacae</i>	Intravascular catheter	5	I	1	0.125	0.5	1	8	IMP d6 to 16	Cured
7	F, 68	<i>E. cloacae</i>	VAP	8	S	1	0.125	0.5	1	4	CIP d8 to 21	Cured
8	F, 35	<i>K. pneumoniae</i>	Intravascular catheter	8	S	0.5	0.064	0.125	0.25	1	GEN d8, FLX d8 to 9, CTO d9, IMP d9 to 26	Cured
9	M, 57	<i>E. cloacae</i>	Intravascular catheter	14	S	0.125	0.125	0.25	0.5	2	GEN d14 to 16, MER d16 to 32, CIP d16 to 18	Cured
10	F, 17	<i>E. cloacae</i>	Intravascular catheter	17	S	0.25	0.064	0.016	0.25	2	CIP d17 to 26, IMP d17	Cured
11	M, 62	<i>E. coli</i>	Biliary tract	22	R	>32	0.064	0.032	0.25	4	CIP d22 to 23, PIP/TAZ d23 to 34	Cured

12	M, 28	<i>E. cloacae</i>	Intravascular catheter	26	I	1	0.032	0.064	0.25	2	CIP d27, GEN d27 to 28, CTZ d28 to 42, AMK d29 to 42 (concurrent <i>P. aeruginosa</i> bacteraemia)	Cured
13	F, 57	<i>E. cloacae</i>	Nosocomial pneumonia	39	R	8	0.25	0.5	0.25	8	IMP d39 to 40	Died d40
14	M, 81	<i>E. coli</i>	Urinary tract	40	R	>32	0.032	0.016	0.25	8	CFX d 40, CIP d 41 to 42, MER d42 to 44, NIT d44 to 51	Cured
15	M, 69	<i>K. pneumoniae</i>	Abdominal collection	43	S	0.5	0.064	0.064	0.25	1	AMO d43 to 44, IMP d44 to 54, GEN d44	Cured
16	M, 60	<i>E. coli</i>	Pancreatic pseudocyst	50	R	>32	0.064	0.064	0.5	4	AMO/CLAV d50, IMP d50 to 51	Died d51

Note: MIC: minimum inhibitory concentration, ESBLPE: extended-spectrum beta-lactamase producing *Enterobacteriaceae*, M: male, F: female, R: resistant, S: susceptible, I: intermediate susceptibility, VAP: ventilator associated pneumonia, d: day, AMK: amikacin, AMO: amoxycillin, AMO/CLAV: amoxycillin/clavulanic acid, CFX: cefuroxime, CIP: ciprofloxacin, CTO: ceftriaxone, CTZ: ceftazidime, ERT: ertapenem, FLX: flucloxacillin, GEN: gentamicin, IMP: imipenem, MER: meropenem, MOX: moxifloxacin, NIT: nitrofurantoin, NOR: norfloxacin, PIP/TAZ: piperacillin/tazobactam, ROX: roxithromycin.

The ertapenem resistant isolate was an *E. cloacae* that was first isolated from the urine of a 75-year-old European man with a history of Duke's C adenocarcinoma during an admission to another hospital for ureteric stenting. One month later, it was again isolated from his urine when he presented to Middlemore Hospital with a bowel obstruction. This isolate had an ertapenem MIC of 8 mg/L (susceptible ≤ 2 mg/L, intermediate 4 mg/L, resistant ≥ 8 mg/L),⁴ meropenem MIC of 0.5 mg/L (susceptible ≤ 4 mg/L)⁴ and imipenem MIC of 4 mg/L (susceptible ≤ 4 mg/L).⁴

Eighteen patients had an ESBLPE isolated from a peripheral blood culture during their admission. Two of these patients remained well, despite receiving no effective antibiotic against the isolate so were excluded from further analysis. Of the remaining 16 patients, 6 (38%) were female and their median age was 63 (range 17 to 91) years. The organisms isolated were *E. cloacae* (n=7), *E. coli* (n=6), and *K. pneumoniae* (n=3). There were approximately 750 episodes of Gram-negative bacteraemia during the review period, thus ESBLPE were responsible for 2% of all Gram-negative bacteraemia.

Characteristics of the bacteraemic patients and their isolates are shown in Table 2. Four patients (25%) presented to hospital bacteraemic with an ESBLPE. Of the 12 patients who developed ESBLPE bacteraemia in hospital, 8 (67%) had previously been admitted to ICU.

All bacteraemic isolates were susceptible to all carbapenems. The MIC₅₀ and MIC₉₀ were 0.064 and 0.125 mg/L respectively for meropenem; 0.064 and 0.5 mg/L for ertapenem; and 0.25 and 1 mg/L for imipenem. All isolates were susceptible to amikacin with a MIC₅₀ and MIC₉₀ of 4 and 8 mg/L respectively (susceptible ≤ 16 mg/L).⁴ Fourteen (88%) of the isolates were susceptible to piperacillin/tazobactam by disc diffusion (MICs were not determined).

Twelve patients received more than 48 hours of antibiotic as treatment for ESBLPE bacteraemia; the majority of antibiotic treatment these patients received was with a carbapenem (n=6), a quinolone (n=4), piperacillin/tazobactam (n=1), or amikacin (n=1). Six patients (38%) died; there were no relapses.

Discussion

During the 3½ years of this review, the number of ESBLPE isolated at Middlemore Hospital have increased significantly. In the first 6 months of 2004, there was the same number of ESBLPE isolated as for all of 2003. During the review period, ESBL production at Middlemore Hospital was detected in 7% of *Enterobacter* spp., 3% of *K. pneumoniae*, and 0.8% of *E. coli*.

ESBLPE were isolated from all departments at Middlemore Hospital; particular departments did not appear to be over-represented in the number of patients from whom an ESBLPE was isolated, however one-quarter of patients had been admitted to ICU before the isolation of an ESBLPE. While almost half of the ESBLPE at Middlemore Hospital were isolated from urine, ESBLPE were also responsible for invasive disease (including 16 patients with bacteraemia).

Originally *K. pneumoniae* and *E. coli* were the most common ESBL-producing bacteria worldwide, although in recent years ESBL production amongst *Proteus mirabilis* and AmpC-producing *Enterobacteriaceae* has become more prevalent.⁷

As the genes encoding ESBLs are contained on plasmids, horizontal gene transfer to many species of bacteria is possible. ESBL production has rarely been transferred to non-*Enterobacteriaceae*; ESBL-producing *Pseudomonas aeruginosa* and *Acinetobacter* spp. have been reported in Europe.^{8,9}

Nationwide, during the review period, *E. coli* (responsible for 73% of all isolates), *Enterobacter* spp. (responsible for 15% of all isolates), and *K. pneumoniae* (responsible for 8% of all isolates) were the most common ESBLPE referred to ESR (personal communication, H Heffernan, 2004).

The numbers of *E. coli* are bolstered by a clonal outbreak in a North Island Hospital. At Middlemore Hospital, ESBL-producing *Enterobacter* spp. are as common as ESBL-producing *E. coli* (both responsible for 42% of all isolates) with ESBL-producing *K. pneumoniae* responsible for only a small number of isolates (16%).

ESBLPE were responsible for 2% of all Gram-negative bacteraemia that occurred at Middlemore Hospital during the review period. While the majority of patients with ESBLPE bacteraemia developed this during their hospital stay, a quarter of these patients presented to hospital with bacteraemia. Two-thirds of the patients who developed bacteraemia during their hospital stay had previously been admitted to ICU. The majority of bacteraemic patients were treated with either a carbapenem or a quinolone.

Carbapenems are recommended as the treatment of choice for ESBLPE bacteraemia.^{7,10} The meropenem, ertapenem, and imipenem MICs for all isolates causing bacteraemia were well within the susceptible range. The MIC₅₀ and MIC₉₀ were lowest for meropenem, followed closely by ertapenem. The choice of carbapenem may be influenced by cost and dosing interval.

Ertapenem, being the narrowest spectrum carbapenem, may be an attractive option in terms of reducing pressure on the development of resistance. However, while all the bacteraemic isolates were ertapenem-susceptible, an *E. cloacae* isolated from urine was found to be ertapenem-resistant. Rare ertapenem-resistance in *Enterobacteriaceae* has been described previously.¹¹ Therefore, susceptibility to ertapenem should not be assumed on the basis of susceptibility to meropenem or imipenem.

The use of ciprofloxacin (to treat ESBLPE bacteraemia) has been associated with increased rates of treatment failure and mortality when compared to treatment with a carbapenem.^{10,12} This is thought, at least in part, to be related to ESBL-producing isolates that have ciprofloxacin MICs close to the susceptibility breakpoint (susceptible $\leq 1\text{mg/L}$)⁴ and the inability to achieve adequate tissue levels of this drug above these MICs.

Of the seven bacteraemic isolates that were ciprofloxacin-susceptible, five (71%) had MICs close to the susceptibility breakpoint (MICs of 0.25 to 1 mg/L). Two patients (patients 7 and 10), who were almost exclusively treated with ciprofloxacin, had ciprofloxacin-susceptible isolates with MICs close to the susceptibility breakpoint (MICs of 1 and 0.25 mg/L respectively). The source of bacteraemia in these patients was ventilator-associated pneumonia and intravascular-catheter sepsis, respectively. Both patients were cured. Given the above concerns, we are not currently using ciprofloxacin as treatment for patients with ESBLPE bacteraemia or invasive disease.

All of the blood culture isolates, and 98% of the total isolates, were susceptible to amikacin. Treatment with this aminoglycoside is currently a treatment option at Middlemore Hospital, especially for patients with a urinary tract source of infection. The other possible treatment option is piperacillin/tazobactam, although there are concerns that treatment failure may occur (as the piperacillin/tazobactam MIC can increase with a large inoculum of infecting organisms).¹³

ESBLPE bacteraemia at Middlemore Hospital has a significant mortality rate (38%). This compares to mortality rates from other series of 19 to 50%.^{12,14,15}

One of the major risks (for colonisation and infection with an ESBLPE) is prolonged hospitalisation,¹⁶ however acquisition in LTCFs,^{17,18} and more recently community acquisition of these organisms, has been reported.^{19,20} The PFGE results for the five patients with an ESBL-producing *E. coli* (residing in one LTCF during a 10-month period) suggest that acquisition of ESBLPE in LTCFs may well be occurring in Auckland, although four of these five patients had been hospitalised in the previous 6 months and may have become colonised at that time.

There is transmission occurring in the Auckland community, as 7 of the 33 patients who had an ESBLPE isolated in the first 48 hours of admission to hospital had no hospital admission in the previous 6 months and were not LTCF residents. Given these findings, all patients are at risk of colonisation and infection with an ESBLPE.

Previous antibiotic use is a well-recognised risk factor for colonisation and infection with an ESBLPE. Hospital- and community-based studies have found an association with previous use of penicillin, a second- or third-generation cephalosporin, or a fluoroquinolone and ESBLPE infection.^{14,19–21}

The use of third-generation cephalosporins and ciprofloxacin is not restricted at Middlemore Hospital, and inappropriate use of these antibiotics may be contributing to the recent increase in ESBLPE isolations. We are currently considering ways in which to limit the use of these antibiotics at Middlemore Hospital.

The transfer of ESBLPE to non-colonised patients in hospitals and LTCFs occurs mainly via the hands of healthcare workers.⁷ Therefore, efforts to prevent patient-to-patient transmission (via the hands of healthcare workers) are necessary to reduce the transmission of ESBLPE in these facilities.

Since many ESBLPE-colonised patients are likely to go undetected, attention to standard precautions (particularly hand hygiene) is essential for all healthcare workers. Standard precautions plus contact precautions are used at Middlemore Hospital for patients known to be colonised or infected with an ESBLPE. An electronic 'multidrug-resistant organism alert' is placed in the clinical record of colonised patients so that staff will be aware of this at the time of any future admission.

Specific screening for ESBL colonisation is performed at Middlemore Hospital when previously colonised patients are readmitted, when transmission within a multi-bedded room or ward is suspected, and in the ICU where ongoing surveillance occurs. Colonisation is detected using faeces or a rectal swab cultured onto media that selects for Gram-negative bacilli with reduced susceptibility to aztreonam or ceftazidime.

Colonisation and infection due to ESBLPE are increasing at Middlemore Hospital and in the Auckland community. Currently ESBLPE are responsible for only a small

percentage of invasive disease or bacteraemia caused by Gram-negative bacilli at Middlemore Hospital. However we anticipate that ESBLPE will become more common in the near future; this may have implications for empirical antibiotic treatment.

Useful treatment options in our hospital for proven ESBLPE infection include amikacin or a carbapenem. Strategies that may reduce the rate of increase of ESBLPE-colonisation and infection in Auckland will include careful infection-control practices in hospitals and LTCFs, avoidance by all prescribers of unnecessarily broad spectrum antibiotics, and avoidance of the use of antibiotics in situations where they are not required.

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Vaginal water-jet injuries in premenarcheal girls

Tevita Aho, Vipul Upadhyay

Vaginal water-jet injuries may occur during activities such as waterskiing,^{1,2} jetskiing,^{3,4} and hydroskiing.^{5,6} Most reported cases involve adults, with only three previously described in premenarcheal girls.⁷⁻⁹ This paper describes two further paediatric cases, and discusses the management and prevention of such injuries.

Case reports

Case 1

An 8-year-old girl noticed lower abdominal discomfort, and heavy vaginal bleeding within minutes of playing on a high-pressure water fountain. She had been wearing a one-piece bathing suit. On examination in Accident and Emergency (A+E) at Starship Children's Hospital (Auckland, New Zealand), she was haemodynamically stable with a soft, non-tender abdomen. There was a large volume of blood clot at the introitus, but no other external evidence of injury.

Emergency examination under anaesthetic (EUA) revealed a 4cm-long flap laceration in the middle-third of the vagina. An actively bleeding artery was diathermied and vaginal packing inserted. The packing was removed under anaesthetic the following day and the patient was discharged 2 days after admission with a serum haemoglobin (Hb) level of 8.8 g/dL (12.1 g/dL on admission).

Case 2

A 10-year-old girl described immediate lower abdominal and pelvic pain after sliding feet-first with legs abducted down a hydroslide. Heavy vaginal bleeding followed soon after. She had been wearing a one-piece bathing suit. On examination in A+E continuing vaginal bleeding was noted but she was haemodynamically stable. There was no abdominal tenderness nor any obvious external injury. At EUA, a 4cm laceration of the proximal vagina was identified. There was generalised bleeding from the laceration but no specific bleeding vessel was identified.

Vaginal packing was inserted and removed uneventfully 30 hours later under anaesthetic. The patient was discharged after 3 days. Serum Hb had fallen from 10.8 g/dL on admission, to 8.8 g/dL at discharge.

Discussion

The cases described are uncommon but potentially serious, and resulted from watersport activities that are common in New Zealand. Vaginal injuries may result from the forceful entry of water into the vagina. Most water-jet injuries have been reported in adults who had a vaginal tampon in place at the time. Indeed, it has been hypothesised that water under pressure may induce a potentially injurious piston-like effect on tampons. However, as these two cases, and the three previously reported in

premenarcheal girls demonstrate, vaginal tampons are not a prerequisite for water-jet injury in this age group. In all cases, heavy vaginal bleeding resulted in early medical consultation. All patients were referred appropriately for specialist paediatric assessment and management, and significant vaginal lacerations were identified and treated.

Vaginal bleeding in premenarcheal girls soon after aquatic activities may be the only sign of significant vaginal injury. Diagnosis and treatment is usually straightforward but requires a general anaesthetic. When vaginal water jet injury is suspected, prompt referral to a paediatric centre for EUA is mandatory. It is important for medical personnel to be aware that major vaginal injury may result from seemingly innocent watersport activities.

Warning signs at aquatic centres and the use of wetsuits when participating in high-speed watersports could be considered as preventative measures.

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Inflammatory breast cancer in a male

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Abstract

Male breast cancer is very rare, especially inflammatory breast cancer, which is an aggressive, rapidly proliferating manifestation of primary breast carcinoma. We present a case report of a 56-year-old man in Lebanon who died 8 months after being diagnosed with inflammatory breast cancer.

Male breast cancer affects 1500 men each year worldwide.¹ Inflammatory breast cancer (IBC) is an aggressive, rapidly proliferating manifestation of primary breast carcinoma.² Male IBC is very rare and our review of the English literature yielded only six reports of eight cases.^{1,3-7} Here, we present the case of a man diagnosed with IBC after he presented with cervical lymphadenopathy, swelling, and redness of the right anterior chest. A biopsy of the breast showed infiltration of dermal lymphatics by tumour cells. His disease was aggressive and he died 8 months after diagnosis.

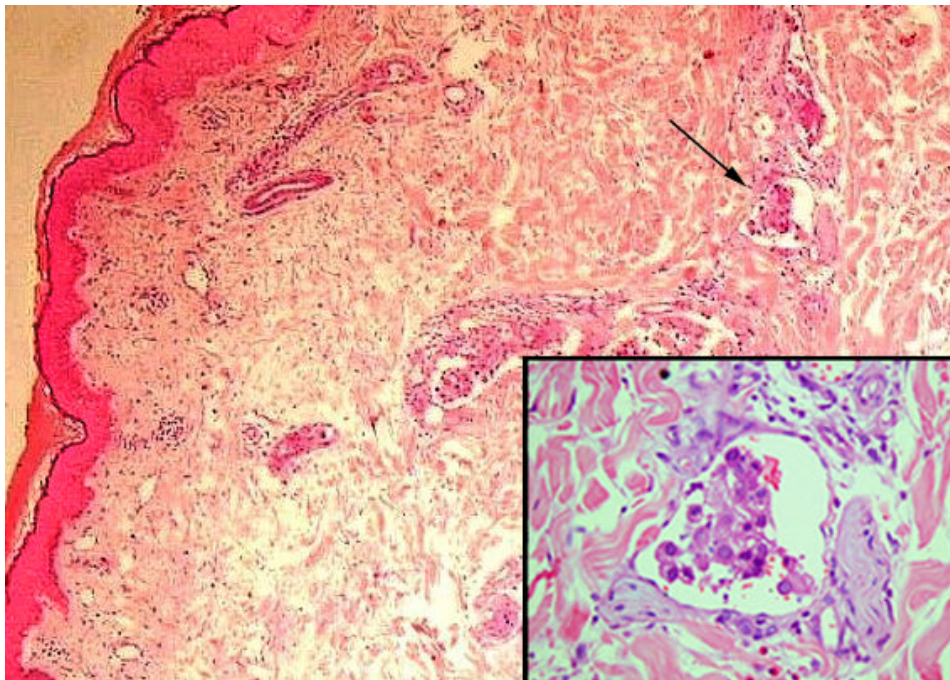
Case report

A 56-year-old man presented to our hospital (American University of Beirut Medical Center, Beirut, Lebanon) with neck swelling, oedema, and warmth of the right anterior chest wall, and gynaecomastia of 2 months duration. He was a 75 pack-years tobacco smoker. He reported that an enlarged right cervical mass appeared 2 months prior to his presentation. This mass was soon complicated by generalised, painless neck swelling that rapidly spread involving the right arm and chest wall with erythema and warmth. The right breast became painful and swollen. He then developed a feeling of suffocation, myalgias, arthralgias, and dyspnoea on exertion. He had no nipple discharge or bone pain.

On physical examination, the patient had a sensitive and painful thickened skin with redness and warmth over the right chest wall, upper abdomen, and upper arm. Both breasts were tender (especially on the right side) but no masses could be felt. Oedema involved the right breast and right anterior chest, and it extended to the right axilla. His right nipple was retracted. He had generalised neck lymphadenopathy with a decrease in the range of motion of the neck. He had no palpable axillary or inguinal lymph nodes.

His complete blood count, liver function tests, and urinalysis were normal. Erythroid sedimentation rate was 97 mm/hour (reference range 0–15 mm/hour). The mammogram showed severe skin-thickening and was suspicious of a malignancy. Biopsy of the breast skin revealed plugs of poorly differentiated adenocarcinoma within dermal lymphovascular channels. The dermal stroma and overlying epidermis were free of tumour cell (Figure 1). At that point, the diagnosis of an inflammatory breast carcinoma was made.

Figure 1. Breast skin with tumour thrombi within lymphovascular channels in the dermis (arrow); haemotoxylin and oesinophil stain; original magnification (x40). Inset shows a higher magnification (x100) of the arrowed position



Immunohistochemical stains showed tumour cells to be negative for oestrogen and progesterone receptors. They showed no HER2/neu overexpression. Almost all tumour-cell nuclei were positive for p53. Work-up was positive for metastatic disease to the lungs and the left frontal lobe of the brain.

Systemic chemotherapy using 5-Fluorouracil, Adriamycin, and Cyclophosphamide was administered. However, he showed no response to chemotherapy and refused further treatment. He was lost to follow-up for 3 months after which time he presented with generalised weakness, weight loss, and respiratory failure—and he died 8 months after diagnosis.

Discussion

The definition of IBC is somewhat controversial. Clinically, the triad of erythema, peau d'orange, and rapid onset makes the diagnosis of IBC.⁸ Pathologically, IBC is diagnosed as involvement of the dermal lymphatics by an infiltrating carcinoma,⁹ causing oedema and vascular congestion in the upper dermis and giving the clinical appearance of erysipelas.³

Our patient did not present with a breast mass but, rather, with skin changes that were clinically consistent with IBC. Pathologically, the carcinoma infiltrated the dermal lymphatics. Thus, when a male presents with inflammatory changes of the chest wall, the possibility of inflammatory breast cancer should be entertained even in the absence of a breast mass.

IBC has been reported to account for 1%–6% of all breast cancers (including males and females); IBC accounts for only about 1% of breast cancers in males however. Therefore, male IBC is extremely rare.⁴ Our review of the English literature yielded six reports of only eight cases of male IBC from 1953 to 2001.^{1,3–7}

Inflammatory carcinoma is the most aggressive variant of breast cancer with a very poor outcome,¹⁰ and the survival period from the onset of clinical symptoms ranges from 6 to 33 months.⁴ In our case, the period from the appearance of clinical symptoms to death was 8 months.

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The riddle of the sands: circumcision, history, and myth

Robert Darby

Abstract

Although many nineteenth century misconceptions about the foreskin have been dispelled since Douglas Gairdner showed that infantile phimosis was not a congenital defect, other old ideas have proved more persistent. Among the most ubiquitous are the proposition that ritual or religious circumcision arose as a hygiene or sanitary measure; and the related idea that allied troops serving in the Middle East during the Second World War were subject to such severe epidemics of balanitis that mass circumcision was necessary. Both these claims are medical urban myths which should be firmly laid to rest.

In a recent article on the ethics of circumcising male minors, JM Hutson stated that circumcision was 'likely to have arisen as an early public health measure for preventing recurrent balanitis, caused by sand accumulating under the foreskin.'¹ A similar statement appears in the policy statement on circumcision issued by the Royal Australasian College of Physicians in 2002: 'Circumcision of males has been undertaken for religious and cultural reasons for many thousands of years. It probably originated as a hygienic measure in communities living in hot, dusty and dry environments.'² No reference was given for either of these claims, and both are questionable.

The idea that circumcision protects the penis, and more especially the glans, from irritation by sand is counter-intuitive. One's natural assumption is that the foreskin guards the glans and meatus from irritation by shielding them from dust and other forms of dirt. This function seems more likely in boys before puberty, when the foreskin is usually longer and less frequently retracted—a point consistent with the fact that most circumcising tribes perform the operation at puberty or later.³ Yet the claim that circumcision protects against sand irritation appears regularly in medical journals, both as an explanation for the ancient origin of ritual circumcision in tribal societies, and as a medical justification for its performance in the twentieth century. What is the evidence for this?

Many primitive cultures carried out various mutilating procedures on different parts of the body, including the genitals of both boys and girls, but the origins and rationale of these practices are obscure and contested, as are the environmental conditions prevailing when such customs emerged. Such societies also practised human sacrifice, widow-burial, foot-binding, scarification, tattooing, piercing, infibulation, head or nose shaping, tooth evulsion, etc.

The idea that these rituals must have a utilitarian basis emerged in the eighteenth century, when Enlightenment thinkers sought naturalistic explanations for phenomena formerly regarded as miracles or attributed to the will of the Deity. Denis Diderot embodied this trend when he suggested that infibulation of women in some tribal societies originated as a birth control measure and only later acquired supernatural

sanction.⁴ Modern anthropology recognises that such customs emerge from the belief structure or cosmology of the cultures which produced them and do not necessarily have utilitarian significance.⁵

Conflicting theories have been advanced to account for ritual operations on the male and female genitals, among which are the following:

- A propitiatory sacrifice or sign of submission to a deity, probably a milder form of a ritual which began as outright human sacrifice.
- An offering to the god or goddess of fertility to ensure children.
- A mark of tribal identification.
- A rite of passage from childhood to adult responsibility.
- The imposition of adult and tribal authority at a time when youthful rebellion might be expected (in the case of boys circumcised at puberty).
- A fertility rite, aimed at giving men the power of procreation by making them shed blood from their genitals like women.
- An attempt to emphasise feminine or masculine characteristics in girls and boys by removing the parts of the genitals (clitoris and foreskin) believed to resemble the genitals of the other sex.
- A means of humiliating and marking defeated enemies and slaves.⁶

The only point of agreement among proponents of the numerous theories is that a practical objective such as health had nothing to do with it. This is not surprising: before aseptic surgery, any cutting of flesh carried a high risk of bleeding, infection and death. Travelling in Iraq in the 1930s, the English doctor Wilfred Thesiger reported that Arab boys undergoing circumcision sometimes took months to recover; in the case of one who sought treatment, 'His entire penis, his scrotum and the inside of his thighs were a suppurating mess from which the skin was sloughing away, the pus trickling down his legs.'⁷ Even today, in the age of antibiotics, scores of South African teenagers die in consequence of their bush circumcision ordeal.⁸

None of the ancient cultures which practised circumcision have traditionally claimed that the ritual was introduced as a sanitary measure. African tribes, Arabs, Jews, Moslems, and Australian Aboriginals explain it different ways, but divine command, tribal identification, social role, family obligation, respect for ancestors, and promotion of self control figure prominently. Jewish authorities make no mention of hygiene, let alone sand, but place stress on the religious significance of circumcision: it is an outward sign of the Covenant between God and his people.^{9,10} The Kaguru of central Tanzania explain circumcision (practised at puberty on both boys and girls) in terms of enhancing gender differentiation and social control. They consider the uncircumcised penis unclean because its moistness makes men resemble women, whose wet and regularly bleeding genitals are considered polluting.

Initiation is also 'a cultural cosmetic' which enables the older men to impress the young with 'the need for conformity to traditional values and beliefs, and...the superior knowledge and authority of elder males.'¹¹

It was only in the late nineteenth century, when mass circumcision was being introduced for 'health' reasons, such as control of masturbation, that doctors sought legitimacy for the new procedure by attempting to explain its origin in terms of hygiene. One of the first English surgeons to make the connection was James Copland, who introduced the idea that 'the neglect of circumcision in Christian countries' was a common cause of masturbation.¹² This theme was taken up by the sanitarians in the public health movement, such as WH Corfield, who praised circumcision as:

one of the most salutary regulations that was ever imposed on a people, especially in an eastern country, where the ... necessity of scrupulous personal cleanliness is so much increased. ... What wisdom was shown by Moses, and by Mahomet in later times, in retaining this wholesome custom as a religious rite, and thereby securing its perpetuation.

It was to the observance of such practices that many nineteenth century writers on hygiene attributed 'the singular immunity of the Jewish race in the midst of fearfully fatal epidemics.'¹³ This 'immunity' was a major theme of epidemiological debate in the late nineteenth century, leading to a search for further health benefits.¹⁴

As enthusiasm grew, other medical men put forward more fanciful suggestions. Dr Dampier-Bennett believed that circumcision originated as a treatment for epilepsy: 'in all primitive peoples there is a peculiar tendency to epilepsy', he thought, which might be caused by cerebral pressure or 'local irritation' such as that generated by a tight foreskin. He had treated 'epileptiform convulsions' in a 4-year-old boy by excising his 'remarkably long and adherent' prepuce, and he considered it 'likely that, amongst wild tribes...it has been discovered that a pacifying result follows...the operation.'¹⁵

James Allen argued that circumcision came into existence as a preventive of parasitic infections such as schistosomiasis,¹⁶ while (Sir) John Bland-Sutton believed that since 'a long foreskin is a recognised hindrance to convenient coitus' the main purpose of circumcision was to ensure fertility.¹⁷

Many of the tribal cultures which practised male circumcision also enforced various forms of female genital mutilation. Western doctors today are horrified by this sort of surgery and do not seek evidence that it might be beneficial to women's health or that it originated as a means of preventing sand from getting under the clitoral hood or labia. It was a different story in the mid-nineteenth century, when many doctors assumed with WF Daniell that female circumcision as practised by savage cultures was important for medical hygiene and that further research would reveal 'the use and purport of this singular custom.'¹⁸

In the 1850s and 1860s, many English doctors believed that clitoridectomy was as valuable as male circumcision in treating nervous diseases like epilepsy, hysteria, and masturbation (as well as their sequelae in madness), and pushed the therapy on women with little attempt to gain consent.¹⁹ And many Egyptian and other Islamic physicians today insist on the hygienic value of female circumcision as a preventive of both organic disease and sexual promiscuity.²⁰

The threat of sand has also been advanced as a justification for the circumcision of normal Western men in the twentieth century. Professor Hutson stated that when Australian soldiers were stationed in the Middle East during the First and Second World Wars 'the incidence of recurrent balanitis caused by sand under the foreskin

reached 'epidemic' proportions, leading to large numbers of soldiers requiring circumcision.'¹

Spencer Beasley, one of the authors of the Royal Australasian College of Physicians (RACP) Policy Statement, similarly stated that 'the fashion for circumcision (in New Zealand) began in the Second World War in North Africa where soldiers often went days without showers and inflammation of the foreskin from sand was the most common cause of absenteeism from the front line.'²¹ With tank battles like El Alamein raging, this seems doubtful.

Circumcision in New Zealand had become widespread in the 1930s,²² following the pattern observed in Australia in the 1910s,²³ and in Britain in the 1890s, when circumcision of male infants and boys was urged as a preventive of 'congenital phimosis,' masturbation, syphilis, epilepsy, hip joint disease, bed wetting, and many minor disorders.²⁴

It is time that the 'sand myth' was laid firmly to rest. In the North African combat zone, surgical resources were limited, and already fully committed to treating the wounded and seriously ill. Surgical procedures were kept to a minimum, since dust in wounds had far more serious effects than it could have under the foreskin. This is confirmed by the official war histories. None of the many medical volumes published by Britain, Australia, and New Zealand so much as mentions 'sand' or the 'foreskin.'

The book *British History of the Second World War* identifies the main medical problems in the Middle East and North Africa as hepatitis, diarrhoea, dysentery, tonsillitis, accidental injuries, burns, malaria, sandfly fever, and 'desert sores—this might include balanitis, but no location is specified, and the condition was not treated surgically.'^{25,26}

Neither sand nor balanitis are among the 'clinical problems of war' discussed by Allan Walker in Australia's official history (although acne gets a couple of pages), and 'desert sores' turn out to be small sores arising from cuts, grazes, and insect bites which became infected with either *Staphylococcus* or *Streptococcus*.²⁷ Nor is there any reference to circumcision in the volume devoted to medical issues in the Middle East and North Africa. As among the British troops, the main health problems encountered were gastric diseases such as diarrhoea, dysentery, and hepatitis. These certainly emphasised the need for hygiene, but not specifically of the penis; it referred to the construction of latrines, correct toilet procedures, and the control of flies.

Interestingly enough, Walker remarks that 'conjunctivitis was remarkably uncommon, in spite of dust and glare and paucity of convenience for washing;' if the blowing sand was rarely able to inflame the exposed and vulnerable eyeball, it seems unlikely that it could do much to harm to the concealed and (in uncircumcised men) well-protected glans penis.²⁸ The New Zealand history similarly states that skin inflammations were a hazard of desert warfare, and that they were exacerbated by fine sand, but it makes no mention of the foreskin as a problem site, nor of circumcision as a treatment, and goes on to comment that every effort was made to minimise cuts to the skin, and to avoid surgery unless it was 'urgent or else offered the prospect of permanent relief of symptoms sufficient to enable men to be retained in useful employment overseas.'^{29,30}

Indeed, in none of the thousands of pages contained in these volumes do the words 'balanitis,' 'circumcision,' or 'foreskin' make a single appearance.

Because the sand-balanitis-circumcision claim has been based on anecdotal evidence and never substantiated, it has not been regarded as sufficiently important to warrant refutation. As a result, it maintains a furtive existence as a medical urban myth, popping up in surprising places with odd variations.³¹⁻³³ A correspondent in the *Journal of the Royal Society of Medicine* reported that 'a German surgeon' had told him that, in the Second World War, German Africa Corps troops had 'suffered in the same way', and had similarly been circumcised.³⁴

But the idea that a German under the rule of Nazism would have submitted to an operation which could have identified him as a Jew, or that anybody in authority would have recommended such a course, is hard to credit. To check this point, Mr Hugh Young wrote to Manfred Rommel, son of the German commander, who replied: 'I have never heard that soldiers in the Africa Corps were circumcised. The veterans I could contact have not either.'³⁵ Even Aaron Fink (long-time crusader for universal neonatal circumcision, and originator of the idea that circumcision was a 'natural condom,' and thus the perfect prophylactic against HIV-AIDS)³⁶ admitted that protection against desert sand was probably not the main reason for the adoption of circumcision by the Arabs and Jews.³⁷

Conclusion—There is no evidence that tribal/ritual circumcision practices arose as a hygiene measure. And 'sand under the foreskin,' balanitis, and circumcision were not significant problems during either of the World Wars.

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Dihydropyridines, felodipine, and PHARMAC

Stewart Mann

Abstract

Calcium antagonists have evolved as useful drugs in the treatment of hypertension and angina. Dihydropyridines are the largest subgroup and various products have been marketed. Safety concerns have largely been allayed by comparative outcome trials but concern remains over short-acting products. In New Zealand, many patients requiring fully subsidised dihydropyridines have had several changes of product imposed due to successive reference pricing and sole-supply arrangements, along with deregistering and reregistering of generic felodipine. Some narrowly avoided a complete loss of access to a suitable low dose of any dihydropyridine. Generic substitution and sole-supply arrangements may make useful savings but can leave the supply of key pharmaceuticals vulnerable and impose significant loss in quality of healthcare from multiple changes in pharmaceutical preparation.

Background

Dihydropyridine calcium channel antagonists (DHPs) are powerful vasodilators used as anti-hypertensive and anti-anginal agents. Although overall benefit in angina has been observed, some patients experience worsening of symptoms or adverse cardiac events¹ and there has been concern over their safety in acute coronary syndromes.^{2,3} Cohort studies undertaken in patients taking DHPs for hypertension showed increased rates of myocardial infarction in comparison with those taking drugs from other pharmaceutical groups.^{4,5} Although a substantial number of prospective trials comparing DHPs with other antihypertensive classes^{6,7} has now largely allayed those concerns, there has been lingering doubt over the safety of the more vasculoselective DHPs and of short-acting preparations.⁸ Some uncontrolled studies of swaps between the different long-acting DHPs have not shown any safety concerns.^{9,10}

PHARMAC and calcium antagonists, 1997–9

In New Zealand in 1997, several DHPs were freely prescribable: nifedipine (as Adalat, Adalat Retard, and Adalat OROS), felodipine (as Plendil ER), amlodipine (as Norvasc), and isradipine (as Dynacirc). Various preparations of verapamil and diltiazem were also available.

For both cost and safety reasons, PHARMAC was concerned that the use of calcium antagonists as first-line agents in hypertension was growing inappropriately. PHARMAC issued proposals to treat all calcium antagonists (excluding verapamil) as an interchangeable class, and use reference pricing to restrict costs. At that time, the cheapest member of the class was short-acting diltiazem.

Consultation with various groups (including the Cardiac Society) occurred in 1998–9 in response to which PHARMAC recognised both the need to separate diltiazem from the DHP group and took note of concerns over patients taking DHPs for angina. In mid-1999, subsidies on all DHPs were referenced to felodipine (Plendil ER,

AstraZeneca Pharmaceuticals) but Special Authority provisions were made for those who were already taking other DHPs for angina or who could not tolerate felodipine.

Bayer Pharmaceuticals had offered an alternative preparation of nifedipine (Adalat Coat Core) but PHARMAC's Pharmacology and Therapeutics Advisory Committee (PTAC), although not the usual body to assess this, did not feel its release characteristics were adequate compared with the current product (Adalat Oros). At that time, felodipine held some 37% market share of all DHP prescriptions (total around 59,000 patients in New Zealand) so over 30,000 patients had to change their medication to continue to receive full subsidy. PHARMAC provided some financial help for GP visits to achieve the change.

Later in 1999, negotiations with AstraZeneca Pharmaceuticals led to a reduced price for felodipine (Plendil ER) in return for some subsidised access for their angiotensin-II receptor blocker, candesartan. This increased the co-payments of those who had chosen to continue other DHPs.

Generic substitution (and back again, and again), 2001–4

By late 2001, the patent protection on the Plendil ER brand of felodipine had expired, and PHARMAC identified and awarded a sole-supply contract for a generic (Felo ER, Pacific Pharmaceuticals) that was available in one other country (Germany).

AstraZeneca obtained results of comparative bioavailability studies under the Official Information Act and felt there was enough concern over these to commence judicial review proceedings with the object of having the approval for Felo withdrawn. While the drug entity was the same, the slow-release characteristics of Felo were questioned—an important consideration given concern over the safety of short-acting DHPs.

MEDSAFE's Generics Subcommittee reviewed the claims but stood by its original approval decision, further ratified by the Medicines Assessment Advisory Committee (MAAC) which deemed Plendil ER and Felo ER to be interchangeable. This stance contrasted with PTAC's earlier opinion of the unacceptability of comparative release characteristics of Adalat Coat Core, although it was based on accepted specific criteria and, in this case, related to different formulations of the same drug.

The switch to the generic felodipine went ahead. There were a number of anecdotal concerns from patients via doctors and pharmacists, and 118 notifications to the Centre for Adverse Reactions Monitoring (CARM) in Felo's first year compared with 69 over 14 years with Plendil. PHARMAC dismissed these as the inevitable disgruntlement that accompanies enforced change. MEDSAFE ascertained that there had been no excess of complaints about Felo ER in Germany.

In October 2002, it came to light that biostudy tests done in Europe on Felo ER used to justify its registration could not be relied on and registration was withdrawn. The 50,000 or so patients taking the drug were therefore switched back to Plendil ER; Pacific Pharmaceuticals having to subsidise the consultations necessary to achieve this. However, there was consequent further disruption¹¹ and patients had to meet the costs of a new prescription. Bioequivalence tests were then repeated on each dose of Felo ER (2.5mg, 5mg, and 10mg) and resubmitted to MEDSAFE.

The Generics Subcommittee initially felt the equivalence fell outside desirable tolerance limits for all doses but, after further submissions, accepted that their initially proposed standards were unrealistic and permitted re-registration of the 5mg and 10mg doses but not the 2.5mg dose. This created a problem for PHARMAC who wished to reintroduce reduced subsidies based on the generic pricing for these doses in late 2003 but AstraZeneca refused to provide the Plendil ER 2.5mg dose alone. (Cutting larger tablets in half destroys the slow release characteristics of either Plendil ER or Felo ER.)

Given that patients taking this low dose were doing so because of safety and tolerance concerns, this created a potentially dangerous situation which was exacerbated by a lack of well-established equivalent low doses of other DHPs.

The Cardiac Society asked PHARMAC to:

- Obtain a clinical perspective of the bioequivalence data from the Cardiovascular Subcommittee of PTAC,
- Request an opinion from its own Community Advisory Committee of the acceptability of reintroducing sole-supply of a product previously withdrawn for safety considerations,
- Ensure there would be continued availability of a felodipine 2.5mg preparation, and
- Consider again providing a full subsidy on at least one other dihydropyridine (less vasculoselective than felodipine).

No information was received to indicate that these requests had been considered, and clearly the vital question of the 2.5mg dose was subject to brinkmanship.

Thankfully, an eleventh hour agreement between PHARMAC and AstraZeneca saw the 2.5mg dose of Plendil ER continue to be available with full funding, but patients taking higher doses had to swap brands yet again to retain full subsidy. A final twist in the tale occurred in March 2004 when Pacific Pharmaceuticals were temporarily unable to satisfy the demand for Felo 5mg and, yet again, patients swapped to Plendil.

Comment

What lessons can be drawn from this tale? Reference pricing of a previously available, diverse but pharmacologically similar group of medications to a single product creates tension for prescribers who believe that there are significant differences between products based on therapeutic quality, safety considerations, or on widely disparate levels of evidence for outcome benefit. The scientifically minded are naturally distraught that such major 'experiments' are forced on a large population with little formal collection of outcome data and with negligible regulatory and ethical approval or documentation of outcome compared with the requirements for clinical trials that may have much less impact on subjects.

In order to retain full subsidy on their medication, some patients on long-term DHP treatment will have had to swap their medication several times in recent years to accommodate the pricing objectives of PHARMAC. There is at least a temporary and sometimes major quality issue here for patients, particularly for the elderly who easily

get confused about their medication. Interactions with healthcare providers become frustrating on both sides given the adverse perceptions of change (whether or not these are due to true organic disturbance), and consequent rebalancing of previously stable medication.

The importance of this loss of quality will undoubtedly remain a contentious issue between those representing their patients and a purchasing agency with a commercial imperative. Dissatisfaction of patients with the imposed substitute product purely due to the change is an undeniable phenomenon but one which cannot be interpreted fully in the absence of detailed data collection or a prospective trial.

Generic substitution is an acceptable and commercially important practice in reducing consumer costs. However, not all preparations of the same drug are exactly the same; such factors as release mechanisms having importance where markedly fluctuating levels of a drug create safety concerns. Analysis of bioequivalence is complex and studies asserting this frequently do not use preparations and experimental subjects directly equivalent to the usual clinical situation.

Extension of reference pricing or generic substitution into sole-supply arrangements can leave healthcare provision vulnerable in the event of a quality issue arising which renders the chosen drug formulation temporarily or permanently unusable.

Alternatives may not be readily available when minimal market share has discouraged suppliers from maintaining infrastructure in the country necessary to facilitate urgent supply (or resupply) of a product.

By analogy, we would not expect the same quality controls when buying an imported used car from a second-hand dealer than we would from a new model bought from a franchised dealer of the original manufacturer. There may be good reasons for purchasing the former but we have to admit the compromise and try not to drive all the other dealers out of town so that when our vehicle breaks down, there are no easy alternatives.

Timeline of various arrangements affecting supply of dihydropyridines in New Zealand

(CAAs = calcium antagonists, DHPs = dihydropyridines, GSC = MEDSAFE's Generics Subcommittee)

Prior to	1997	Nifedipine OROS, amlodipine, isradipine, and felodipine available and fully subsidised.
	1997	PHARMAC initiates discussions on reference pricing for CAAs.
Dec	1998	Formal proposal for reference pricing DHPs to felodipine (Plendil, AstraZeneca Pharmaceuticals).
Jun	1999	Reference pricing implemented.
	1999	Plendil price reduction; subsidies for all CAAs reduced.
Feb	2000	Felo (Pacific Pharmaceuticals) approved for use in NZ by MEDSAFE.
	2001	Successful sole-supply tender for Felo. AstraZeneca commence judicial review proceedings.
Dec	2001	MEDSAFE'S GSC confirms soundness of original Felo approval.
Jun	2002	GSC again confirms Felo approval and interchangeability with Plendil.
Oct	2002	Felo de-registered as registration data 'unreliable', Plendil resubsidised. Further bioequivalence work commissioned.
Jun	2003	GSC rules bioequivalence of Felo (all doses) unacceptable.
Aug	2003	GSC approves bioequivalence of Felo 5mg and 10mg; rejects 2.5mg.
Dec	2003	Reintroduction of reference pricing to Felo 5mg and 10mg. 'Last minute' deal with AstraZeneca to supply Plendil 2.5mg.
Mar	2004	Felo 5mg temporarily unavailable; interim supply of Plendil.

Disclosures: I acted in the late 1990s as a designated representative on pharmaceutical policy for the Cardiac Society and from 2002–4 as New Zealand Regional Chairman of the Society, periods when these issues were very much on the agenda. I have provided occasional consultancy advice to AstraZeneca Pharmaceuticals. Since 2004, I have been a member of the cardiovascular subcommittee of the Pharmacology and Therapeutics Advisory Committee (PTAC) to PHARMAC but have not participated in this capacity in any discussions on DHPs.

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Human instincts, normal and pathological: self-preservation, or the love of life

This extract comes from a speech read at the Annual Meeting in Auckland by Herbert Barraclough, M.B., Auckland, and published in the New Zealand Medical Journal 1905, Volume 4 (16), p202–203.

“Are all suicides due to insanity?” And my answer is an emphatic affirmative. As an example to the contrary, we may conceive of a murderer fleeing from justice and in imminent danger of capture, blowing out his own brains, and still not be insane. But this is scarcely a case in point, as the man is only choosing between two forms of death—that at his own hands or those of the executioner—and deliberately choosing the former.

I remember the case of a medical student in Leeds, who poisoned himself by prussic acid, against whom the jury returned this very unusual verdict of “*Felo de se*”, on the ground that there was no motive for the act. Why, the very fact of there being no notice was presumptive proof of insanity; and this case was probably one of those impulsive, unconscious acts of suicide which occasionally occur. Frequently careful investigation reveals signs of insanity too slight to have been noticed by the eyes of a layman which have preceded the suicidal act.

In the absence of these, the very nature of the act sometimes points to insanity, as in a case which occurred some years ago when a young man who had every opportunity of committing suicide by other means deliberately thrust a red-hot poker through the roof of his mouth, penetrating the brain, and lived for twenty-four hours afterwards in mortal agony.

The whole balance of accumulated evidence points only in one direction, both theoretically and practically, and leads us to the inevitable conclusion that when no previous symptoms of insanity have been noticed they have been overlooked; and though every case must be judged on its own merits, I think that the very general verdict of “Temporary insanity”, given in cases of suicide, is technically correct, and not merely a matter of sentiment.

Whatever crazy sorrow saith,
No soul that breathes with human breath
Hath ever truly longed for death.
Ah, no. 'Tis life, not death, we ask—
More life and fuller that we want.

We no longer bury our suicides at midnight at the nearest cross-roads with a stake through their bodies. No, we are too civilised for that. But the Church still brands their souls by denying that they die “in sure and certain hope of everlasting life”, though it accords that same hope to those who have led a vicious life. And the emissaries of the law still hale before the Bench those who have attempted suicide, treating them as common criminals.



Proceedings of the Christchurch Medical Research Society's AGM and Scientific Meeting, 11 May 2005

The role of the natriuretic peptides in cardiac development

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Atrial (ANP) and Brain (BNP) natriuretic peptides protect against the adverse changes in cardiac structure and function, known as cardiac remodelling, that occur during heart disease progression. This cardiac remodelling is largely attributed to the disease state re-activating a poorly-defined fetal gene program. Mice that lack the *Npr-1* receptor mediating ANP and BNP bioactivity exhibit cardiac remodelling. We have observed that the number of surviving *Npr-1* knockout (KO) embryos declines significantly during gestation and the neonatal period, and propose that the natriuretic peptides play a previously unrecognised role in fetal cardiac development. We compared cardiac anatomy, histology and gene expression of *Npr-1* KO and wild-type (WT) hearts at three key time points in cardiac development, 12.5 and 15.5 days post coitum (p.c) and neonatal day one in both male and female mice of each genotype (n=6 per group). Increased heart size was apparent in KO mice from 15.5 days p.c, but cardiac fibrosis was not evident until eight weeks of age. Microarray analysis on 22k Oligo arrays of *Npr-1* KO and WT embryos and neonates indicated altered expression (p<0.05) of genes involved in cardiac structure (Myosin light chain, Collagen I & III), developmental axis determination and regulation of transcription (GATA-4 & 6, Mef 2A & 2B, Activin IIB Receptor pathway), myocyte cell proliferation and hypertrophy (ANP, CamK4, MAPKKK5), as well as genes involved in energy utilisation and metabolism (GAPDH, GSK-3B). In summary, in addition to their cardioprotective effects in the adult heart, the natriuretic peptide family appears to interact extensively with several developmental signalling pathways.

The pattern electroretinogram in normal myopic eyes

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Myopia, commonly referred to as short sightedness, is a common cause of visual disability throughout the world and affects 25% of the western population. Myopia causes parallel light to focus in front of the retina. It is usually a consequence of the axial length (AL) of the eye being too long. An association between myopia and glaucoma has been documented. In glaucoma, the pattern electroretinogram (PERG) amplitude is often reduced before it is possible to detect a scotoma in the patient's visual field.

The aim of our study was to determine whether the AL can also influence the PERG amplitude. Thirty five normal myopic volunteers (mean age = 32 years; SD = 5.5 years) participated in this study which received approval from the Canterbury Ethics Committee. Each volunteer had passed a complete ophthalmic screening examination and had a best corrected visual acuity of 6/9 or better. Only the results of the right eye from each volunteer were included in the statistical analysis.

Our findings confirm a significant correlation between the AL of normal myopic eyeball and the PERG amplitude (correlation coefficient $r = -0.42$; $p < 0.01$).

In conclusion, the longer axial length of a myopic eyeball may also be responsible for the reduction of PERG amplitude and therefore needs to be considered when interpreting the PERG results.

Doppler ultrasound evaluation of arterial blood velocity during vasovagal syncope

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Vasovagal syncope (or fainting) is a fall in blood pressure secondary to generalised vasodilation, although the exact mechanism is unknown. This reaction can be reproduced in the laboratory using tilt testing. We hypothesised that during tilt-induced syncope, blood flow to the gut is increased.

We measured superior mesenteric arterial blood velocity using pulsed doppler ultrasound, with blood pressure, heart rate and sympathetic nerve activity continuously in 40 patients. Patients were initially monitored in the horizontal (base-line) position, then at 60 deg head-up tilt until syncope, or for 30 minutes if they tolerated the tilt position. Recordings were made at one minute intervals. The Doppler wave-forms were analysed off-line using running averages to plot a maximum velocity envelope. An area ratio was derived to show blood flow changes in each patient during tilt.

Mean horizontal ratios in 5 syncopal patients were 1.36 ± 0.38 ; after a 10 minute tilt 1.14 ± 0.23 ; and at syncope 15.10 ± 5.86 . In tilt-tolerant patients horizontal and tilt ratios were 1.38 ± 0.22 and 1.56 ± 0.31 respectively, and remained constant throughout tilt.

Using running average imaging techniques it is possible to process pulsed Doppler wave-forms and gain consistent data from patients during tilt and tilt syncope. This has allowed us to demonstrate that surprisingly, blood flow velocity is severely decreased during tilt induced syncope.

Active insulin control with variable nutrition for targeted glucose control in critically ill patients

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Stress-induced hyperglycaemia is prevalent in intensive care. Tight glucose control can reduce mortality up to 43% if levels are kept below 6.1 mmol/L. This research develops adaptive control algorithms varying both insulin dose and nutritional inputs for targeted glucose control of critically ill patients

To verify the effectiveness of the protocol, retrospective data from 19 ICU patients were used for test simulations. Results are evaluated against the hospital sliding-scale data recorded and insulin-only adaptive control. Results indicate a 312% increase in time spent in the 4-6mmol/L normal glucose range compared to the standard sliding-scale approach and a 240% increase compared to an insulin-only control algorithm. A normally distributed $\pm 7\%$ sensor error of the standard *Glucocard II* glucose sensor added a mean variability of 2.9% and standard deviation of 1.7% to the results. Finally, note that this protocol has a 25-30% higher average nutrition input rate than the retrospective data for the patients.

The protocol was clinically tested in the Christchurch ICU in seven 10-hour trials. Glucose targets were achieved 82.5% of the time within the 7% measurement error, with the remainder having a 24.4% mean difference and standard deviation less than 12%. However, for missed targets, the absolute errors range of [0.8, 2.9] mmol/L was very small indicating small errors at low glucose values rather than a failure of the algorithm. Glucose levels at the end of the trial were 40% lower, on average, compared to the starting value. Overall, the protocol is very effective at tight control to 5 mmol/L over a wide range of ICU patients, despite changes in condition, while also providing greater nutritional input.

Computer simulations of tight glucose control in critically ill patients using a specialized insulin-nutrition-table

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Critically ill patients often have stress-induced hyperglycaemia and keeping glucose levels below 6.1 mmol/L can reduce mortality by 43%. Computerized protocols are effective but require frequent measurement. This research presents a table-based method suitable for clinical practice. Retrospective data from 19 ICU patients

representing a broad range of conditions was used to model insulin-glucose dynamics. The table-based scale and two “standard” sliding scales were tested with this model.

The specialized table can be easily applied by clinical staff and is based on prior computerized trials. It is designed to work using 1-2 hour measurement intervals to minimise clinical effort. The approach varies both insulin dose and nutritional input rate to achieve tight control. The standard sliding scales simply match current glucose level to an insulin dose rate until the next measurement. The two sliding scales represent different levels of aggressiveness in insulin dosing. All scales are simulated using 1, 2 and 4 hour measurement intervals.

Time in the 4-6 mmol/L band increased from 41% to 67% for the specialized table versus the sliding scales. Time above 6 mmol/L went from 56% to 28.5% with no hypoglycaemia recorded. The specialized table matches computerized protocols and also provides greater nutritional input over the patient stay while providing better control. Finally, increasing measurement interval decreases effectiveness of sliding scales 10- 20%. Thus, frequent measurement is critical to maintaining tight control given highly variable patient condition. Clinical testing will be required to validate these results.



Proceedings of the 178th meeting of the Otago Medical School Research Society, 7 July 2005

Possible modulation of serotonin receptor 2A by filamin-C. Erin Cawston, A Fitches, D Markie, R Olds. Department of Pathology, Dunedin School of Medicine, University of Otago, Dunedin.

Serotonin receptor 2A (5-HT_{2A}) has many biological roles. In the peripheral nervous system it has been shown to mediate contraction of vascular and extra-vascular smooth muscle and is involved in platelet aggregation. In the central nervous system, clinical studies have shown that 5-HT_{2A} may be differentially regulated in schizophrenia and other psychiatric disorders, but the direction and magnitude of these changes remains controversial. It has also been shown that classic hallucinogens and atypical antipsychotic drugs act principally through 5-HT_{2A} to modulate human perception and cognition. We hypothesised that intracellular proteins that interact with the cytoplasmic C-terminal of 5-HT_{2A} may modulate the function of the receptor.

The C-terminal cytoplasmic region of murine 5-HT_{2A} was used to find binding partners from a 7 day mouse embryo cDNA expression library. This mating strategy screened the C-terminal of 5-HT_{2A} (amino acids 384-471) by a yeast two-hybrid approach. BLAST searches of sequence databases showed similarity to murine filamin-C for seven of the interacting clones. The interaction between the human homologues of 5-HT_{2A} (aa387-471) and filamin-C (aa2141-2705) was confirmed using the yeast two-hybrid system and by an *in vitro* capture assay. The interaction is biologically plausible, as co-expression of both 5-HT_{2A} and filamin-C was identified in two of eight neural cell lines. The 5-HT_{2A}-binding domain of filamin-C has been further localised to repeats 20 and 21 and appears to be enhanced by the presence of a unique sequence, found only in filamin-C and not in the closely related filamins A or B.

Filamin proteins have been shown to act as scaffolding proteins, therefore we suggest the interaction between filamin and 5-HT_{2A} may provide a mechanism for internalisation or recycling of the receptor, so influencing the sensitivity of the cell.

The interaction between the delta epithelial sodium channel (δ ENaC) and its downregulator Murr1. Tina Chang, FJ McDonald. Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

The epithelial Na⁺ channel (ENaC) is an important regulator of salt and water balance in the body, and hence blood pressure. The classic ENaC consists of three subunits, α , β and γ . A fourth ENaC subunit, named δ , is highly expressed in "non-traditional" epithelial tissues, such as brain, testis, ovary and pancreas, and the δ ENaC channel is activated by acidic extracellular pH. However, the physiological role of δ ENaC remains uncertain. A copper-toxicosis related protein called Murr1 was identified as a binding partner of δ ENaC, and it inhibits δ ENaC channel activity through direct interaction. The present study aims to explore the interaction between δ ENaC and

Murr1 by investigating the sites of protein-protein interaction and subcellular locations.

Deletion constructs for both δ ENaC and Murr1 were prepared, and a glutathione S-transferase pull down assay and Western blot analysis were used to identify the sites of protein interactions. The Murr1 binding site was narrowed down to amino acids 592-615 of δ ENaC, and the δ ENaC binding site in Murr1 was located between amino acids 140-150. Immunocytochemical studies and subcellular fractionation revealed that FLAG-tagged δ ENaC (δ ENaC_{FLAG}) is located in the cytosol, in association with vesicular compartments, and HA-tagged Murr1 (Murr1_{HA}) is located around the nucleus in COS-7, HEK293 and MDCK cell lines. When coexpressed, Murr1_{HA} colocalised with δ ENaC_{FLAG}, suggesting regulation of δ ENaC by Murr1 occurs in the cytosol.

In summary, the binding site for Murr1 was narrowed down to a 24 amino acid region in the δ ENaC C-terminal domain, whereas the binding site for δ ENaC was narrowed down to a 10 amino acid region in Murr1. The cytosolic colocalisation of δ ENaC_{FLAG} and Murr1_{HA} suggests that Murr1 may be a regulator of δ ENaC intracellular trafficking.

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Clomethiazole spares mitochondrial dysfunction during hypoxia-ischaemia-induced hippocampal diaschisis. Andrew N Clarkson, DM Jackson, DS Kerr, IA Sammut. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.

Transhemispheric diaschisis describes remote changes that occur contralateral to an ischaemic insult. We have shown that cerebral hypoxia-ischaemia (HI) results in lasting suppression of hippocampal CA1 region activity, contralateral to a carotid artery occlusion in post-natal day 26 rats. Mitochondria have been shown to be specifically damaged by oxidative stress arising during an ischaemic insult, resulting in diminished respiratory function and ATP production.

The present study assessed mitochondrial dysfunction in rat hippocampal tissue homogenates, contralateral and ipsilateral to carotid artery occlusion. Assessment of mitochondrial impairment was carried out using oxygen electrode-derived respiratory measurements and mitochondrial electron transport chain enzyme kinetics (enzyme complexes I-V). Mitochondrial dysfunction was also assessed following clomethiazole (CMZ; 414 mg/kg/day via subcutaneously implanted mini-pumps), a GABA_A receptor agonist with known neuroprotective properties. Mitochondrial FAD-linked (succinate-driven) respiration and complex kinetics were assessed 1 and 3 days post-HI. Results are expressed as the mean \pm SEM for n = 6-8 separate observations. Statistical analysis was performed using two-way ANOVA and Newman-Keuls' multiple pair-wise post-hoc comparisons.

Mitochondrial function was impaired contralaterally at 3 days (2.76 ± 0.23 versus 3.74 ± 0.06 ; $P < 0.01$) post-HI compared to controls. CMZ treatment resulted in improved mitochondrial function contralaterally compared to HI + saline treatment at 3 days (3.32 ± 0.22 ; $P < 0.05$), and was not different compared to controls at either 1 or 3 days post-HI.

This study demonstrates for the first time that HI induces impaired mitochondrial function in hippocampi contralateral to a carotid artery occlusion, and further extends the range of neuroprotective properties previously described for CMZ.

This work was supported by an Otago Medical Research Foundation Grant and an Otago University Research Grant.

Adult phytoestrogen exposure reduces the fertility of male rats. Amy Glover and S Assinder. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Phytoestrogens are plant-derived compounds that are able to activate oestrogen receptors α (ER α) and β (ER β). While oestrogen is vital for male reproductive function, exposure to endogenous oestrogen has been implicated in declining male fertility. The aim of this study was to determine whether phytoestrogen exposure reduces male fertility, and via what mechanism.

Male Wistar rats were bred and raised on a low phytoestrogen diet containing 112 $\mu\text{g}\cdot\text{g}^{-1}$ isoflavanoid. Six adult males were transferred to a high phytoestrogen diet (465 $\mu\text{g}\cdot\text{g}^{-1}$ isoflavanoid), while 9 adult males remained on the low phytoestrogen diet. After 3, 6 and 12 days all males were mated with low phytoestrogen fed females, and the litter sizes recorded. A second group of male rats was kept on the same dietary regimen and euthanased 3, 6 or 12 days after the change in diet. The mRNA levels of ER α , ER β and androgen receptor (AR) in the epididymides of the male rats were measured by real time PCR.

The average litter size (mean \pm SEM) was significantly reduced from 13.0 ± 1.1 to 8.6 ± 1.7 pups when male rats were fed the high phytoestrogen diet for 3 days ($P < 0.02$; paired t -test), but were at control levels by day 12. ER α and AR mRNA levels (mean \pm SEM) were significantly reduced from 0.572 ± 0.042 to 0.244 ± 0.045 $\text{ng}\cdot\text{g}^{-1}$ of total RNA ($P < 0.001$) and from 7.92 ± 1.8 to 2.67 ± 0.67 $\text{ng}\cdot\text{g}^{-1}$ of total RNA ($P < 0.02$) respectively in the cauda region of the epididymides of rats fed the high phytoestrogen diet for 3 days.

Acute exposure of adult male rats to a high phytoestrogen diet transiently reduces their fertility. We propose that the alteration in steroid hormone receptor mRNA in the epididymis results in decreased steroid hormone receptor protein which increases sperm oxidative stress and leads to reduced fertility.

Supported by a University of Otago Research Grant, the Community Trust of Otago and a University of Otago Postgraduate Scholarship.

Community behaviour of stem cells: Quantitative evidence from cellular patterning during myogenesis *in vivo*. Antonio S J Lee, N Yoon, M Zhang. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

The fate of stem cells is controlled by intrinsic and extrinsic factors. Integration of both factors is achieved by single cells and across cell populations, the so-called “community effect”. This study examined the *in vivo* behaviour of stem cell communities during skeletal myogenesis.

Isolated cells from extensor digitorum longus (EDL) and tibialis anterior (TA) muscles of 229 Wistar rat embryos in 15 litters at embryonic days (E) 15 ~ E19 were prepared for immunocytochemistry. Anti-Pax3, anti-Pax7, anti-myosin heavy chain (MHC) and a cocktail of antibodies against myogenic regulatory factors (MRF), including anti-MyoD, anti-Myf5 and anti-myogenin antibodies, were employed to mark various myogenic stem cell (MSC) communities at various differentiation stages.

The MSC communities displayed two distinct behaviours during myogenesis. Pax3^{+ve} and MRF^{+ve} communities maintained their relative size throughout myogenesis ($51.4 \pm 1.6\%$, mean \pm SEM, $n = 15$ and $56.4 \pm 2.0\%$, $n = 15$, respectively), contributing to the main stream of the MSC pool in a developing muscle. The MHC^{+ve} community was also constant, yet extremely low in proportion ($0.8\% \pm 0.1\%$, $n = 15$) throughout myogenesis. In contrast, the relative size of the Pax7^{+ve} community increased significantly from E15 ($5.3 \pm 0.3\%$, $n = 3$) to E18 ($32.8 \pm 1.4\%$, $n = 3$, $P < 0.001$; ANOVA single factor) and was maintained at E19 ($31.3 \pm 1.5\%$, $n = 3$).

In MSC, Pax3 and/or Pax7 act upstream of MRF followed by expression of MHC immediately prior to fusion. The observed features of the MSC community behaviour will be essential for normal skeletal myogenesis. Stable Pax3^{+ve} and MRF^{+ve} communities may provide a consistent source of differentiating cells for muscle growth, while an expanding Pax7^{+ve} community may supply cells to meet specific requirements as development proceeds. A small yet stable MHC^{+ve} community may contribute to the consistent rate of growth.

The neuroprotective effects of melatonin in stroke-induced brain damage, via modulation of L-arginine metabolism. Shiva M Nair, RMA Rahman, I Appleton. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.

Currently, there are no agents that can prevent the neurodegeneration which occurs after a stroke. Melatonin has been implicated in numerous physiological processes. Recently it has also been shown to exhibit neuroprotective effects against acute focal cerebral ischaemic damage possibly by acting as a free radical scavenger. However, the mechanisms of these neuroprotective effects have not been determined. Therefore, this project explored the mechanism(s) by which melatonin acts as an acute neuroprotective agent post-stroke.

Male Sprague-Dawley rats, 285 ± 15 g underwent a 2-hour transient occlusion of the middle cerebral artery by filament insertion. Rats were treated with 5 mg/kg i.p. daily melatonin or vehicle (5% DMSO in 0.9% saline) for 3 days. Brain damage was assessed histologically at 72 hours post stroke with the use of 2,3,5-triphenyltetrazolium chloride stain. We focused on pivotal inflammatory enzymes, namely nitric oxide synthase (NOS) and its product, nitric oxide which breaks down to nitrite, and the enzyme arginase. Enzyme activity was measured in brain homogenates taken 3 days post-stroke.

A decrease in infarct volume in the melatonin group (46 ± 8 mm³, mean \pm SEM, $n = 6$, $P < 0.05$; Mann Whitney U test) compared to controls (155 ± 33 mm³, $n = 7$) was observed. No significant effects on arginase and NOS activity were observed with

melatonin. However, a significant decrease in nitrite levels occurred with melatonin treatment (9.5 ± 1.1 μ M nitrite/mg protein, $n = 7$, $P < 0.001$) compared to non-intervention controls (32 ± 5 μ M nitrite/mg protein, $n = 6$). Elevation of inducible NOS activity was seen in the vehicle treated group (510 ± 140 [3 H] L-citrulline/mg protein, $P < 0.05$) in comparison to control (230 ± 60 [3 H] L-citrulline/mg protein) and this was reduced towards normal (non-intervention controls) with melatonin treatment.

This study clearly demonstrated that melatonin is a key mediator in the post-stroke neuronal inflammatory response. In addition, this is in part, due to its inhibition of the enzyme inducible NOS as well as its free radical scavenging properties on its product nitric oxide.

Supported by a grant from the Health Research Council of New Zealand (SN), Bright Future Fellowship NZ (RR) and Lottery Health New Zealand (IA).

The role of melatonin on scarring in an incisional model of dermal wound healing in rats. Kamali Pugazhenth, M Kapoor, P Young, A Clarkson, I Hall, I Appleton. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.

Melatonin possesses anti-oncotic, anti-inflammatory and immunomodulatory effects. However, its role in wound healing has not been established. In this study we determined the effects of melatonin on scarring using a full thickness incisional wound healing model in rats.

Wounding was initiated in male Sprague Dawley rats 250 ± 25 g ($n = 6$ /group/time point). Four 1cm incisions were made on the dorsum of each rat. The treated and control animals received 1.2 mg/kg melatonin or saline intradermally, at 11 pm (compliant with the rats daily melatonin rhythm), 24 hours prior and daily for a further 7 days post-wounding. Rats were sacrificed on days 0, 1, 3, 7, 14, and 21, at 2 pm (the time of maximal melatonin levels), and biopsies extracted. Homogenates of biopsies were assessed for arginase and nitric oxide synthase (NOS) activity and nitrite levels. Immunohistochemical studies on melatonin receptor expression were also determined. A collagen stain (van Gieson's) was used to assess the quality of scarring.

Melatonin receptors (MT1 and MT2) were expressed in the epidermis and macrophages on day 1 in both groups. Thereafter the expression of MT1 decreased post day 3. The blind study showed that melatonin treatment significantly reduced scarring on day 21. Melatonin treatment also significantly ($P < 0.01$) increased arginase activity at 1 and 3 days and iNOS activity at day 7 ($P > 0.01$). Statistical analysis at each time point for the controls and melatonin group was performed using Mann Whitney-U test.

Arginase generates proline, the building block for collagen synthesis. Melatonin treatment increased arginase activity and thus collagen synthesis from day 1. It has been reported that increased nitric oxide (NO) production prolongs the inflammatory phase of wound healing. We conclude that melatonin improves scarring and that this effect is, in part, mediated by modulation of iNOS and arginase.

Spatial distribution of COX-1, COX-2 and iNOS enzymes in the rat brain following hypoxia-ischaemia. Odette M Shaw, AN Clarkson, BA Sutherland, I Appleton. Department of Error! Bookmark not defined., School of Medical Sciences, University of Otago, Dunedin.

Cyclooxygenase-2 (COX-2) is induced following various stimuli in the brain. As well as an increase in COX-2 mRNA there is a concomitant increase in the inducible isoform of nitric oxide synthase (iNOS) expression in animal models of stroke. Both COX-2 and iNOS are reported to cause cellular injury by mediating inflammation and free radical damage. The aim of this study was to determine the spatial and cellular distribution of COX-1 (the constitutive isoform of COX), COX-2 and iNOS proteins in the hypoxia-ischaemia (HI) model of neurodegeneration.

HI was induced in male Wistar rats (n=4 for both controls and HI) by permanently ligating the left common carotid, followed 2 hours later by 1 hour of hypoxia. Animals were sacrificed 3 days post HI. The distribution of COX-1 and COX-2 in the brain were determined by immunohistochemistry. The colocalisation of COX-2 and iNOS were determined by double immunofluorescence labelling.

COX-1, in control and HI brains, was uniformly expressed throughout all brain cells and regions. In the HI sections, COX-2 positive astrocytes, neuroglia, activated microglia and hippocampal neurons were localised throughout the ipsilateral hemisphere. In addition, infiltrating macrophages within the ischaemic penumbra were immunolabelled for COX-2. The double immunolabelling studies demonstrated high levels of COX-2 expression in the nucleus of cells which were colocalised with iNOS. This demonstrates that COX-2 expression is up-regulated in immune cells that are distributed widely throughout the ipsilateral hemisphere. COX-2 and iNOS were colocalised within the ipsilateral hemisphere illustrating the potential neurodegenerative relationship between these two enzymes.

Previous studies have shown that COX-2 can mediate the resolution of inflammation. It is therefore possible that during the time course of HI, COX-2 up regulation may not be detrimental by itself. However, the coexpression of iNOS and COX-2 is more indicative of the greater neurodegenerative potential of the iNOS enzyme.

Funding provided by the Lottery Board and the Neurological Foundation of New Zealand.

Use of the oestrogen receptor antagonist ICI-182,780 to alter central steroid actions. Frederik J Steyn, GM Anderson, DR Grattan. Centre for Neuroendocrinology and Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

This experiment evaluated the effectiveness of intracerebroventricular administration of the oestradiol receptor antagonist ICI-182,780 (ICI) in blocking central oestradiol action without compromising peripheral steroid effects. This was done as part of our studies on the central actions of steroids during late pregnancy. Two centrally regulated endpoints were measured: luteinising hormone (LH) pulse frequency and hypothalamic progesterone receptor (PR) expression.

Crystalline ICI-containing cannulae were implanted into a lateral ventricle of ovariectomised, oestrogen-treated rats ($n = 7$). Two ovariectomised control groups (no ICI) were treated with oestrogen ($n = 9$) or placebo ($n = 6$). At 70 h post-surgery blood samples were collected every 10 min for 3 h and analysed for LH concentration. Animals were paraformaldehyde-perfused, their uterine weight was recorded and their brains were sectioned ($40\ \mu\text{m}$) for PR immunocytochemistry.

In controls oestrogen suppressed LH pulse frequency (2.2 ± 0.4 versus 5.2 ± 0.18 pulses/3 h in placebo rats, mean \pm SEM, $P < 0.05$, Student's t -test). This effect was blocked by ICI (4.4 ± 0.2 pulses/3 h, $P < 0.05$ versus oestrogen-treated controls). Oestrogen increased the number of PR positive stained cells in the arcuate nucleus of controls (92.8 ± 3.9 versus 0.1 ± 0.07 , $P < 0.001$). In contrast to the LH results, ICI did not affect oestrogen induced PR expression (82.8 ± 2.1). Oestrogen stimulated uterine growth ($P < 0.001$) irrespective of ICI treatment (placebo controls: 0.18 ± 0.01 g, oestrogen-treated controls: 0.51 ± 0.05 g, ICI-treated: 0.60 ± 0.07 g).

These data show that ICI can block the central effects of oestrogen's negative feedback on LH pulses. However the PR results suggest that this central effect was incomplete. The absence of an effect on uterine growth confirmed that central delivery of ICI does not disrupt peripheral steroid actions.

Supported by a University of Otago Postgraduate Scholarship

Domoic acid directly inhibits cardiac mitochondrial respiratory function.

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Domoic acid (DOM) is a potent neurotoxin produced by marine phytoplankton, and has been implicated in numerous cases of animal and human poisoning. Haemodynamic disturbances and cardiac lesions have been documented and questions have been raised as to whether these effects are directly mediated by DOM.

In the present study, embryonic rat H9c2 cardiomyocytes exposed to DOM ($0.05 - 10\ \mu\text{M}$; 40 min) were shown (Enzyme linked immunosorbent assay) to contain DOM within the cytosol of cells, with little evidence of binding to the cellular membrane ($n = 5$, $P < 0.001$, One way ANOVA, Bonferroni Post hoc). Lactate dehydrogenase (LDH) assays indicated that DOM did not induce cellular damage acutely (20 min, 40 min, 24 h; $0.05 - 10\ \mu\text{M}$) which may indicate active transport of DOM across cellular membrane rather than a physical perturbation of the membrane.

Acute DOM exposure (20 min; $1 - 5\ \mu\text{M}$) did not alter haemodynamic parameters in an *ex vivo* whole heart preparation. Mitochondria isolated from our *ex vivo* model however exhibited increased mitochondrial FAD-linked state 4 and decreased state 3 respiratory function following exposure to DOM ($n = 7$; $P < 0.001$). The DOM analogue kainic acid (KA) ($50\ \mu\text{M}$) exhibited similar effects.

Isolated mitochondrial FAD ($n = 5$) and NAD^+ -linked ($n = 6$) respiratory function was markedly reduced ($P \leq 0.001$) by *in vitro* treatment with DOM ($50 - 250\ \text{nM}$; 10min). Individual mitochondrial enzyme complex activities were assessed to examine the extent of mitochondrial damage. Complexes I, II/III, IV, V and matrix marker enzyme

citrate synthase activities were all decreased by DOM and KA (0.5 - 2 μ M) in a concentration-dependant manner ($P \leq 0.01$). Results obtained using fluorescent probe analysis of superoxide and hydrogen peroxide levels in mitochondria and H9c2 cells indicated reactive oxygen species were not significantly elevated in DOM induced mitochondrial damage. These results confirm the potential for DOM to access and affect mitochondria directly within cells.

This work was supported by the NZ Health Lottery Board and the NZ Heart Foundation.



Cervical Asymmetry

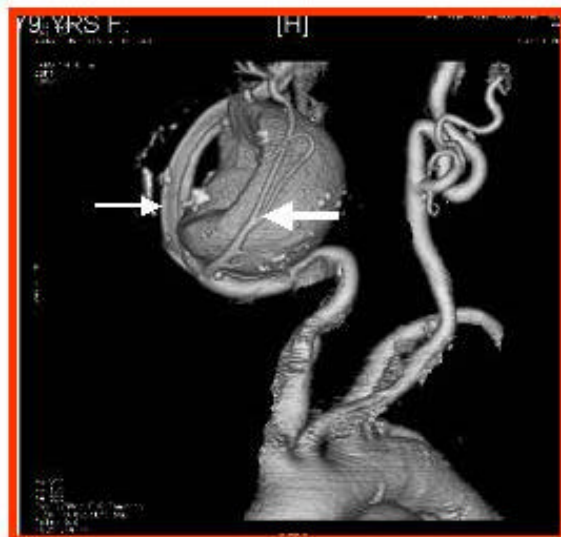
A 79-year-old Asian Indian female presented with a 10-year history of a pulsatile swelling in the right side of her neck, which increased in size without causing symptoms other than disfigurement (Figure 1).

Questions: What is the differential diagnosis and what does Figure 2 show?

Figure 1



Figure 2



Answers

Differential diagnosis of pulsatile mass in neck:

- Carotid artery aneurysm.
- Carotid artery pseudoaneurysm.
- Malignant lymphnode mass.
- Carotid body tumour.
- Soft tissue sarcoma.
- Tortuous carotid artery.

Discussion:

CT Angiography (Figure B) shows a broad-necked fusiform aneurysm arising from a tortuous internal carotid artery (small arrow), displacing the external carotid artery anterolaterally (bold arrow). The distal internal carotid artery and the intracranial circulation were patent. The most likely aetiology for this giant carotid aneurysm was atherosclerotic or degenerative.

The causes of carotid aneurysm include atherosclerosis, trauma, and previous carotid surgery.¹ Resection of the aneurysm with restoration of flow is the preferred method of treatment.² Complete excision of a giant carotid artery aneurysm entails difficulty in exposure of the most distal internal carotid artery. The risk of injury to nearby cranial nerves is also increased. Endovascular techniques with placement of covered stents have been successfully used for the treatment of external carotid artery (ECA) pseudoaneurysms,³ and a covered stent is particularly advantageous in complicated cases like aneurysms associated with an arteriovenous fistula.⁴

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Call the doktor

In the United Kingdom, health sector recruitment agencies are bombarding German doctors with faxes begging them to cover for British general practitioners who have opted out of weekend work. According to the German Medical Association, more than 2,600 doctors are commuting at weekends to Britain, where they can earn up to £2,000 for a couple of days' work. Apparently British family doctors believe that the current system for out-of-hours consultations, including the recruitment of overseas doctors, is putting their patients' lives at risk. At a British Medical Association conference they voted overwhelmingly for more money for weekend and overnight work to protect the service. The BMA is also strongly opposed to the trend of replacing local surgeries with large-scale group practices, to save money. The weekend bit sounds a bit familiar.

Guardian Weekly, 24–30 June 2005

An ex-editorial blast on drug trials

Richard Smith, former editor-in-chief of the *British Medical Journal*, was renowned for his intelligent and often cynical views on matters medical. He now works for a private health care organisation but his cynicism remains unabated. He has recently written that “medical journals have become little more than marketing tools for the pharmaceutical industry and most trials are paid for by companies, and they have become adept at ensuring that the results are favourable.” But there is more. He also says “I must confess that it took me almost a quarter of a century editing for the *BMJ* to wake up to what was happening.” Smith argues that more trials need to be publicly funded, while the results of those that are paid for by companies should be published only on regulated websites, not in journals.

New Scientist, 21 May 2005

Atypical antipsychotic drugs for dementia?

The US Food and Drug Administration (FDA) is still weathering the storm about its part in the COX-2 debate. Notwithstanding, it is still widely respected and its advice requires consideration. It has recently issued a public health advisory warning of fatal adverse events in patients with dementia treated with atypical antipsychotic drugs. The drugs named include risperidone, clozapine, olanzapine and quetiapine, all of which seem to be in quite common use in New Zealand. Apparently 17 controlled studies of elderly patients with dementia have shown that patients treated with the drugs were 1.6 to 1.7 times more likely to die than patients given placebo. The causes of death included congestive heart failure, sudden death, and infections such as pneumonia.

BMJ 2005;330:922



Transient ischaemic attacks—warfarin versus aspirin

Efficacy for stroke prevention with warfarin is well documented in those patients with atrial fibrillation. Similarly, aspirin and dipyrimadole are helpful in those at risk in sinus rhythm. Some believe that warfarin is even better for the latter group. Not so. In fact, dangerous. In a recently reported trial, researchers randomly assigned patients at very high risk, but in sinus rhythm, to receive warfarin (target international normalized ratio, 2.0 to 3.0) or aspirin (1300 mg per day) in a double-blind, multicenter clinical trial. The primary end point was ischemic stroke, brain hemorrhage, or death from vascular causes other than stroke. The results—warfarin was associated with significantly higher rates of adverse events and provided no benefit over aspirin in this trial. Aspirin should be used in preference to warfarin for such patients. I wonder why they used a 1300 mg dose of aspirin? 75–150 mg/day is the usual dose.

N Engl J Med 2005;352:1305–16

Routine episiotomy??

Apparently, episiotomy is among the most common surgical procedures experienced by women in the United States. Thirty percent to 35% of vaginal births include episiotomy. Those in favour believe that episiotomy should be used to prevent perineal trauma and to prevent pelvic floor relaxation and the consequences that follow. However, the hypothesis has not been proven. A recent review of 986(!) English-language articles from 1950 to 2004 reached a different conclusion “evidence does not support maternal benefits traditionally ascribed to routine episiotomy. In fact, outcomes with episiotomy can be considered worse since some proportion of women who would have had lesser injury, instead had a surgical incision.”

JAMA 2005;293:2141–8



PHARMAC welcomes debate

As an agency responsible for allocating New Zealand taxpayer funding, PHARMAC should be open to scrutiny (<http://www.nzma.org.nz/journal/118-1217/1544/>). Criticism can be uncomfortable, but things need to be said, and the NZMJ's 'Special Series' does so.

The *Journal* has an important role in healthcare in New Zealand, and we appreciate the *Journal's* efforts to promote local content.¹ Debate about PHARMAC's operations forms a necessary part of that content. We will seek to respond in order to maintain a balanced and constructive debate, because we believe that ultimately such debate can enhance healthcare in New Zealand.

From our discussions with the *Journal's* editors we understood that the 'Special Series' would include a series of ten articles highlighting how PHARMAC adversely affects population health through restricting the availability of certain medications.² We were also told that the *Journal* intended to allow PHARMAC the right of reply, but as a single response to all the articles and one-off at the end of the series. We thought this unfair.

In the past the *Journal* has provided PHARMAC with the chance to respond to a viewpoint article³ in a timely manner,⁴ and we have appreciated this. We felt disappointed not to be given a similar opportunity to respond in a similar way to the articles making recent direct criticisms.^{2,5-7}

We are happy to debate funding issues in the *Journal*, particularly when all the evidence is presented. We will work to keep the debates informative and interesting, so that the sector has a deeper understanding of the roles and practices of PHARMAC—and increase the reading of other important issues that the *Journal* raises.^{8,9}

Conflict of interest: Scott Metcalfe is externally contracted to work with PHARMAC for public health advice. Peter Moodie and Wayne McNee declare no conflicts.

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Bupropion, public funding, and smoking cessation

Clearly Holt et al have a different view from that of PHARMAC on whether bupropion should be publicly funded in New Zealand as a smoking cessation adjunct.^{1,2} What is not clear is why Holt et al conclude that the current PHARMAC position not to fund bupropion “seriously questions the Ministry of Health’s commitment to smoking cessation and the health of disadvantaged groups in New Zealand, particularly Maori.”¹

In fact, the Ministry and wider health sector commitment to the health of disadvantaged groups,³ particularly Maori,⁴ is absolutely clear. This includes making tobacco control a priority and the funding and successful delivery of a range of smoking cessation initiatives. Many of these initiatives are targeted to Maori and disadvantaged groups. Currently, there is around \$6.4 million for smoking cessation services targeted to Maori, and \$1.5 million for Maori smokefree promotion. Nicotine replacement therapy (NRT) is an integral part of many of these initiatives, in keeping with its identification in the NHC guidelines as the appropriate first-line therapy.⁵

In its response, PHARMAC cites reasons for its current position not to fund bupropion, including the large difference in cost-effectiveness compared with nortriptyline, an equally effective product according to the current evidence.² PHARMAC argues that, perversely, funding bupropion would have adversely affected the health of New Zealanders by restricting the availability of other medicines with greater health gains for the same spending.² To support their position, Holt et al cite three of the eight principles used to determine the actions in the Ministry of Health’s five-year plan for tobacco control; effectiveness, reach, and appropriate use of targeting.⁶ They overlook the other principles with that also need to be considered. One of these is cost-effectiveness, which is highly germane to this discussion.

In addition, the principle of “appropriate use of targeting” needs clarification. This principle is described as “maximising the benefits of targeted interventions and minimising potential adverse effects.”⁶ This refers not to minimising the potential adverse effects of therapeutic interventions, but rather to minimising the potential downsides of targeting, described as “a potential stigmatising effect and for middle-income groups to miss out.”⁶

Tobacco control is afforded a high priority in New Zealand, not least because there is major potential for improving health and reducing health inequalities.⁷ Smoking cessation remains an integral part of tobacco control. Within the funding allocated for smoking cessation, it is important to ensure the prudent use of public money by funding interventions that are effective, cost-effective and acceptable. This is, of course, work in progress and the recently-published research by Holt et al⁸ will help to inform this work. The Ministry looks forward to working with the researchers, PHARMAC and others towards a common objective of further reducing the serious impact of tobacco on health and health inequalities in New Zealand.

Competing interests: Ashley Bloomfield is a full-time Ministry of Health employee and was involved in the development of the 1999 and revised 2002 National Health Committee smoking cessation guidelines.

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Alcohol drinking guideline

The Nutrition Taskforce, which I chaired, agonised over recommendations for alcohol drinking.

After prolonged consideration, we agreed to the statement “If you drink alcohol, do so in moderation” with intake recommendation to limit alcohol intake to (or less than) 20 grams per day for women and 30 grams per day for men.¹

The statement has remained the same in subsequent revisions, although there has been some variation in intake recommendations (up to 40 grams per day). The variation in the base statement that I would now wish is the addition of the words “preferably with food”.

In a recent paper published in your *Journal*,² the authors calculate (from undeclared data, much reasoning, and the use of accepted analytical methodology) very precise conclusions purporting to accurately represent *The burden of death, disease, and disability due to alcohol in New Zealand*.

For many, but not all of the listed “alcohol related conditions included in the study”, there is a body of literature supporting benefits with moderate intakes. These benefits risk change to harm after an intake threshold is exceeded. Others conditions have no discernible alcohol benefit at moderate intakes and in others there are marginal, difficult to quantify adverse effect of low to moderate intakes. There are thus two populations which need separate analysis.

A provocative conclusion of this paper is “there are no health benefits before middle age”. In fact, it is likely that the demonstrated benefits may occur earlier in life. Most degenerative diseases such as coronary artery disease become manifest in middle or old age after a long incubation. The initiation of deterioration is due to multiple factors including adverse life styles. Surrogate markers may be present long before clinical disease is evident and provide an opportunity for an early insight into the evolution of risk factors and their relationship to alcohol intake.

There is a substantial literature directed to the effects of alcohol on disease risk markers. Most, but not all, suggest moderate alcohol intake is beneficial. I list a small sample³⁻⁶ A number of these studies include some younger adults but few focus specifically on moderate young adult drinkers.

Younger drinkers are usually the research target of studies of binge drinking and overall high alcohol intake.⁷ High alcohol intakes, especially if ingested rapidly (binging), is hazardous with risk of serious adverse social and long-term health consequences. The intake threshold for adverse events is generally considered to be about 60 grams of alcohol in 1 day, but for some people it may be lower. There are two results of inappropriate alcohol use; unsafe social behaviours (particularly in the young), and medical problems with loss of life at an older age. The benefits of moderate drink apply to all age groups.

I believe that the base alcohol guideline recommended by the Nutrition Taskforce is validated. The guideline would be strengthened by the addition of “preferably with



food”, a concept supported by published evidence^{8,9} and offers a pleasurable approach to foster a culture of moderate drinking for those who choose to drink alcohol.

Clifford Tasman-Jones
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Missed adult onset coeliac disease; the saga continues

One would have thought that the Canterbury study¹ and Mark Lane's accompanying editorial² (both in the 20 February 2004 issue of the *Journal*) should have dispelled the notion that coeliac disease is rare in New Zealand. In fact, the prevalence here is reported to be one of the highest (1:83) in the Western World.

However it seems that there still is little awareness of the *adult* onset coeliac disease. We continue to come across these patients who have been misdiagnosed as either 'refractory iron deficiency anaemia' or 'irritable bowel syndrome'. One patient was even labelled as a 'hypochondriac'. And a couple of patients were diagnosed 30 to 40 years after the onset of their symptoms!

Our experience is that the GPs are not the only group at fault here. We have observed the same lack of awareness even among the hospital staff. It is not uncommon to come across patients with chronic refractory iron deficiency anaemia who have been referred to the endoscopy department and scoped 'top and bottom' but no duodenal biopsies taken to rule out coeliac disease. Although the macroscopic features of coeliac disease on endoscopy are well known, none are pathognomonic, and biopsies are mandatory. Dual pathology in patients scoped for iron deficiency anaemia are also not uncommon³ and we think it is a good practice that, in high prevalence areas like New Zealand, duodenal biopsies should be taken as a routine in such cases.⁴

Also, before labelling iron deficiency anaemia as 'menstrual related' in young and middle-aged females, a simple serological screening test for coeliac disease should be performed.

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The risk of metachronus (asynchronous) contralateral torsion following perinatal torsion

The risk of asynchronous testicular torsion occurring following unilateral perinatal torsion has been generally assumed to be extremely low such that most surgeons do not perform pexy (fixation) of the contralateral testis, or pex the contralateral testis many months later.^{1,2}

We would like to draw attention to our recent experience where we have been made aware of two cases of asynchronous torsion occurring within the first 15 months of life.

The first case involved a full-term boy with right-sided torsion evident at birth. No scrotal exploration was performed at that stage. Ultrasonography performed in the neonatal period showed a normal left testis. Elective fixation of that testis was planned at 16 months of age but on surgical exploration the left testis was found to be completely atrophic.

The second case infant was diagnosed with left perinatal torsion, with swelling and induration of the scrotum noted at birth. On review at 3 months, the left testis had largely disappeared, and the right testis felt normal. At 6 months, he was admitted for fixation of the contralateral testis electively. The night before his elective surgery his mother noted the right scrotum had become swollen and tender. At exploration the next morning the right testis had undergone haemorrhagic infarction. Excision of the left testicular remnant found it to be atrophic.

These cases represent asynchronous torsion of the testis following unilateral perinatal torsion where the contralateral torsion occurred in the first year or so of life. Both patients were consequently rendered anorchic.

This experience suggests that:

- It may be prudent to ‘pex’ the contralateral testis following unilateral neonatal torsion in all cases; and
- That the surgery best be undertaken within the first few weeks of life.

The fact that only fifteen similar cases have been recorded previously in the literature raises the question as to whether this is an under-reported occurrence.

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John Milne Loughlin

10 February 1930–9 September 2004

Originally from Auckland, where he attended Auckland Grammar, John Loughlin received his medical training at Otago after initially qualifying in Pharmacy and acquiring his University Entrance at night school.



He supported himself and young family during the vacation periods by organising cleaning and painting contracts as well as pharmacy dispensing after hours.

This diligence and initiative, energy and capacity for hard work was to distinguish his ophthalmology.

He trained at the Institute of Ophthalmology in London, acquiring the Diploma before returning to his family in New Zealand. He often expressed a regret that he had accepted the advice given not to proceed to the Fellowship.

After a short period in Nelson, in 1968 he joined David Sabiston in practice in Napier and an appointment to the Hawkes Bay Hospital Board which proved to last for over 30 years.

He was instrumental in leading a fundraising effort in 1973 that founded the Hawkes Bay Ophthalmic Trust which proved instrumental in introducing new technology such as operating microscopes, argon and YAG lasers, and ultrasound into the hospital.

With an active concern for those in need, he was a founding trustee for the Hawkes Bay Trust for the Elderly providing minivan transport to medical appointments and for rest home residents. He also guided a local Lions Club in funding children's spectacles before the Under 8 Spectacle grant was established.

In the 80s he developed a lifestyle block, first as a citrus orchard and then as Waimarama Estate Winery succeeding to craft a Bordeaux-style wine with a remarkable Gold Medal 1991 vintage.

On retiring from his hospital appointment and from surgery, he maintained his private medical ophthalmic practice until ill health in late 2003. He is survived by his wife, Audrey, and three of his four children.

This obituary was written by Dr John Beaumont, a friend and colleague of Dr John Loughlin, and is reprinted with permission from the January 2005 issue of the *Ophthalmologists' Exchange* (Royal Australian & New Zealand College of Ophthalmologists) to whom we are grateful.



David Stewart Hogg

David Hogg died in Wellington at the end of December 2002. He was born in Glasgow, Scotland in 1927 and educated in that city, graduating in medicine from the University of Glasgow in 1949.

As a boy, and in early adult life, he was an excellent sportsman. He excelled in soccer, golf, tennis, and badminton, gaining a University Blue in soccer.

After qualifying, he did various jobs at hospitals in the Glasgow area, and followed these with GP locums until he became a partner in a group in Turriff, a rural town in north-east Scotland. There he met and married Hazel Beaddie, a local school teacher. She converted him to cards—initially solo whist and later bridge. They were both talented players and played competitive bridge up to Scottish Championship level.

They migrated to New Zealand in 1964 and David joined a practice in Tawa. He was an excellent doctor admired by patients and colleagues in both New Zealand and Scotland. David was a very private, quiet, and genteel gentleman, never seen to lose his temper despite provocation. The Hogg's social life was largely bridge-associated.

In 1979, David had the first of many cardiac events and this forced him out of general practice to work in the Health Department where he was highly regarded. Even this became too much and he retired because of ill health after a few years.

Life has been difficult for David over the past years and he bore his disappointment and the frustration, and the pain and discomfort that was his lot, with admirable, non-complaining fortitude and dignity.

He was survived by Hazel and two adopted daughters, both of whom live in the UK. Hazel died suddenly in her sleep in October 2004.

Dr A Leslie Florence, a long-time partner of Dr Hogg, contributed this obituary.



Illustrated clinical anatomy

Peter Abrahams, John Craven, John Lumley. Published by [Hodder Education](http://www.hoddereducation.co.uk), 2005. Contains 390 pages and over 900 illustrations. Price GBP 24.99

Anatomy is a vast and timeless subject. The length and detail of an anatomy book is determined by the authors with a target audience in mind. On the cover, it is stated that the book is 'by clinicians for clinicians'. But, reading the Preface, it seems that this book is written primarily for medical students.

The British authors have followed the American Association of Clinical Anatomists' core curriculum as a guide to what is 'core'. Chapters for each anatomical region have sections of clinically relevant information highlighted neatly in blue. Reading through my own area of interest, I was disappointed to see the example of shoulder dislocation to be a backstroke swimmer colliding with the end of the pool—an uncommon cause. Perhaps the infamous O'Driscoll incident (British Lions rugby tour of New Zealand in 2005) will remind the authors of a more common mechanism.

The book is beautifully laid out, with clear illustrations. Some core information is well summarised in chart form—for example muscle attachments, innervations, and functions. The clinical sections provide the link between structure, function, and disease. In doing so, it fulfils the criteria for the modern style of anatomical teaching adopted by many medical schools. A Questions section would give the book a reassuringly complete feel for those studying for an undergraduate exam.

This book will not satisfy everyone. No anatomy text can. Even when the information provided is well structured and presented, the issue is what is left out. A text of less than 400 spaced-out pages obviously leaves out a lot of detail; for example, the nervous system is not included. Indeed, the debate on the detail required for undergraduates will continue, and is not the subject of this review.

I believe this is a good book, and if medical students knew everything in this book, their anatomy foundations would be sound.

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A Stereotaxic Atlas of the New Zealand Rabbit's Brain

Ivan Urban, Philippe Richard. Published by [Charles C Thomas Publisher Ltd](#), 1972.
ISBN: 0398024316. Contains 92 pages. Price \$US27.95

This atlas of the rabbit brain uses an updated technique to more reliably provide stereotaxic coordinates for the rabbit brain. Due to rabbits having ears that are oriented quite differently to other species, a modification of the head fixation technique has been used which consists of using specially modified ear bars. This allows rabbits to be adapted to a suitably sized stereotaxic apparatus, and provides a more reliable and consistent head fixation method.

The atlas shows a series of coronal sections of the rabbit brain (averaged from 1 year old rabbits) at 0.5mm intervals—the sections are stained with a histological Nissl stain to show the location of brain nuclei on one side of the page and a corresponding drawing identifying and labelling the principal nuclei and fibre tracts on the other side of the page. The stereotaxic coordinates are prominently displayed on the axes. A list of abbreviations of the structures labelled in the atlas appears before the figures.

This atlas will be useful for neurobiologists and neurophysiologists studying the rabbit brain, but they should bear in mind that they will need the modified apparatus for accuracy. The atlas illustrations are clear and easy to follow, and as the atlas is relatively inexpensive, it should prove to be a popular choice for those in the field.

The only criticism I would make is that the atlas does not extend more caudally to include lower medulla and spinal cord, and does not include sagittal sections for orientation.

Henry Waldvogel

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