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This Issue in the Journal

How do intoxicated patients impact staff in the emergency department? An exploratory study

Fiona Imlach Gunasekara, Shaun Butler, Taisia Cech, Elizabeth Curtis, Michael Douglas, Lynda Emmerson, Rachel Greenwood, Sara Huse, Julia Jonggowisastro, Camilla Lees, Yang Li, Daniel McConnell, Andreea Mogos, Nur I M Azmy, Scotty Newman, Kirstie O'Donnell

Despite a high burden of intoxicated patients in emergency departments in New Zealand, there has been little research into the impact of these patients on staff or on how staff view these patients. We surveyed and interviewed staff at Wellington Regional Hospital Emergency Department for their experiences. We found that staff commonly encountered verbal assault from intoxicated patients, and staff reported increased workload and increased overall waiting times with intoxicated patients. Staff also observed that intoxicated patients impacted negatively on the care and safety of other patients in the emergency department. Alcohol-related presentations may impair the quality of care for all patients in the emergency department and reduce the safety and wellbeing of staff.

Off-label use of atypical antipsychotic medications in Canterbury, New Zealand

Erik Monasterio, Andrew McKean

The purpose of the current study is to determine how often the new generation of antipsychotic medications (designed and licensed to treat illnesses such as schizophrenia and manic depressive illness) are used to treat other, non-psychotic related problems. This is known as “off-label” prescribing and while it is not illegal, the use of medications in this way is seldom backed by scientific evidence of efficacy and safety. We surveyed 70% of all the psychiatrist's in the Canterbury region and found to 96% of them prescribed these medications “off-label”. More than 50% of psychiatrists did so at least once a week and the most common problems for which they were prescribed were anxiety, sleeplessness, post-traumatic stress disorder and dementia. The findings suggest that there is an over reliance on antipsychotic medications to treat common symptoms of psychological distress.

Five-year follow-up of an acute psychiatric admission cohort in Auckland, New Zealand

Amanda Wheeler, Stuart Moyle, Carol Jansen, Elizabeth Robinson, Jane Vanderpyl

This study reports the 5-year readmission rates of a cohort of mental health patients admitted to an acute inpatient service in 2000. Almost 40% of the group had no further acute admissions for psychiatric reasons anywhere in New Zealand during the 5-years after discharge. The two factors associated with readmission were having a history of previous admissions or being of Māori ethnicity. One-third of the group had

three or more admissions in the 5 years following index discharge with longer bed stays. The findings highlight the need for reliable community-based data to enable exploration of the use of community services following acute discharge, especially in preventing readmission, and the impact of acute service alternatives to inpatient units for times of crisis. This is particularly important given the negative and distressing experience reported by people admitted to acute psychiatric inpatient units.

Quetiapine for the treatment of behavioural and psychological symptoms of dementia (BPSD): a meta-analysis of randomised placebo-controlled trials

Gary Cheung, Janli Stapelberg

The efficacy of quetiapine for the treatment of behavioural and psychological symptoms of dementia (BPSD) was examined using six sets of international data published on this issue. This analysis found a small magnitude of improvement of these symptoms but the observable change by clinicians is questionable.

Narcolepsy in New Zealand: pathway to diagnosis and effect on quality of life

Angela J Campbell, T Leigh Signal, Karyn M O'Keeffe, Jessie P Bakker

Narcolepsy is a neurological sleep disorder characterised by excessive daytime sleepiness, with other symptoms including sudden episodes of muscle weakness, disrupted sleep, sleep paralysis and hallucinations occurring during sleep onset or while awakening. We aimed to survey patients diagnosed with narcolepsy in New Zealand in order to gather data regarding their pathway to diagnosis, treatment, and general quality of life. Responses were received from 54 patients, on average 55 years old. Diagnosis of narcolepsy took place at an average age of 33, despite symptoms first appearing by age 21. The respondents reported greater daytime sleepiness and poorer health-related quality of life compared with the general New Zealand population, and we also identified a number of inconsistencies between the diagnostic/treatment pathway of these patients and internationally published guidelines.

Patterns of prescription drug misuse presenting to provincial drug clinics

Geoffrey Robinson, Graeme Judson, Richard Loan, Timothy Bevin, Patrick O'Connor

In the absence of imported heroin into NZ, prescription opiate drugs have become the street supply. Prescription opiates have been increasingly misused in the USA. In our region, this study found morphine and methadone were the commonest street opiates being used. The costs to users of these drugs are considerable, but may be reducing. Prescription opiate misuse is a complex Public Health issue which requires multiple responses to contain.

Controlled intoxication: the self-monitoring of excessive alcohol use within a New Zealand tertiary student sample

Brett McEwan, David Swain, Maxine Campbell

The majority of drinkers in the study who consumed alcohol with the intention of getting intoxicated, typically drank to a predetermined level of intoxication, and maintained that level by monitoring a range of drinking effects—this behaviour was termed ‘controlled intoxication’. Future harm-minimisation strategies could be developed that encourage heavy-drinkers to adopt ‘safer’ drinking-effect signals as indicators to slow down or stop their drinking.

Alcohol’s harm to others: self-reports from a representative sample of New Zealanders

Sally Casswell, Jessica F Harding, Ru Q You, Taisia Huckle

A survey of New Zealand households asked about the experience of heavy drinkers in people’s lives. One in four reported having a heavy drinker in their life and a wide range of negative experiences were reported by interviewees to have been associated with this exposure.

The benefits and harms of alcohol use in New Zealand: what politicians might consider (viewpoint article)

Nick Wilson, Fiona Imlach Gunasekara, George Thomson

The New Zealand Government is currently considering ways to reduce alcohol-related harm, following on from a detailed report by the Law Commission. To inform discussions we briefly summarise the benefits and harms of alcohol use in this country. The most substantive benefits to society are probably pleasure to users and economic benefits (largely to industry). The most substantive harms are probably those to mental and physical health, harm to society (e.g., from crime) and adverse net economic impacts. Overall the picture is suggestive that New Zealand society would be likely to achieve a large net benefit from reducing heavy and binge drinking, and shifting alcohol consumption towards a pattern of smaller amounts. The substantial harm to non-users is a key argument for democratic governments to use regulations and taxes to minimise harm from alcohol.

The impact of alcohol-related presentations in the emergency department and the wider policy debate

Taisia Huckle, Sally Casswell, Sarah Greenaway

The recent Law Commission review of the laws and policies that govern the sale and supply of liquor in New Zealand has sparked much public debate.

The Gunasekara et al study, in this issue of the *NZMJ*,¹ while exploratory in nature, adds to this debate in three important ways. Firstly, by providing additional evidence about the externalities of alcohol use; secondly by highlighting the need for effective policy implementation; and thirdly by reinforcing the need for the systematic collection of alcohol-related data in the Emergency Department (ED) setting nationally.

The externalities of alcohol use—that is, the harm that results from other peoples drinking—is relevant to the policy debate.² Gunasekara et al¹ report that alcohol-related presentations increased waiting times in the ED and negatively impacted on the quality of service delivery to non-alcohol-affected patients. Furthermore, almost half of the nurses reported being physically assaulted by alcohol-affected patients while at work.¹ It is not only alcohol-affected patients who experience consequences due their own drinking; the staff, other patients and hospital resources are also negatively affected in the ED setting.

It is not surprising that the burden of alcohol in the ED is high; in New Zealand 50% of all alcohol consumed by those aged 14–65 years is done so in heavier drinking occasions (defined as 8 or more drinks for males and 6 or more drinks for females).³ Among young people (12–19 years) 75% of all alcohol consumed is done so in heavier drinking occasions.⁴ Gunasekara et al¹ report that 12–14 year olds have presented to the ED after drinking alcohol, only since the lowering of the purchase age.

Gunasekara et al¹ link the problems of alcohol in the ED to the wider social and policy environment. They state “the burden of alcohol on the health system will only be reduced effectively and sustainably by policy changes that lead to a reduction in heavier alcohol consumption in wider society”. They point to the legislative changes suggested in the Law Commission’s review of the liquor laws as potential means for reducing alcohol-related presentations, and their associated burden, in the ED. However, some of the most effective policies recommended by the Law Commission to reduce alcohol-related harm have not been adopted in the subsequent Alcohol Reform Bill which is currently before Parliament.

Key recommendations that are currently not included are: returning the purchase age to 20 years; restricting alcohol marketing effectively via legislation; and raising the price of alcohol through taxation. This is despite considerable support for such moves.

Results from social surveys indicate that there is public support for more controls on alcohol. In April/May of 2011, 444 respondents aged 16–65 years were interviewed using a rigorous sampling frame and data collection methods (as used in SHORE/Whariki general population surveys e.g. Huckle et al 2010⁵).

Seventy-eight percent of respondents supported the return of the minimum purchase age to 20 years (while 42% supported the split age). Sixty-six percent supported restrictions on numbers of alcohol outlets and 59% supported restrictions on alcohol marketing. An increase in the price of alcohol was supported by 30%.

Another survey from New Zealand found similar results.⁶ Hoek and Gendall (2006) (cited in Kypri et al⁶) found that 75% of respondents supported returning the minimum purchase to 20 years and 59% supported increasing the tax on ready-to-drink (RTD) mixed spirits—otherwise known as 'alcopops'.

Gunasekara et al is a useful study as it documents the impact of alcohol in the ED from the perspective of the staff and this is an under-researched area. There have also been some quantitative studies of the involvement of alcohol in presentations to ED.^{7–9} However there is no systematic collection of alcohol-involvement in ED presentations nationally.

A national project is underway with DHB's to develop and implement data collection and analysis of alcohol related harm data including alcohol-related presentations to EDs. A nationally consistent and systematic approach will contribute to the accurate identification of the burden of alcohol on New Zealand society.

Competing interests: None.

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Off-label use of quetiapine in New Zealand—a cause for concern?

Paul Glue, Chris Gale

Off-label prescribing is common in psychiatry and often reflects a pragmatic clinical approach to managing refractory symptoms in complex patients. Although often not supported by clinical trial data sufficient to gain regulatory approval for use, there will be supporting data in the form of open trials, case reports, or case series. For rare or treatment-resistant patients, formal controlled clinical trials may not be feasible. Furthermore, some authors have seen off-label usage as important in discovering new indications.¹

In this issue, Monasterio and McKean² have presented survey data from a cohort of Canterbury psychiatrists on frequency and reasons for off-label use of atypical antipsychotic drugs (AAPs). Their main finding is that survey respondents commonly report off-label use of AAPs, and this is almost entirely accounted for by use of quetiapine, for a range of symptoms/indications. Focussing on quetiapine, the drug most commonly identified as being used off-label, how should their findings be interpreted?

The data presented are difficult to evaluate because of study design issues. In contrast to an earlier study³ that examined off-label quetiapine use, the Canterbury study is impressionistic rather than quantitative, and does not collect data on dose, frequency of administration, and patient location (e.g. in- vs out-patient). It is also not clear whether the authors provided a checklist of possible symptoms/indications or these were derived from the responses of survey respondents. These data are important to understand patterns of quetiapine use. For example, in an earlier off-label use survey,³ the majority of patients receiving off-label quetiapine were dosed on an as-needed basis, with 80% receiving doses of 25–50 mg, for agitation, anxiety and insomnia. Such information is important to interpret the significance of the Canterbury survey findings.

In the Canterbury survey, the three most common symptoms/indications for off-label quetiapine included anxiety, sedation, and post-traumatic stress disorder (PTSD), and are discussed further below. The next most frequent symptoms/indications for which quetiapine is reported to be used are of some concern, in terms of a lack of evidence base (e.g. augmentation of another antipsychotic) or potential for harm to patients (e.g. treatment of symptoms of dementia).

Focussing on the most common reported reasons for off-label use (anxiety and insomnia), we disagree with the authors' unduly negative assessment of the quality of published data to support any of these uses. Effects of quetiapine on Hamilton Anxiety Scale scores and individual anxiety scale items in depressed patients with comorbid anxiety are indicative of a broad anxiolytic action with a rapid onset of effect.⁴

Other reviews on the efficacy of quetiapine in generalized anxiety disorder (GAD) are positive,^{4,5} as was a recent FDA assessment of efficacy endpoints.⁶ Studies demonstrating efficacy in anxiety disorders besides GAD (e.g. PTSD) are also positive, however data are more limited.⁴ The effects of quetiapine on sleep have been studied using polysomnography, and increases in total sleep time and sleep efficiency have been reported in primary insomnia⁷ and mixed insomnias,⁸ with no evidence of tolerance development. The rapid onset of sedative and anxiolytic effects is consistent with quetiapine's antihistaminic and antiadrenergic pharmacology.⁴

If quetiapine were not to be used off-label for anxiety and insomnia, the range of alternative approved anxiolytics and hypnotics in New Zealand is limited. For anxiety, the only approved drugs with a rapid onset of action are benzodiazepines. Sedating antidepressants, older sedating antihistamines and antiadrenergic drugs (e.g. clonidine, prazosin) are also available but not approved for this symptom/indication. (Non-sedating antidepressants and buspirone are effective anxiolytics, however with a much slower onset of action). For insomnia, only benzodiazepines and zopiclone are approved (sedating antihistamines and antidepressants are also available however their use would also be off-label).

If doctors decide to use quetiapine off-label for symptoms of anxiety or insomnia, what are the risks? The most common side effects appear to be metabolic, and occur even at relatively low daily doses.⁹ Other safety issues include a dose-related increase in sudden cardiac death rate in the short term,¹⁰ and tardive dyskinesia with long-term usage. Concerns about longer-term safety risks were influential in quetiapine's non-approval for anxiety and depression indications at a recent FDA advisory panel.¹¹ However concerns were not raised about short-term or intermittent use with regard to these indications.

Abuse potential has also been identified, however most reports appear to be in forensic settings, in polysubstance abusers.¹² In relative terms, abuse and dependence liability appears to be much greater for benzodiazepines (the main alternative anxiolytic/hypnotic drugs) than for quetiapine.

Ultimately, any clinical decision to use quetiapine off-label has to include an assessment of risks and benefits. Based on published data, there is a solid evidence base to support short term or intermittent use of low doses (up to 150 mg/day) in symptomatic anxiety (e.g. in the context of a major depressive illness), in GAD, and for doses of 25–50 mg/day in insomnia.

Short-term use in other anxiety disorders is also supported by published data. This type of off-label use pattern would be consistent with a previous report from a more methodologically rigorous survey.³ The use of quetiapine in these circumstances is not risk-free, however the reduced potential for abuse and dependence over benzodiazepines, the main alternative drug class with a rapid onset of action, is an important consideration.

The case for using quetiapine long term or at higher doses in any of the above symptoms/indications is much less clear, with few published data to support such use. Any decision to prescribe in this way would involve a risk/benefit decision for an individual patient, and would at minimum require demonstrated intolerance to or

failure of first line medications, along with trials of psychological and/or behavioural treatments.

Medsafe provides a useful guidance on professional and ethical considerations for doctors planning to use approved drugs for unapproved indications,¹³ including advice on when and how to use informed consent. The guidance states, “*For an unapproved medicine or unapproved use, the consumer should be advised of the unapproved status. The consumer should also be advised of the degree and standard of the support for the use of the medicine...*”. The guidance recommends use of written informed consent if the treatment has minimal supporting evidence, if there is equivocal evidence for safety or efficacy, or if the treatment is experimental.

In conclusion, the Canterbury survey² identifies that off-label quetiapine use is common. There is a solid evidence base to support its short term low dose use in anxiety and insomnia. As part of an ethical prescribing process, it is important for doctors to highlight to patients the quality of clinical evidence for the proposed off-label use of quetiapine, bearing in mind the Medsafe guidance.¹³

Competing interests: Professor Glue is currently on the Scientific Advisory Board of Demerx Pharmaceuticals, and has attended a scientific advisory board for Janssen. Dr Gale has been on speakers’ bureaux for Lilly and Janssen, and has had travel costs supported by Lilly.

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How do intoxicated patients impact staff in the emergency department? An exploratory study

Fiona Imlach Gunasekara, Shaun Butler, Taisia Cech, Elizabeth Curtis, Michael Douglas, Lynda Emmerson, Rachel Greenwood, Sara Huse, Julia Jonggowisastro, Camilla Lees, Yang Li, Daniel McConnell, Andreea Mogos, Nur I M Azmy, Scotty Newman, Kirstie O'Donnell

Abstract

Aim To investigate staff perceptions of the burden of alcohol-related presentations on emergency departments (ED) in New Zealand and the impact on staff of alcohol-related ED presentations.

Methods A survey of Wellington Regional Hospital ED staff was conducted using a written questionnaire to measure the impact of alcohol on: staff assault rates, perceived workload, quality of care, and staff mood. In addition, semi-structured interviews were conducted with six ED staff to further explore impacts of alcohol on ED, analysed using thematic analysis.

Results Forty-seven staff members responded to the questionnaire. Assault rates from alcohol-affected patients were high, particularly amongst nurses. These were mostly verbal assaults. Staff mood was negatively affected and perceptions of workload increased by alcohol-related presentations. Views on whether quality of care of intoxicated patients was affected were mixed although most reported a negative impact on other patients. Interviews confirmed the survey results, confirming the negative impacts of alcohol-related presentations on staff and on the treatment of both intoxicated and non-intoxicated patients.

Conclusion This small exploratory study found that alcohol-related presentations have a negative impact on ED staff workload and safety, and may compromise treatment of all patients. More research is needed to corroborate these findings and to investigate policies to reduce the impacts of alcohol-related presentations in the ED.

Alcohol law reform is currently on the political agenda, with the Law Commission having undertaken a comprehensive review of New Zealand's liquor laws in 2009–2010,¹ prompting the writing of a new *Alcohol Reform Bill*.

Several high-profile alcohol-related deaths in young adults have also increased public awareness of the detriments to a culture of binge drinking. Drinking patterns in the population have changed, with higher rates of drinking in young people, and more binge drinking, and this has impacted on hospital emergency departments (ED).¹ These changes have been linked to changes in alcohol policy.

New Zealand (NZ) lowered its alcohol minimum purchasing age from 20 to 18 years in 1999 and in the subsequent year there was a significant increase in the number of ED presentations in people aged under 20 years, increasing the burden on hospital staff and ED resources.¹ Increases in the number of excess breath alcohol

prosecutions and alcohol-related road accidents in the 15-19 year old age group also occurred in the years following the lowering of the purchase age.²

Previous studies have explored the impact of alcohol on EDs, finding alcohol to be a major factor in unintentional injuries, and causing higher costs to the health system for treatment than from injured patients where alcohol was not involved.³⁻⁶ Alcohol-related presentations are the main causes of violence and acute behavioural disturbance in the ED.⁷

Staff exposure to violence from such presentations is an inevitable event, given their high frequency. Alcohol-related ED presentations represent not only a fiscal burden on a constrained health sector, but a potential source of harm for staff working in the ED.

Comprehensive studies assessing the impact of alcohol on EDs in New Zealand are lacking, despite high rates of binge drinking and drinking among minors⁸ and anecdotal reports of a high level of burden on EDs from alcohol-related presentations.⁹ A study of Auckland Hospital ED in 2000 found that alcohol was involved in 30% of all injury presentations¹⁰ and in Hawke's Bay, alcohol was a factor in 18% of all injury presentations to the ED, but 67% of injuries presenting between 12am and 6am.¹¹

In 2008/09, staff from Canterbury District Health Board reported 670 physical assaults and hundreds of verbal assaults, often provoked by alcohol.¹² A survey of 550 first year graduate nurses in 2004 found many experienced violence and abuse from patients at work.¹³ Despite high levels of violence and alcohol-related presentations in ED, investigation into the impact of these presentations on ED staff, and how they are perceived by staff, has not been performed in NZ. We undertook an exploratory study to assess the impact of alcohol-related presentations on ED services, as reported by ED staff, using a survey of staff attitudes and perceptions, and in-depth qualitative staff interviews at Wellington Regional Hospital ED.

Methods

Ethics—Ethics approval for the study was given by the head of the Department of Public Health, Wellington School of Medicine and Health Sciences with the authority of the University of Otago Human Ethics Committee.

Written survey—An initial meeting was held with the head of the Wellington Regional Hospital ED (Dr Paul Quigley) to discuss problems faced by ED staff when dealing with intoxicated patients. A written survey was subsequently developed for staff members working in the ED to complete. The questionnaire asked about the effects of alcohol-related presentations on other patients, waiting times, work load, quality of care to intoxicated patients and staff mood; levels of assault experienced from and staff attitudes towards intoxicated patients; and whether suitable measures were in place to manage intoxicated patients.

Seventy surveys were distributed and 47 responses were received. Numbers working in the ED are fluid but it was estimated that around 70-75 people were working in the ED around the time of the survey. Surveys, along with information sheets and consent forms, were placed in the ED staff room with a returns box. Surveys were also handed out directly to staff members on four occasions, including to ambulance officers at Thorndon Ambulance Station. Participants were given approximately 2 weeks to complete the survey before they were collected for analysis. Survey responses were analysed using Microsoft Excel.

Staff interviews—Face-to-face semi-structured interviews with ED staff were undertaken using an interview template created by the study authors, including questions expanding on the written questionnaire, such as: How do you feel the ED services are impacted by alcohol-related presentations?

In the waiting room, what impact do intoxicated patients have on other patients? Do you feel that the quality of care given to patients is altered if they present with alcohol-related presentations? What measures do you know of that are in place to reduce the impact of alcohol on ED services? What is your personal attitude towards patients with alcohol-related presentations?

A purposive sampling technique was used to select two representatives from each of three professional groups working in ED: nurses, doctors, and radiographers. Two of the authors (JJ, YL) approached staff directly in ED. Interviews were conducted in a private room so that responses could be kept confidential and audio-taped. All participants were given an information sheet and provided written consent. Recorded interviews were analysed using thematic analysis.¹⁴

Results

Written survey—47 responses to the written survey were received from 23 nurses, 8 doctors, 8 ambulance officers, 3 ED radiographers, and 5 receptionists (see Table 1).

Table 1. Characteristics of respondents to written survey

Variables	Ambulance service	Nurse	Doctor	Radiographer	Reception	Total (N)
Male	5	8	2	1	1	17
Female	3	12	3	2	4	24
NA*	0	3	3	0	0	6
Age (years)						
<25	2	1	2	2	0	7
25–30	2	9	4	1	3	19
31–40	2	4	0	0	1	7
41–50	2	4	1	0	0	7
50+	0	6	0	0	1	7
NA*	0	0	1	0	0	1
Years worked in NZ ED						
<1	1	8	6	1	1	17
1–5	3	5	1	2	2	13
5–10	2	6	0	0	1	9
10+	2	5	1	0	1	9
Total	8	23	8	3	5	47

*NA = No answer.

Assault—Overall, half of the staff report ever being assaulted by an intoxicated patient while at work, with nurses having the highest exposure (see Table 2).

Table 2. ED staff reports of assault by intoxicated patients

Work category of ED staff	Reports of assault by intoxicated patients	Verbal assaults	Physical assaults
	(N reporting assault/Total N)		
Ambulance service	4/8	4/8	3/8
Nurse	15/23	12/23	10/23
Doctor	3/8	3/8	0/8
Radiographer	0/2	0/2	0/2
Reception	2/5	2/5	0/5
Total	24/47	21/47	13/47

Verbal assault is the most prevalent form of abuse experienced (by 21 respondents). Physical assaults are also commonly reported by ambulance officers and nurses (not

other staff groups) but no sexual assaults are reported. Of these respondents who reported assault, three experienced this at least once a week, eleven on a monthly basis and the remainder once a year or less frequently.

Impact on workload and quality of care—Almost all participants (45) report that alcohol-related presentations increase staff workload. Many participants also report that alcohol-related presentations increase overall waiting times (37) and triage scores of the intoxicated patient (19), which is an indicator of the severity of a patient's condition assigned by a nurse when patients first enter the ED.

Half of participants report that the presence of alcohol does not impact on the quality of care given to the intoxicated patient but 19 report that an alcohol-related presentation receives decreased quality of care and a few anticipate an increased quality of care.

Impact on other patients and staff reactions—None of the participants record that alcohol related-presentations have a positive impact on other patients and the majority state that the impact is negative (two indicate a neutral effect).

More than half of staff surveyed (22) also record that alcohol-related presentations negatively affect staff mood and only two respondents (radiographers) reported a positive effect.

Measures in place to decrease alcohol-related presentations—Staff are divided in their opinion of how alcohol-related patients are managed within the department, with half stating that suitable measures are in place and half stating that there are no suitable measures.

In contrast, responses regarding measures to reduce alcohol-related patients presenting to the department in the first place are more stark, with most of the staff (40) feeling that no suitable measures are in place.

Staff interviews—Staff interviews found common themes of abuse from intoxicated patients, increased workload, mixed staff reactions to intoxicated patients, impaired quality of care and negative effect on other patients. Staff also commented on possible measures to reduce alcohol-related presentations in ED. Extracts from interviews are given as examples of common themes.

The first three themes demonstrate how intoxicated patients can negatively impact on ED staff, by creating an unsafe work environment, increasing workload, and for some staff on some occasions, causing negative emotions.

Abuse from intoxicated patients—Staff describe a range of abuse from intoxicated patients. As observed from the written questionnaires, verbal abuse is common, with physical abuse occurring less frequently and often perceived as low level.

“[These assaults are] usually pretty small, either spitting or the occasional thump on the arm when you're trying to assess them. Always verbal, with always profanities, obscenities coming at you, but you do develop thick skin” (*ED Registrar 1*)

The burden of intoxicated patients appears to be unequally distributed between staff members who are seen as able to cope with them and those that are not. This is noted as a strategy for dealing with intoxicated patients and rather than a problem by staff.

“...based on who I am, I pick these patients and I get asked to see these patients by other people who don't want to see them or can't see them” (*ED Registrar 1*)

Staff are able to request support in dealing with intoxicated patients. Support is frequently sought from other members of the clinical team, security staff, orderlies, and police if required. Being able to access this support contributes to a feeling of safety.

“I have no hesitation in calling police, just cos [because] they're drunk. If you assess someone and they're breathing, talking, yelling, and going to physically hurt you, then they're probably just as safe in a police cell as they are waiting in the emergency department. As much as they have a right to be treated, we have a right to be safe in our work environment” (*Staff Nurse 1*)

Increased workload—Staff report that alcohol-related presentations greatly increase the workload in ED, particularly on Thursday, Friday, and Saturday nights, and during major sporting events or concerts.

“The big rugby matches, you can always guarantee that there's going to be an increase in problems, music concerts as well, there's going to be an increase in problems, usually purely related to alcohol” (*Staff Nurse 1*)

In addition to increasing the total number of presentations, staff describe alcohol-related presentations increasing the load on ED because they are harder to deal with, and take up more staff time.

“If you've got someone that's really pissed [drunk], they can cause problems while they're waiting, which means a lot of your time is wasted supervising them or even just keeping them in the back of your mind, so things do slow down” (*ED Registrar 2*)

Finally, the staff believe that intoxicated patients impact negatively on ED performance indicators such as the 6-hour rule (the target of keeping time taken from triage to discharge to less than 6 hours).

“Especially with this new 6-hour rule in place, it's impossible if you've got someone really intoxicated... often you can't assess them completely to be fully compos mentis, or if they have an injury, to be able to clear it completely, because they're drunk, so you have to wait until they are more clear with their responses... or even till they wake up and stop being a complete bastard to you, and that can take way longer than 6 hours” (*ED Registrar 2*)

Mixed staff reactions to intoxicated patients—The ED staff interviewed vary in their personal perceptions and responses to alcohol related presentations. Some ED staff do not see them any differently from normal patients.

“I not going to judge the [intoxicated] patient for why they're there because they could have been assaulted while they were intoxicated” (*Radiographer 1*)

Other staff have very negative views of intoxicated patients, seeing their ailments as self-inflicted, and an unnecessary burden on the ED and society in general.

“The drink drivers that come in and absolutely destroy people lives, and have absolutely no insight into what they've done at times, they are really hard to treat as well. Morally we have to be completely objective in our level of care, but it can be hard if you know that the person you are treating has killed two other people just because they did this drink driving thing. That can again cause increased frustration for staff” (*Staff Nurse 1*)

The next two themes demonstrate how intoxicated patients create difficulties in the treatment not only of themselves but also in the care and feelings of safety of other patients in the ED.

Impaired quality of care of intoxicated patients—The staff interviewed all believe that the care given to intoxicated patients is impaired, either because of ED staff's negative perceptions of intoxicated patients or because the patient's level of intoxication made it too hard to assess, treat or gain informed consent from them.

“The patients who are drunk and obnoxious and dangerous, often some of our staff will say, 'yes you're fine, get out of our department now'” (*Staff Nurse 2*)

Staff report that intoxicated patients often do not realise the extent of their injury or ailment and leave before being properly assessed or treated which may also impact negatively on their care. Staff also describe damage caused by intoxicated patients, that puts a strain on the facilities of the ED.

“One of our bathrooms has been smashed up probably three times in the last couple of months, and we get a lot of our beds hit and damaged, doors broken” (*ED Registrar 1*)

Negative effect on other patients—Staff observe that intoxicated patients are a major problem for non-intoxicated patients, frequently making them feel unsafe.

“Imagine your 80 year old grandmother sitting around with 10 drunken 20 year olds. The grandmother's not going to have a nice time. Or the mum sitting with her three year old child. They get scared, they get frustrated” (*Staff Nurse 2*)

Intoxicated patients can impact on the care received by non-intoxicated patients.

“You've got potentially worse people in the waiting room, and they cannot get into the department to be seen because of somebody who's pissed” (*ED Registrar 2*)

The final section elicited staff opinions on what could be done to minimise the problems they were experiencing related to intoxicated patients in the ED.

How to reduce the impact of alcohol-related presentations in ED—Staff believe that the drinking culture in New Zealand is a barrier to reducing alcohol abuse.

“The culture of drinking needs to be looked at. A lot of the time, people will wake up in EDs in the morning and go 'oh god, what's happened?' and have a laugh and walk out. They don't realise how bad it is” (*Staff Nurse 2*)

A variety of strategies to reduce the number of alcohol-related presentations in ED are suggested, particularly individual interventions.

“This department is looking at brief interventions in the area of making people acknowledge their drinking patterns and showing them that they are at risk” (*Staff Nurse 2*)

Other suggestions include increasing the legal age limit for drinking, having zero tolerance policy for drunk driving, and controlling the sale of alcohol.

“We're getting examples of 12–14 year olds drinking and ending up at ED. Those people have only been presenting to ED since the drinking age was lowered” (*Staff Nurse 1*)

“There seems to be a higher percentage of young females now drinking and drinking to dangerous levels, and there's a lot of research linking this to an increase in the sale of premixed RTDs [ready to drink alcoholic beverages]” (*Staff Nurse 1*)

In addition, because Wellington ED has only designated security orderlies from 12am-3am Thursday to Saturday nights, increasing the security operating time to 24 hour on those nights is identified as a useful Wellington-specific intervention.

Discussion

The results from the survey and interviews with ED staff indicate that staff believe alcohol-related presentations cause an important negative impact upon staff in terms of workload and workplace safety, and, in the quality of care of both non-intoxicated and intoxicated patients. Other research has found that staff predominantly attribute health problems to patients, which can be a barrier to providing high quality care.¹⁵

A high proportion of all staff experience verbal assault from intoxicated patients, and a large proportion of nurses and ambulance officers report physical assault during work. In this study, the severity of reported assaults was not explored. The subjective nature of verbal abuse needs to be acknowledged with people having differing ideas as to what constitutes an incident of verbal assault. From the interviews with staff, aggressive and abusive behaviour from intoxicated patients seems to be regarded as fairly standard and endured with stoicism, and may even be under-reported, due to staff becoming accustomed to such conduct.

Staff commented that they respond to abuse by developing a “thick skin” over time. Despite this, the majority of staff report feeling threatened by intoxicated patients, and the vast majority feel that these patients negatively impact upon their mood at work. Ten people were unsure whether alcohol impacted on triage score, which may have occurred due to non-medical staff’s unfamiliarity of this term (not defined in the questionnaire), causing an under-reporting of an effect.

This was a small, exploratory study, recruiting staff from only one hospital and as such the results need to be replicated and confirmed with a larger sample and at other locations in New Zealand. Non-response and self-selection bias could have affected results, as the response rate for ED staff (excluding ambulance officers) was probably only around 50% and respondents may not have been representative of all ED staff. The inclusion of ambulance officers in the sample, although adding depth to the results, may also have complicated the picture as these staff may have a different experience of intoxicated patients (on the street) than ED staff in the hospital.

It would be of interest to survey these groups in more detail separately in the future. We also developed our own questionnaire as no validated tool could be found. However, the responses from the in-depth interviews correlated well with the overall findings from the survey, and complemented the surveys by providing concrete examples of negative impacts from alcohol-related presentations.

Some estimates of the impact of alcohol-related presentations may be inflated (such as effects on waiting time and triage), due to the large clinical (and psychological) burden from these patients causing them to be recalled more clearly.¹⁵ However, the level of reported negative effects on staff and other patients is high, raising occupational health and safety concerns, in particular around the high likelihood of staff being assaulted by intoxicated patients, and issues around the treatment of both intoxicated and non-intoxicated patients, especially at peak times in the ED.

An unanticipated impact of intoxicated patients upon ED was the effect on the mood and well-being of other patients. Interviewed staff commented that other patients frequently felt threatened and afraid of intoxicated patients, and that this can significantly contribute to the stress of their conditions.

Policies to separate intoxicated patients from non-intoxicated patients are in place, however this current practice is clearly insufficient. Resource limitations are an obvious barrier to isolating intoxicated patients. The modern layout of Wellington Regional Hospital places most patients in individual rooms, and the impact of intoxicated patients on other patients is likely to be higher in other centres.

The impact of alcohol on EDs may be tightly linked to social changes within societies. An Australian study looking at alcohol presentations to ED found that among 43 hospitals alcohol presentations rose from 110 to 150 per 100,000 people between 2005 and 2008.¹⁶ This increase was attributed to broader social changes, with more young people drinking and significant peaks around times of large public gatherings. A further increase in young people being admitted to ED was attributed to an increase in the availability of ready-to-drink alcoholic beverages.

These results indicate that the impact of alcohol on EDs may be responsive to social and policy change. Many of the legislative changes suggested by the Law Commission's review of Liquor Laws were raised as potential means for reducing alcohol-related presentations, and their associated burden, the ED.

The ED can also be a setting for alcohol interventions, usually in the form of screening for alcohol misuse and administration of some form of brief intervention, with follow-up appointments with alcohol specialists in some cases.¹⁷ Such interventions can be effective in the short term at reducing alcohol consumption and visits to the ED.¹⁸

A systematic review of brief interventions found significant positive results in many studies and improved effectiveness with scheduled follow up appointments but much diminished effectiveness over time.¹⁹ These are recommended for implementation in EDs, due to short term success,²⁰ however, policy changes to reduce problematic alcohol consumption in the wider society, such as pricing increases and raising the purchase age, are more likely to have an enduring impact on alcohol-related ED presentations.

Conclusions

Alcohol is a significant cause of burden on the health system, particularly EDs. However, little research assessing the impact of alcohol on EDs in New Zealand has been performed. This study investigated the impact on staff of alcohol-related ED presentations. From staff reports, assault from intoxicated patients is common and alcohol related presentations impact negatively on other patients as well as workload and staff mood. Both ED-specific and social policies to reduce alcohol-related ED presentations are needed, to reduce the burden on hospital services.

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Off-label use of atypical antipsychotic medications in Canterbury, New Zealand

Erik Monasterio, Andrew McKean

Abstract

Aim To estimate the frequency and characteristics of “off-label” use of atypical antipsychotic medications (AAPs) by psychiatrists in Canterbury, New Zealand.

Methods Data on “off-label” prescribing of AAPs including the choice of medication, frequency of prescribing, and the indications for its use was collected using a postal survey of psychiatrists registered with the NZ Medical Council in the Canterbury region.

Results 48 psychiatrists (71%) completed the survey. Forty-six (96%) prescribed AAPs “off-label”. By far the most common agent was quetiapine (94%). Twenty-eight respondents (58%) prescribed “off-label” at least once a week. The most common reasons for the use of these agents was: anxiety (89%), sedation (79%), post-traumatic stress disorder (57%), treatment augmentation of another antipsychotic agent (48%) and behavioural and psychological symptoms of dementia (33%).

Conclusion “Off-label” prescribing, particularly of quetiapine is very common in the Canterbury region, despite little scientific evidence for this kind of use, increasing evidence of abuse and potential for significant side-effects.

The term “off-label” for the use of a medication generally relates to the prescription of a drug without approved official authorisation. Within New Zealand this authorisation is provided by Medsafe (the New Zealand Medicines and Medical Devices Safety Authority). For prescription medications the approval process requires robust scientific evidence of efficacy and safety for specific clinical situations.

Off-label prescribing has been around for decades, is relatively common in most aspects of clinical practice and is considered to be legal.¹

Although conventional antipsychotic medications, such as thioridazine and chlorpromazine have traditionally been prescribed off-label, before the early 1990’s their use was largely reserved for adults with severe psychotic disorders;² unpleasant extrapyramidal side-effects and cardiovascular risks arising from widening QTc interval appear to have largely limited their use outside these disorders.

The introduction of better tolerated, second generation atypical antipsychotics (AAPs) such as risperidone, olanzapine, quetiapine, ziprasidone and aripiprazole from the mid-1990s led to a rapid expansion of antipsychotic medication use for a wide variety of unlicensed conditions and in more diverse clinical populations. This unlicensed use has predominantly not been supported by scientific evidence.³

Studies examining the use of AAPs across specialist inpatient and outpatient populations and general practice indicate that between 43% and 70% of atypical antipsychotic use is off-label.⁴⁻⁹

In an illuminating analysis of reports from the 2001 National Disease and Therapeutic Index (which tracks epidemiological trends and treatment patterns among private physicians in the United States) Radley et al found that 73% of the off-label use of 160 commonly prescribed drugs lacked evidence of clinical efficacy, and only 27% was supported by strong scientific evidence: the greatest disparity between supported and un-supported off-label uses was found among prescriptions for psychiatric uses (4% strong support vs. 96% limited or no support).¹⁰ Although the literature indicates a high prevalence of off-label prescribing, there is little guidance on how to address this issue or limit the practice.

Expanded use of atypical antipsychotic agents has come with a substantial cost burden. In NZ the cost of all antipsychotics was \$23.1 million in 2000 and the cost for only AAPs rose to \$54.5 million in 2010 (Personal Communication: G MacGibbon, PHARMAC, 27/10/2010).¹¹ In 2007 US spending on AAPs was estimated at US\$13.1 billion, exceeded only by lipid regulators and proton pump inhibitors.¹²

Peer-reviewed scientific publications have paid substantial recent attention to reporting on the illegal promotion of off-label prescribing by pharmaceutical companies and the legal repercussions of this practice; these illegal marketing efforts would appear to have substantially contributed to the expanded use of AAPs.¹³⁻²⁰

Within the United States the Food Drug Administration (FDA) has been criticized for its poor monitoring of drug companies promotion of off-label uses of their drugs.¹⁴⁻¹⁷ Recent landmark legal cases by the US Department of Justice, charging that the drug companies Eli Lilly and AstraZeneca illegally promoted the off-label use of the AAPs olanzapine and quetiapine have settled before trial for payments of US\$1.4bn and US\$520m respectively.¹⁸⁻²⁰

In commenting on the legal case against AstraZeneca US Attorney General Eric Holder said that illegal acts by drug companies “can put the public health at risk, corrupt medical decisions by health care providers, and take billions of dollars directly out of taxpayers’ pockets.”²⁰

Determining the appropriateness of off-label prescribing in current clinical practice is particularly challenging as physicians must weigh up risks and benefits of various medications across diverse clinical presentations, with limited scientific evidence of efficacy, and under pressure from various stakeholders including patients, advocacy groups, medical staff and the pharmaceutical industry.

The frequency and characteristics of off-label AAPs prescribing in New Zealand is not known. It is the author’s clinical experience (in New Zealand) that the off-label use of AAPs is common in primary and specialist care settings. The aims of this study were to ascertain the frequency, patterns and characteristics of off-label prescribing of AAPs.

Methods

Psychiatrists in Canterbury, New Zealand were identified through the New Zealand Medical Council website.²¹ A postal survey and an addressed return envelope were sent to all psychiatrists based in Canterbury (pop. 504,000), New Zealand between January and February 2010. The questionnaire was anonymous and asked about generic prescribing and the characteristics of off-label prescribing of AAPs: The choice of medication, frequency of prescribing, and the indications (ranked in order of frequency).

Results were collated and analysed on an Excel spreadsheet (Microsoft, USA).

Results

There was a 71% (48/68) response rate. Of the 48 who responded to the questionnaire 37 (77%) always prescribed generically and 11 (23%) sometimes prescribed generically. Forty-six (96%) had prescribed AAPs for an off-label indication.

Of those psychiatrists that had prescribed AAPs for an off-label indication, the most common first-line agent was quetiapine (94%) followed by olanzapine (2%), risperidone (2%) and clozapine (2%).

With respect to the frequency of off-label prescribing: 3 (6%) did so on a daily basis; 13 (27%) two to three times a week; 12 (25%) once a week; 7 (15%) every 2 weeks and 13 (27%) once a month.

The most common indications for off-label use are summarised in Table 1. The majority of psychiatrists prescribed AAPs for anxiety (89%) and sedation (79%), followed by symptoms of post-traumatic stress disorder (57%) and treatment augmentation of another antipsychotic agent (48%).

Other relatively common indications include management of behavioural and psychological symptoms of dementia (33%), adjustment disorder (20%) and psychotic symptoms of Parkinson's disease (20%).

Table 1. Indications for off-label AAPs prescribing

Off-label indication	Percentage of psychiatrists that prescribed for this indication
Anxiolytic	89 %
Sedative	79 %
PTSD	57 %
Augmentation of another antipsychotic	48 %
Behavioral and psychological symptoms of dementia	33 %
Adjustment disorder	30 %
Delirium	20 %
Parkinson's disease - psychotic disorder	20 %
Obsessive Compulsive Disorder	11 %
Tardive Dyskinesia	9 %
Gilles de la Tourette's syndrome	9 %
Behavioral disturbance/impulsivity in children without ADHD	4 %
Insomnia	2 %
Agitation/ Overarousal	2 %
Behavioral disturbance in ID	2 %
Huntington's Disease	2 %
To target depressive ruminations and aid sleep	2 %

Discussion

The findings of the current survey support the observations of a number of other studies which highlight a high frequency of off-label use of AAPs across diverse patient populations.^{5-10,22} The results reveal high levels of off-label prescribing

amongst Christchurch psychiatrists, with most prescribing generically. The reported rate of 96% is significantly higher than the 65% rate of off-label use found in a 2000 British study with similar methodology.²³ Whether this is due to overall higher use in NZ, a local phenomenon or a reflection of increased use over time is not possible to determine from our data.

The systematic literature reviews that have examined off-label use of AAPs indicate that there is no current evidence for monotherapy in the management of anxiety disorders, although there is some evidence to support the use of: adjunctive risperidone in the treatment of refractory obsessive compulsive disorder, post-traumatic stress disorder, pervasive developmental disorder and Tourette's syndrome; and olanzapine in the treatment of refractory depression and borderline personality disorder.²⁴⁻²⁶ Given that the most common symptoms and conditions for which these agents were used included anxiety, sedation and PTSD, it is important to note a general lack of evidence in support of the current use of off-label AAPs.

However, perhaps the most striking finding of the survey is the extent to which quetiapine is the most commonly prescribed off-label agent: it is the first preferred choice of 94% of respondents, and more than half of all respondents prescribed this medication off-label every week. A recent study examining the use quetiapine in an inpatient setting over an 18 month period determined that it was used for licensed conditions in only 25% of patients.²⁷ Although the extent to which side-effects are dose related is still debated, short- and long- term use of quetiapine is associated with an increased risk of side-effects including weight gain, dyslipidaemia and insulin resistance.²⁸

It is also important to note that cases of quetiapine abuse have been increasingly reported in the literature.²⁹⁻³⁴ In the USA it has been referred to as Quell, Susie-Q, Baby Heroin and Q-Ball²⁹⁻³¹: It has reportedly been crushed and administered intranasally^{39,33} and intravenously.^{30,31}

The annual cost of quetiapine in NZ to May 2010 was \$13.5 million (excluding any confidential rebates) and with an estimate that up to 70% of its use is off-label, the cost burden approximately \$9.5 million per year or 17% of the total annual cost for all AAPs.³⁵ However this cost has decreased with the introduction of cheaper generics in August 2010.

Limitations to this study include the retrospective nature of the survey, and the possibility that psychiatrists do not have an accurate recollection of the information requested. In order to ensure anonymity, information on the specialty area where psychiatrists worked was not requested, therefore it is not possible to determine to what extent off-label use in our survey reflects prescribing practices in areas of particular concern, such as child and adolescent and elderly services.

Conclusion

In summary, off-label use of AAPs, particularly quetiapine appears to be common in current specialist mental health practice, despite limited scientific support for this kind of use. Considering that even low dose AAPs can have significant side-effects, are of unknown efficacy, and appear to have a potential for abuse, we recommend a more considered and measured approach to their use.

There is a pressing need to know to what extent they are used in primary care settings, what factors contribute to their popularity and to ensure that patient safety is not jeopardised and valuable resources are not wasted.

We recommend that whenever these agents are used off-label, informed consent is obtained, treatment is monitored, and an end- or review point of treatment is identified.

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Five-year follow-up of an acute psychiatric admission cohort in Auckland, New Zealand

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Abstract

Aim This paper describes a follow-up of acute psychiatric hospital contact in Auckland, New Zealand for an admission cohort in the 5-years past an index admission (published in the *NZMJ* in 2005).¹

Methods A 5-year follow-up study of hospital psychiatric service utilisation by 924 patients admitted (index admission) in Auckland during 2000. Hospital admissions within New Zealand for this population were extracted from electronic records. Relevant demographic information (gender, age and ethnicity) and clinical data (primary diagnosis at index admission and admission history) were included for each person. Descriptive analysis of inpatient data and negative binomial regression models were conducted.

Results Of 924 patients, 38.5% had no readmissions anywhere in New Zealand in the 5-years following index discharge. 41.0% were readmitted within 12 months and 61.4% were readmitted within 5 years of index discharge. Only 5.6% experienced an admission every year for the 5-years post index admission. Readmission was least likely for those with index discharge diagnosis of depression. A history of admissions prior to index admission and Māori ethnicity were characteristics associated with higher numbers of readmission. Those who were younger, or a diagnosis of schizophrenia/schizoaffective disorder or previous admissions tended to have longer total length of stay over the 5-years.

Conclusions More than a third of patients had no further hospital contact and the two factors associated with readmission were a history of previous admissions and Māori ethnicity. Reliable community-based data needs to be a priority to enable exploration of community service utilisation and impact of service alternatives to hospital for acute care.

Acute hospital psychiatric care remains an integral component of the treatment continuum in New Zealand despite the increased emphasis on community-based mental health care. Patients who continue to require hospitalisations deserve attention; both due to the discontinuation of care as patients shift between community and inpatient services and because hospital admissions make considerable demands on resources.

The shift to community care associated with deinstitutionalisation embraced the principle of avoidance of hospitalisation whenever possible. Increased readmissions following closure of the large mental health asylums have been a source of concern.²⁻⁴

An earlier study reviewed 932 people who were consecutively admitted during the period 1 January 2000 to 31 December 2000 to three acute psychiatric units located in the north, west and south areas of Auckland.¹

The aim of this present follow-up study was to look at engagement with hospital and community-based mental health services in the 5 years after discharge. Inconsistencies in the recording of community-based care contacts during the earlier years of the study period between the two health services involved meant that data could not reliably be aggregated.

Accordingly, this paper only reports on the 5-year acute hospital contact for psychiatric reasons in New Zealand for the original admission cohort; in particular, readmissions in the 5 years after index admission. Potential predictors of higher admissions and length of inpatient bed stay (for example, demographic, diagnosis and service related characteristics), were examined.

Methods

There were three acute units providing psychiatric beds for adults aged 18-65 years living in the community catchment populations of north, west and south Auckland between 2000-2006 (total population 805 293 Census 2001; 914 694 Census 2006⁵). The ethnic groups making up the population were diverse; 48% European, 12.0% Maori, 12.4% Pacific Peoples, 14.9% Asian and 13% other.⁶

The present study retrospectively reviews the utilisation of acute hospital services for psychiatric reasons by a cohort of people during the 5 years after discharge from their respective first admission episode in 2000 (index admission). Eight of the original admission cohort were found to be aliases of people already included in the study group. The study population comprised 924 people once aliases had been merged with primary records.

All hospital admission episodes within New Zealand for the study population were extracted from the New Zealand Health Information System (NZHIS) via the Patient Information Management System (PiMS also known as iPM) [iSoft, Oxfordshire, UK] used by both health services involved in the study. This data was entered into a Microsoft Access® database (Microsoft Corporation, Redmond, Washington, USA) where reasons for admissions were coded as; psychiatric, poisoning/overdose, other medical or unknown. Coding decisions were based on the discharge diagnosis recorded for each episode or the service admitted to, as recorded in PiMS. Only those admissions coded as psychiatric and poisoning/overdose were included in the analyses.

The number of admissions for each of the aforementioned reasons was calculated for each person for each of the 5-years of follow-up. Total inpatient length of stay was also calculated for each person following discharge from their respective index discharge. Relevant demographic information (gender, age and ethnicity) and clinical data (primary diagnosis at index admission (DSMIV), psychiatric admission history and date of death where applicable) were included for each person from the earlier study.¹

Descriptive analysis of study data was undertaken with SPSS version 15 (SPSS Inc., Chicago, IL, USA). Chi-squared and Kruskal-Wallis tests were used to investigate differences between groups. Negative binomial regression models were conducted using SAS version 9.1 software (SAS Institute Inc, Cary, NC, USA) to determine whether gender, ethnicity, age (at index admission), primary diagnosis (categorised as schizophrenia [including schizoaffective disorder], bipolar disorder, depression and other disorders) or previous psychiatric admission were associated firstly with number of admissions and secondly (for those who were admitted in the 5 years) the total length of stay (LOS). The total number of days in the study (log transformed) was included in the model as an offset because some people from the original study group died before the five year follow-up period concluded.

This study was conducted as a retrospective review of patient/client notes or data and as such it was defined as an observational study not requiring formal ethical committee review.⁷

Results

Patient population—The cohort making up this review comprised 924 individuals who had at least one acute psychiatric admission in 2000. Table 1 shows the demographic and clinical data for the study population at the time of their index admission.

The most common discharge diagnoses were schizophrenia (including schizoaffective disorder) and bipolar disorder (manic, depressive, mixed and unspecified episodes). Just over half the study population had at least one previous psychiatric admission (59.6%) prior to their respective index admissions.

Table 1. Study population description at index admission (2000)

Variables	Total population (n=924)
	n (%)
Gender	
Male	517 (55.9)
Female	407 (44.1)
Age (years)	
Mean (SD)	35.8 (11.7)
Median (range)	34 (16-68)
Ethnicity	
European	571 (61.8)
NZ Māori	217 (23.5)
Pacific people	97 (10.5)
Asian	39 (4.2)
Primary diagnosis (DSMIV)	
Schizophrenia/schizoaffective	352 (38.1)
Bipolar disorder	222 (24.0)
Depression	116 (12.6)
Other disorders*	234 (25.3)
Previous psychiatric admission	
No	373 (40.4)
Yes	551 (59.6)

*Other disorders includes adjustment, anxiety, cognitive, delirium, dissociative, eating, substance use, somatoform, other psychotic, other mood and no Axis I disorders).

Over the 5-year study duration a total of 40 people died (4.3%); one before the end of first year of follow-up, 11 during year one, 16 during year two, five during year three, three during year four, and four during year 5. Two of the deceased had no previous psychiatric admission history prior to 2000. Admission data were included for the year that an individual died.

Five-year follow-up—More than a third of the original cohort (n=356; 38.5%) had no further acute psychiatric hospital contact during the entire 5-year study duration. A greater proportion of those with no psychiatric admission history prior to index admission had no further hospital contact compared to those with previous psychiatric admissions; 47.7% versus 32.3% (Chi-squared=22.32, df=1, p<0.001).

Over the follow-up period 16.7% (n=154) of the original cohort had only one subsequent admission, 10.7% (n=99) had two and 34.1% (n=315) had three or more

admissions (range 3-43). The median length of stay (LOS) was 22.6 bed-nights for admissions occurring during the study period (Table 2).

There was a significant difference in median LOS by the total number of admissions over 5-years; patients with multiple admissions (≥ 3) had longer admissions (median 26.6 bed-nights).

Table 2. Number of admissions and length of stay (LOS) over 5 years (n=924)

Number of admissions	n (%)	Average LOS per admission ^a		Total LOS for 5-year period ^b	
		Mean (SD)	Median (Range)	Mean (SD)	Median (Range)
None	356 (38.4)	-	-	-	-
1	154 (16.7)	34.1 (45.1)	21 (0-260)	34.1 (45.1)	21 (0-260)
2	99 (10.7)	31.5 (49.0)	18.5 (0-358.5)	62.9 (98.0)	37 (0-717)
3 or more	315 (34.1)	35.7 (34.9)	26.6 (0.33-236)	237.4 (277.6)	141 (1-1743)
Total with admissions	568 (61.5)	34.5 (40.5)	22.6 (0-358.5)	151.9 (232.6)	67 (0-1743)

Note: Admissions that did not cross 12pm midnight were recorded as an admission with zero length of stay.

^a Kruskal-Wallis Chi-squared (2def)=12.349, p=0.002 ; ^b Kruskal-Wallis Chi-squared (2def)=222.855, p<0.001.

In terms of the duration of time until a subsequent admission, 41.0% of the original cohort had another admission within 12-months or less of the index admission, 11.1% were readmitted sometime in the second year, 4.1% sometime in the third year and 2.6% sometime in the fourth and fifth year after discharge from their index admission.

There was a significant difference in the proportion of people having further admissions over the 5-year period between diagnostic groups; 68.5% patients with bipolar disorder were readmitted, followed by 67.3% with schizophrenia or schizoaffective disorder, then 52.1% of those with other disorders and the lowest proportion was found for those diagnosed with depression, 49.1% ($\chi^2=25.75$, df=3, p<0.001).

For those who were readmitted at least once in the follow-up period (n=568), no statistical difference was found in the period of time to readmission between the four diagnostic groups [schizophrenia, bipolar, depression, other disorders] ($\chi^2=12.70$, df=12, p=0.39).

Only 5.6% (n=52) of the study population experienced at least one admission in every 12-month period following their index admission. Figure 1 shows the decay curve of those having continuous psychiatric hospital contact over 5 years and the total number of the original cohort who had an admission in any of the 12-month periods after the index admission.

The negative binomial regression found evidence that ethnicity (p=0.001) and previous admissions (p<0.0001) were associated with total number of admissions in the 5-year follow-up period after adjusting for other factors in the model (Table 3). Māori and people with previous admissions were more likely to have a greater number of admissions and Pacific peoples were more likely to have fewer admissions.

Of those who had been admitted at least once in the follow-up period, the median length of stay during the 5-year follow-up was 67 days but ranged from very brief day admissions with no overnight stay to a total of 1743 days over the 5–years.

The regression model found that diagnosis ($p<0.0001$) and previous admissions ($p<0.0001$) were significantly associated with total length of stay after adjusting for other factors in the model. Those with bipolar disorder or depression or ‘other’ disorders had shorter total length of stay than those with schizophrenia (including schizoaffective disorder). Those with previous admissions also stayed longer.

Age was also found to be associated with total length of stay ($p=0.017$); there was a small but real decrease in incidence with age.

Table 3. Negative binomial regression analysis: incidence ratios for number of admissions and length of stay

	Number of Admissions		Total Length of Stay	
	Incidence Ratio ^b (95% CI)	Statistics	Incidence Ratio ^b (95% CI)	Statistics
Gender				
Female	1	$\chi^2=3.44$, df=1, p=0.064	1	$\chi^2=0.31$, df=1, p=0.58
Male	1.21 (0.99-1.49)		1.06(0.86-1.32)	
Ethnicity				
European	1	$\chi^2=15.861$, df=3, p=0.0012	1	$\chi^2=5.49$, df=3, p=0.14
NZ Māori	1.37 (1.08-1.75)		0.98 (0.54-1.80)	
Pacific Peoples	0.66 (0.47-0.92)		1.22 (0.96-1.56)	
Asian	1.03 (0.63-1.70)		0.79 (0.56-1.13)	
Age	0.99 (0.98-1.00)	$\chi^2=1.28$, df=1, p=0.26	0.99 (0.98-0.99)	$\chi^2=5.698$, df=1, p=0.017
Diagnosis ^c				
Schizophrenia	1	$\chi^2=1.03$, df=3, p=0.80	1	$\chi^2=21.69$, df=3, p<0.0001
Bipolar	1.12 (0.86-1.46)		0.75 (0.58-0.98)	
Depression	1.12 (0.80-1.57)		0.55 (0.38-0.80)	
Other	1.02 (0.78-1.33)		0.54 (0.41-0.71)	
Previous Admission				
No	1	$\chi^2=44.18$, df=1, p<0.0001	1	$\chi^2=15.65$, df=1, p<0.0001
Yes	2.10 (1.70-2.60)		1.61 (1.28-2.02)	

^a Model includes gender, ethnicity, age at index admission, previous admission and primary diagnosis.

^b All Incidence Ratios are adjusted for other variables in the model.

^c Diagnosis includes: schizophrenia (including schizoaffective), bipolar, depression and other disorders combined. Other includes other psychotic and other mood disorders, adjustment, anxiety, cognitive, delirium, dissociative, eating, somatoform, substance use, and no Axis 1 disorder.

Discussion

This study describes acute hospital utilisation for psychiatric reasons (including poisoning/overdose) in the 5 years post-index discharge for the original cohort. Over one third (38.5%) of the cohort had no further readmission to any hospital facility in New Zealand in the 5 years after their index admission for acute psychiatric reasons. First time users of acute inpatient psychiatric services were less likely to be readmitted in the 5 years following index discharge. It is not known whether the group without further readmissions remained in contact with community services during that period.

The proportion of the cohort readmitted within 12 months of index discharge (41.0%) is consistent with the 40-50% range reported in other studies, but the proportion readmitted in the 5-year range (61.4%) is slightly lower than the reported range of 65-70%.¹⁰⁻¹³ However, 34% of the New Zealand cohort experienced three or more admissions over the 5-year period. This group tended to experience much longer admissions than those with fewer readmissions.

Hospital care is costly and consequently those admitted more frequently account for significantly higher costs; not only due to a higher number of admissions that lead to an increased cumulative length of stay over 5-years but also the median duration of these admissions is longer for those who had multiple admissions. Hospitalisation is not only fiscally expensive to health services but can also have high personal costs to the individual and their family.

Only a small group (5.6%) were 'high user's' if Kent's definition of an average of at least one admission per year for the period under review is used.¹⁴ An earlier study of all first-ever psychiatric hospital admissions in New Zealand (1980-81) found that 9.7% had five or more admissions over a 5-year follow-up period.³ While this is higher than our finding (5.6%) it is important to remember that there were major changes to treatments and the way mental health services were delivered in New Zealand over the 20 years making comparison very difficult.

Those who experienced higher numbers of readmission in the 5-year period after index discharge were more likely to have experienced prior psychiatric admissions or to be Māori. The number of previous psychiatric hospitalisations has consistently been shown to be a significant predictor of rehospitalisation,^{10,15-17} and this New Zealand study is in agreement.^{15,16,20-22}

Similar to previous work, the findings of the present follow-up study found that Māori were more likely to be readmitted than non-Māori.^{23,24} Literature that explores Māori experiences of acute mental health services has highlighted gaps and concerns.^{20,25} These observations and the disparity in hospitalisation rates emphasize the need for further investigation into how acute mental health services can more effectively meet the needs of Māori and improve outcomes.

In contrast, the likelihood of higher numbers of readmission was not associated with sociodemographic factors such as younger age or male gender that has been reported by other research.^{3,15,17,18} This finding may be partly explained by the regression analysis we used which adjusted for other factors in the model. For example, a Canadian study of 3-year follow-up that examined a number of patient-related

variables associated with readmission identified a number of factors including gender, diagnosis, admission history and social factors in univariate analyses however in the multivariate analysis the only variable that consistently identified those at risk of readmission was a history of repeat admissions.¹⁶

Other patient-related factors (including psychopathology, aggression, substance use and levels of social support) and system variables (including service structure, admissions policies and bed pressures) have been reported to influence the decision whether to admit a person or not.^{15,16,19} However, it was beyond the scope of this study to examine all of these factors.

Bernardo and Forchuk have also suggested that staff attitudes and perceptions may contribute to readmission.¹⁶ They suggested that 'high user' patients may have issues contributing to their return to hospital but these may be overlooked because the patient is seen as familiar with unchanging issues. Roick et al also found a factor that contributed to increased hospital admissions was patients' satisfaction with the effectiveness of mental health services; when this was low, inpatient care increased.¹⁵

The authors suggested that an inefficient mental health system could cause a high number of hospital admissions. Internationally and nationally, the readmission rate has been suggested to be a valuable indicator of the quality of hospital care.²¹ Further evaluation of the quality of acute inpatient mental health care is needed and whether this rate is increasing.

The study has a number of limitations. As highlighted above, the retrospective design utilising routinely collected hospital data meant that many factors that may have been associated with readmission were not included. The information collected in this retrospective review is limited by the degree, extent and accuracy of the original data source. Community contacts had been a key variable of interest in this study but it was not possible to obtain reliable community contact data for the period of interest.

Different methods of collecting and reporting community contacts by health services to the Mental Health Information National Collection (MHINC) meant community data could not be aggregated meaningfully across different services. The presence of data quality issues in the earlier periods are not unexpected and caution was recommended when interpreting patient access information (contacts and bed stay) from the 2000-2006 period.^{9,26} Because of these limitations this study collected only hospital event data via PiMS at both health services.

Finally, poisoning/overdose admissions were included in the 5-year follow-up after an acute psychiatric admission as they are often indicative of admissions for acute psychiatric reasons. However, it is important to acknowledge that subsequent hospital admissions for poisoning/overdose may have been briefer than those to a psychiatric unit.

In summary, this study reports the 5-year readmission rates of a cohort of mental health patients admitted to an acute inpatient service in 2000. Almost 40% of the group had no further acute admissions for psychiatric reasons anywhere in New Zealand during the 5-years after discharge. The two factors associated with readmission were having a history of previous admissions or being of Māori ethnicity. One third of the group had three or more admissions in the 5 years following index discharge with longer bed stays.

The findings highlight the need for reliable community-based data to enable exploration of the use of community services following acute discharge, especially in preventing readmission, and the impact of acute service alternatives to inpatient units for times of crisis. This is particularly important given the negative and distressing experience reported by people admitted to acute psychiatric inpatient units.^{20,25}

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Quetiapine for the treatment of behavioural and psychological symptoms of dementia (BPSD): a meta-analysis of randomised placebo-controlled trials

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Abstract

Aim This meta-analysis is aimed to determine the efficacy of quetiapine for the treatment of behavioural and psychological symptoms of dementia (BPSD).

Method Our electronic search included MEDLINE (1950-2009), Cochrane Central Register of Controlled Trials and PsychINFO. We also did a hand search of the International Psychogeriatric Association poster presentations and checked the National trial registry data bases from USA, UK, RSA, Holland, Australia and New Zealand. We included double-blinded randomised placebo-controlled trials studies that measured BPSD with the Neuropsychiatric Inventory (NPI). The Clinical Global Impression of Change scale (CGI-C) was our secondary outcome.

Results Six sets of data were included in this meta-analysis. Patients receiving quetiapine improved when compared to placebo with a weighted mean difference of -3.05 (95% CI: -6.10, -0.01) and -0.31 (95% CI: -0.54, -0.08) respectively on the NPI score and CGI-C score.

Conclusion This meta-analysis found that quetiapine is statistically more efficacious than placebo in the treatment of BPSD as measured by the NPI and CGI-C. However, improvement is of a small magnitude and observable clinical significance is questionable.

Dementia is a growing public health concern. The recently published Dementia Economic Impact Report by Alzheimers New Zealand estimated by 2050, 2.7% of the New Zealand population will have dementia or 146,699 people, and new cases will comprise 0.8% of the population (44,375 people) each year.¹ In 2008, the total financial cost of dementia was estimated as \$712.9 million and 27,449 years of healthy life were lost due to dementia across New Zealand. Similar findings were reported in Australia.²

Behavioural and psychological symptoms of Dementia (BPSD) occur frequently (50-80%) in people with dementia at some point during their illness.^{3,4} These symptoms include anxiety, depression, irritability, delusions, hallucinations, insomnia, wandering, aggression and agitation. BPSD can result in premature institutionalisation, diminished quality of life for patients and caregivers, excess disability, caregiver stress and significant financial cost.⁵

Non-pharmacological management should always be the first line treatment for BPSD and pharmacological treatment can be considered when non-pharmacological management fails or when there is a significant risk (for example, physical aggression; agitation; psychosis).

In New Zealand, risperidone is the only atypical antipsychotic listed on the Pharmaceutical Schedule for the treatment of BPSD including aggression, activity disturbance and psychotic symptoms. A number of non-randomised, non-placebo-controlled and non-blinded studies of quetiapine have shown some effectiveness for the treatment of BPSD.

Rainer et al have found quetiapine and risperidone were equally effective and generally well tolerated.⁶ Rocca et al reported that quetiapine, risperidone, and olanzapine produced similar significant improvements in behavioural disturbances.⁷ Quetiapine was also found to have a good efficacy in reducing behavioural symptoms, particularly delusions, agitation and aggression in three other open-label trials.⁸⁻¹⁰

Ballard et al recently published an updated review on the management of agitation and aggression associated with Alzheimer's disease.¹¹ For the treatment of agitation/aggression the best evidence is for risperidone. There is also some evidence for aripiprazole but there is no clear evidence from published randomised controlled trials to indicate that other atypical antipsychotics are efficacious.^{12,13}

Despite the lack of evidence in published systematic reviews and meta-analyses,^{12,14-16} we have observed in our clinical practice that quetiapine is continued to be used off-label by psychiatrists, geriatricians and general practitioners for the treatment of BPSD, particular in residential care facilities.

The recently published clinical recommendations developed by the Royal Australian and New Zealand College of Psychiatrist Faculty of Psychiatry of Old Age (New Zealand) also listed quetiapine (up to 100mg) as one of the antipsychotics to treat psychotic symptoms in dementia.¹⁷ Bishara et al reported in an expert opinion paper where quetiapine was seen as the drug of choice for BPSD in the UK.¹⁸

The perception that quetiapine is "safer" is most likely due to the report that risperidone was associated with a three-fold increased risk of serious cerebrovascular adverse events compared to placebo.¹³ There was a subsequent advisory note from the United States Food and Drug Administration warning of an increased mortality in patients with dementia who are treated with atypical antipsychotic medications.¹⁹

Previous meta-analyses of double-blinded randomised placebo-controlled studies included a limited number of studies of quetiapine for the treatment of BPSD. For example, the Cochrane review by Ballard et al included only one study of quetiapine.¹⁴ Schneider et al included three studies of quetiapine in their meta-analysis of atypical antipsychotics for dementia.¹² There was a lack of evidence for or against quetiapine because the three studies used different selection criteria and outcomes could not be statistically combined using a common rating scale.

We believe there have been new randomised placebo-controlled trials investigating the use of quetiapine for the treatment of BPSD and an updated meta-analysis is warranted to guide our clinical practice and to determine whether there is any evidence supporting the off-label use of quetiapine in dementia. The objective of this meta-analysis is to determine the efficacy of quetiapine for BPSD in elderly patients with dementia.

Method

Search strategies—Our search included MEDLINE (1950-2009), Cochrane Central Register of Controlled Trials and PsychINFO. Search terms were: quetiapine, behavioural and psychological symptoms of dementia, dementia and Alzheimer's dementia. We did a hand search of the International Psychogeriatric Association poster presentations and checked the National trial registry data bases from USA, UK, RSA, Holland, Australia and New Zealand. The Medical Department of AstraZeneca New Zealand was also contacted for their database on clinical trials conducted for quetiapine in dementia.

Selection criteria—We only included double-blinded randomised controlled trials (RCTs) that used placebo as their control. As data was limited we included all trials of adequate design. We included studies that measured BPSD with the Neuropsychiatric Inventory (NPI) among patients with any stage and any subtypes of dementia living in any clinical setting. We decided to include different subtypes of dementia because medical practitioners working in primary care setting often do not have the specialist skill to diagnose subtypes of dementia. The Clinical Global Impression of Change scale (CGI-C) was our secondary outcome. We contacted the authors of three studies (Tariot et al, Kurlan et al and Paleacu et al) to obtain unpublished and/or missing data on NPI and CGI-C in order to complete this meta-analysis.

Neuropsychiatric Inventory (NPI)²⁰—Neuropsychiatric Inventory is a tool for assessment of psychopathology in patients with dementia and other neuropsychiatric disorders. The NPI is based on a structured interview with a caregiver who is familiar with the patient. The following 10 neuropsychiatric domains are evaluated: delusions, hallucinations, dysphoria, anxiety, agitation/aggression, euphoria, disinhibition, irritability/lability, apathy, aberrant motor activity, night-time behaviour disturbances. For each domain a screening question is asked to determine if the behavioural change is present or absent. If the answer is positive the domain is explored at greater depth with the sub-questions. If the sub-questions confirm the screening question, the severity and frequency of the behaviour are determined according to the criteria provided for each domain.

Frequency is rated 1 to 4 and severity is scored 1 to 3. The product (severity x frequency) is calculated for each behavioural change present during the previous month or since the last evaluation (e.g. in order to evaluate treatment efficacy). A total NPI score can be calculated by adding the scores of the ten behavioural domain scores together.

Two versions of NPI are available: standard version and Nursing home version. The 10 behavioural domains and scoring system in the two versions are identical.

Clinical Global Impression of Change Scale (CGI-C)²¹—The CGI-C is a 7-point scale that requires the clinician to assess how much the patient's illness has improved or worsened relative to a baseline state at the beginning of the intervention. It is rated as: 1=very much improved; 2=much improved; 3=minimally improved; 4=no change; 5=minimally worse; 6=much worse; or 7=very much worse.

Data abstraction—The following data were obtained from each study: country, total number of randomised patients, baseline demographic data (age, gender), mean Mini-Mental Status Examination score (MMSE), diagnosis, setting, duration of study, quetiapine daily dose, concurrent use of cholinesterase inhibitors (CEIs), primary indication to enter the study, primary and other outcome measures, mean total NPI score, mean change from baseline of the total NPI score for both the quetiapine and placebo groups (and the level of significance for the mean difference in NPI between the two groups), mean CGI-C scores at the end of the study period for both the quetiapine and placebo groups (and the level of significance for the two groups).

Quantitative Data Analysis—We used Review Manager Software Version 5 (<http://www.cc-ims.net/revman>) to calculate the effect size and confidence interval (CI) of each individual study and the combined results. The average effect size across all studies is computed as a weight mean. We consulted a biostatistician from the Department of Population Health, University of Auckland, for the data analysis.

Results

Our search strategies yielded a total of six double-blinded RCT studies comparing quetiapine with placebo.²²⁻²⁷ One study (Ballard et al) was excluded because it did not have NPI or CGI-C as their outcome measures.

The study published by Zhong et al presented separate data for the two dosages of quetiapine (100mg daily and 200mg daily) used in the trial. An overall result for the two dosages was not published. We have therefore a total of six sets of data (from 5 studies) included in this meta-analysis (see Tables 1 and 2).

Table 1. Baseline variables of the included studies

Study	N	Mean Age (Overall)	% Female (Overall)	Mean MMSE (Quetiapine Group)	Mean MMSE (Placebo Group)	Total NPI Score (Quetiapine Group)	Total NPI Score (Placebo Group)
Schnieder et al 2006	421	77.9	56	14.9	14.7	37.6	39.1
Tariot et al 2006	284	83.2	73	12.4	13.2	39.1	35.7
Kurlan et al 2007	40	73.8	37.5	19.2	17.2	25.1	25.9
Zhong et al 2007	333	83.2	74.2	100mg=4.8 200mg=5.6	5.5	100mg=38.5 200mg=34.9	35.7
Paleacu et al 2008	40	82.2	65	14.5	14.3	43.4	38.6

N=Total number of participants underwent randomisation; NR=Not reported

The included studies were conducted among of heterogenous group of participants with moderate to severe dementia (mean MMSE ranged from 5.5 to 17.2) from different settings (outpatient=1, residential=2, not reported =3). Three studies included only Alzheimer's dementia, one study included both Alzheimer's and vascular dementia and one study included dementia with Lewy bodies, Parkinson's disease with dementia and Alzheimer's dementia with parkinsonism.

The length of the studies ranged from 6 to 12 weeks. Various dosages of quetiapine were used in the five studies (daily dose of quetiapine ranged from 0 to 600mg). NPI was used as the primary outcome in one study (Paleacu et al). CGI-C was used as the primary outcome in two studies (Schnieder et al & Paleacu et al). The mean baseline NPI scores ranged from 25.1 to 43.4.

Participants who completed the entire duration of each study were included in the meta-analysis.

Figure 1 showed the effects of quetiapine on the total NPI among patients with moderate to severe dementia. Patients receiving quetiapine improved on the NPI score when compared to placebo with a weighted mean difference (WMD) of - 3.05 (95% CI: -6.10, -0.01).

Figure 2 showed the CGI-C scores in the quetiapine and placebo groups. Patients receiving quetiapine improved on the CGI-C score when compared to placebo with a weight mean difference (WMD) of -0.31 (95%CI: -0.54, -0.08).

There was no evidence of heterogeneity across the studies (NPI: p-value=0.78; CGI-C: p-value=0.37).

Table 2. Description of the included studies

Study	Countries	Diagnosis	Setting	Duration	Quetiapine daily dose	Concurrent use of CEIs	Primary indication	Primary outcome	Other outcome measures
Schnieder et al 2006	USA	AD	Outpatient	12 weeks	Mean=56.5mg Range=0-100mg	No	Psychosis, aggression or agitation	Time to discontinuation of treatment	CGI-C, NPI, BPRS
Tariot et al 2006	USA	AD	Residential	10 weeks	Medium=96.9mg Range=25-600mg	12.1% quetiapine group 10.1% Placebo group	Psychosis	BPRS, CGI-S	CGI-C, NPI-NH2
Kurlanl et al 2007	USA	DLB, PD with dementia, AD with Parkinsonism	NR	10 weeks	Mean=120mg Range=25-300mg	45% quetiapine group 70% Placebo group	Psychosis or agitation	BPRS	CGI-C, NPI, ADCS ADL
Zhong et al 2007	USA	AD, VD	Residential	10 weeks	Fixed dosing 100mg or 200mg	39% 100mg group 32% 200mg group 34% Placebo group	Agitation	PANSS-EC	CGI-C, CMAI, NPI-NH
Paleacu et al 2008	Israel	AD	NR	6 weeks	Medium=200mg Range=75-300mg	32% overall	A score >6 on any NPI items	NPI, CGI-C	

AD=Alzheimer's disease; ADCS-ADL=Alzheimer's Disease Cooperative Study Activity of Daily Living Questionnaire; BPRS=Brief Psychiatric Rating Scale; CEIs=Cholesterase inhibitors; CGI-C=Clinical Global Impression of Change; CGI-S=Clinical Global Impression-Severity of Illness; CMAI=Cohen-Mansfield Agitation Inventory; DLB=Dementia with Lewy Bodies; NPI=Neuropsychiatric Inventory; NPI-NH=Neuropsychiatric Inventory – Nursing Home version; NR=Not reported; PANSS-EC=Positive and Negative Syndrome Scale – Excitement Component; PD=Parkinson disease; SIB=Severe Impairment Battery; VD=Vascular dementia.

Figure 1. Neuropsychiatric Inventory (NPI)

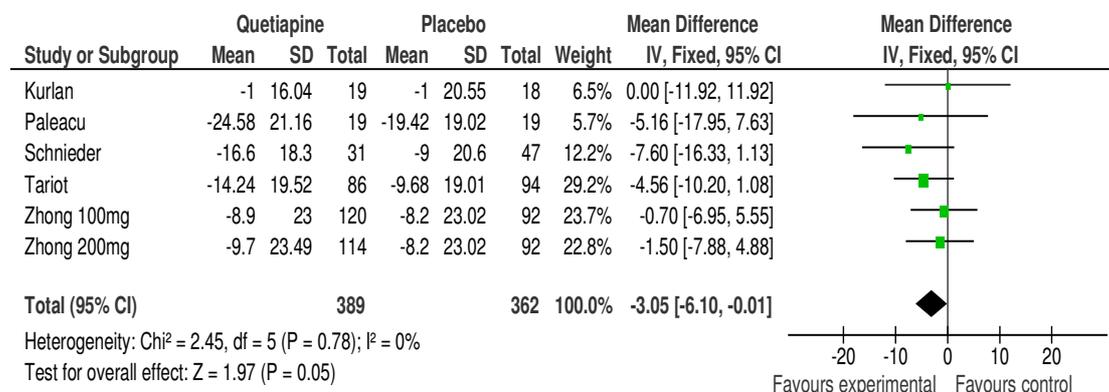
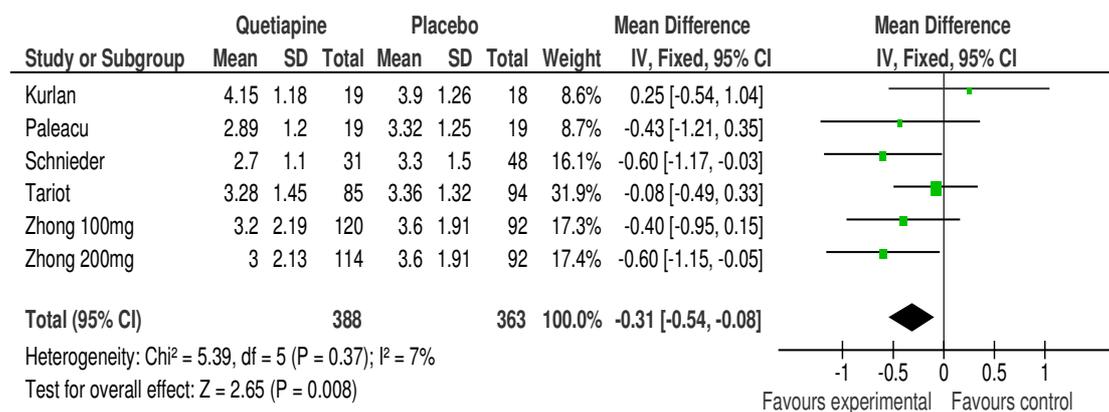


Figure 2. Clinical Global Impression of Change Scale (CGI-C)



The study by Paleacu et al is a significant outlier in terms of the effectiveness (as measured by the NPI) in both of the placebo and quetiapine groups. This study had the highest baseline total NPI score for the quetiapine group (43.4) and the shortest trial period (6 weeks).

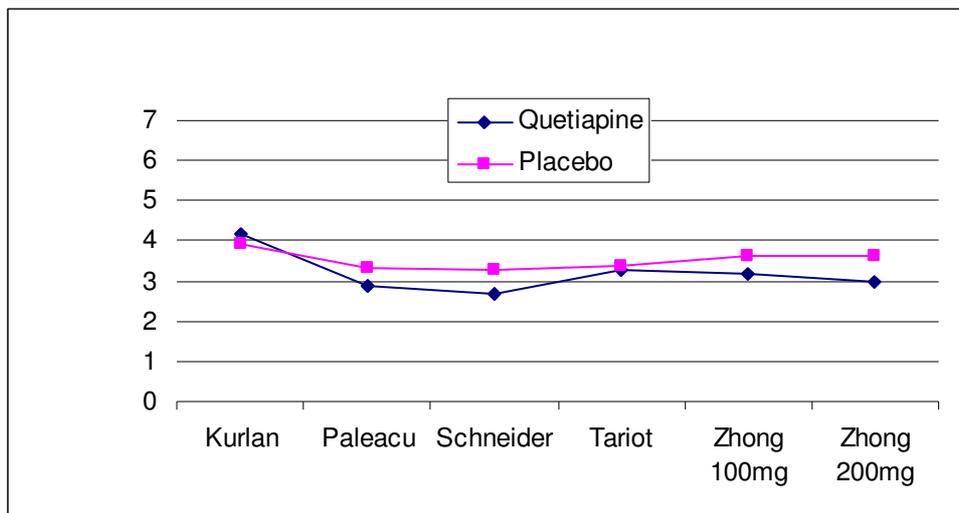
Discussion

This meta-analysis found that quetiapine is statistically more efficacious than placebo in the treatment of BPSD as measured by the NPI and CGI-C. However, the mean weight difference between the quetiapine and placebo groups was just over 3 points on the NPI. Previous authors have defined clinical response as a minimum change of 4 points (Mega et al) and 9 points (Kaufer et al) on the NPI.^{28,29} The improvement on CGI-C is also of a small magnitude (0.3 point).

Figure 3 showed the CGI-C scores in the quetiapine and placebo groups in the 5 studies. CGI-C is a 7-points scale with a one-point difference between each anchor

point. We believe it would be difficult to observe the 0.3 difference clinically. These results suggest that although there is a statistical evidence for quetiapine in the treatment of BPSD, observable clinical significance is questionable.

Figure 3. CGI-C scores (at the end of the trials) in the quetiapine and placebo groups



CGI-C: 1=very much improved; 2=much improved; 3=minimally improved; 4=no change; 5=minimally worse; 6=much worse; or 7=very much worse.

This meta-analysis also suggested that placebo had a comparative clinical effect as quetiapine in the treatment of BPSD. This finding could be a result of the non-specific benefits obtained from the increased attention patients received during the trial. This in itself has an important clinical message that non-pharmacological management including increased attention should always be included in the overall management of BPSD.

In New Zealand, quetiapine and risperidone are the only two listed atypical antipsychotic medications which do not require special authority application from a specialist psychiatrist. Risperidone was the first atypical antipsychotic medication reported to have an increased mortality and morbidity with cerebrovascular events when it is prescribed for the treatment of BPSD.

There is a perception that other atypical antipsychotic medications are “safer”. However, we now have evidence to suggest that such risks are common to both typical and atypical antipsychotic medications. In a retrospective cohort study involving 22,890 patients 65 years of age or older, Wang et al have found typical antipsychotic medications were at least as likely as atypical agents to increase the risk of death.³⁰

Schneider et al reported a significant increase in mortality, but there was no difference between specific atypical agents in a meta-analysis of 16 studies of atypical antipsychotics for the treatment of BPSD.³¹ In addition, among the atypical

antipsychotic medications quetiapine was found to have the highest risk for overall mortality in a 11-year follow up cohort study of patients with schizophrenia in Finland.³²

Haloperidol has been compared to risperidone and quetiapine in the treatment of BPSD.^{24,33–35} Only one of the four trials found a significantly greater efficacy with the atypical than with the typical agent. However, in all four studies, haloperidol was associated with more extrapyramidal symptoms than the atypical agent. We believe that with the current knowledge of antipsychotic medications risperidone should be prescribed as the first-line treatment of BPSD in primary care setting and this is consistent with the guideline published by the Faculty of Old Age Psychiatry of the Royal Australian and New Zealand College of Psychiatrists.³⁶

The use of olanzapine and aripiprazole can also be considered. However, a risk-benefit analysis should be performed for each individual before an atypical antipsychotic medication is prescribed for the treatment of BPSD. Atypical antipsychotic medications should only be prescribed for people with dementia presenting with aggression, agitation or psychosis which is associated with severe distress or risk of physical harm to those living and/or working with the patient.

In addition to the treatment of cognitive deficits in dementia, cholinesterase inhibitors (donepezil, galantamine, rivastigmine) have been used in the treatment of BPSD. Campbell et al recently published a meta-analysis to determine the efficacy of cholinesterase inhibitors in the treatment of BPSD in patients with mild to severe Alzheimer's disease.³⁷

They found patients who were prescribed with cholinesterase inhibitors improved by 1.38 point on the NPI as compared to the placebo group. Although the reduction in NPI was statistically significant, the authors concluded that the clinical relevance of this effect remains unclear.

Since 2010 donepezil has been fully subsidised by the prescription funding agency in New Zealand, Pharmac, for the treatment of Alzheimer's disease. The use of cholinesterase inhibitors can be considered as an alternative treatment for BPSD if there is significant risk involved in prescribing antipsychotic medications, for example, cerebrovascular events, falls and extrapyramidal side effects.

There is also some evidence for the use of citalopram in the treatment of BPSD.³⁸⁻⁴⁰ This medication is useful if there is a comorbid depression (which is not uncommon in dementia) and a rapid onset of action is not required. Other psychotropic medications can be considered for the treatment of BPSD includes memantine and carbamazepine.¹¹

Benzodiazepines are often prescribed by non-psychiatrists for the treatment of BPSD. Jeste et al identified two trials of benzodiazepines for BPSD in their literature search.⁴¹ Alprazolam was found to be as effective as low dose haloperidol in a randomised, double-blind crossover study.⁴²

Coccaro et al reported oxazepam, haloperidol and diphenhydramine appeared to have a similar efficacy for short-term management behaviour in severely demented patients.⁴³ However, benzodiazepines should be cautiously used in older people

because of their adverse side effects including sedation, drowsiness, confusion, falls and their long-term effects of tolerance and dependence.

Quetiapine has an advantage over other atypical antipsychotic medications (except clozapine) in terms of extrapyramidal side effects and has become a favourable choice for the treatment of psychosis in Parkinson's disease and BPSD in Lewy Body Dementia and Parkinson's disease with dementia. However, previous RCTs found quetiapine has no benefit in the treatment of psychosis in Parkinson's disease.⁴⁴⁻⁴⁶

The study by Kurlan et al (which was included in this meta-analysis) was specifically designed to determine the efficacy of quetiapine in the treatment of BPSD in Lewy Body Dementia, Parkinson's disease with dementia and Alzheimer's disease with parkinsonism. There is no evidence for quetiapine in the treatment of psychosis or agitation as measured by the Brief Psychiatric Rating Scale, CGI-C and NPI.

In contrast there have been two randomised placebo-controlled trials with clozapine in the treatment of psychosis in Parkinson's disease and both studies showed significant improvements in psychopathology with no or minimal worsening in motor symptoms of Parkinson's disease.^{47,48} The efficacy of rivastigmine on BPSD in Lewy Body Dementia has also been reported.⁴⁹ McKeith et al found patients taking rivastigmine were significantly less apathetic and anxious, and had fewer delusions and hallucinations than controls.

In summary, the currently available literature does not support the use of quetiapine in the treatment of BPSD and more efficacious medication such as risperidone and medication with less risk such as cholinesterase inhibitors, citalopram, memantine and carbamazepine are available.

Clozapine has the most evidence in the treatment of psychosis in Parkinson's disease and rivastigmine can be considered for the treatment of BPSD in Lewy Body Dementia. With the exception of risperidone, general practitioners may not have the expertise prescribing the other medications mentioned here and guidance from the specialists will be required. In addition, clozapine can only be initiated by psychiatrists and there is a central registration programme for the monitoring of agranulocytosis. We like to highlight that the advice given in this article applies primarily in the New Zealand context, although the evidence is based on international research.

The main limitation of this study is that the individual domains of NPI were not analysed separately. Only the study by Tariot et al reported the data on the individual domains and they found no significant advantage with quetiapine in the NPI domain scores for agitation, hallucinations and delusions. All studies, except the one by Paleacu et al, included agitation and/or psychosis as their primary indications (Table 2).

It would be useful to obtain the original data from each of the study to perform further analysis on the NPI delusions, hallucinations and agitation/aggression domains. The failure to report the individual domains of NPI in the literature also raises issues of obscuring the effectiveness of some agents for specific targeting symptoms (e.g. agitation, aggression and psychosis) and masking the potential harms of some agents for other symptoms (such as apathy from antipsychotic medications).

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Narcolepsy in New Zealand: pathway to diagnosis and effect on quality of life

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Abstract

Aims There has been no attempt to survey New Zealanders with narcolepsy to determine their pathway to diagnosis, symptoms, treatment, or quality of life. We therefore aimed to develop a comprehensive questionnaire, and compare responses on measures of daytime sleepiness and quality of life between individuals with narcolepsy and the general New Zealand population.

Methods A questionnaire was developed encompassing descriptive information, daytime sleepiness and sleep habits, general health and wellbeing, diagnosis and treatment of narcolepsy, symptoms, and quality of life. Ninety-two individuals were identified through medical specialists and a local support group.

Results Complete responses were obtained from 54 individuals (63% female, mean age 54.7 ± 18.3 years). The mean Epworth Sleepiness Scale score was 16.4 ± 5.4 (/24). Symptoms first appeared at 20.7 ± 9.7 years of age on average, although diagnosis did not take place until 33.4 ± 13.8 years of age. Individuals with narcolepsy reported substantially lower health-related quality of life than the general New Zealand population. Less than half of those diagnosed with narcolepsy had undergone an objective evaluation including a sleep study.

Conclusions New Zealanders with narcolepsy suffer from an excessive level of daytime sleepiness, and have significantly poorer health-related quality of life than the general population. There are a number of inconsistencies between the diagnostic pathway in New Zealand and best-practice guidelines for diagnosis and treatment.

Narcolepsy is a neurological sleep disorder characterised by excessive daytime sleepiness either with or without cataplexy (a sudden episode of muscle weakness often triggered by emotional reactions such as laughter, elation, surprise or anger¹). Auxiliary symptoms include automatic behaviour, disrupted night-time sleep, sleep paralysis and hypnagogic/hypnopompic hallucinations.¹

The prevalence of narcolepsy is difficult to reliably establish, given the changes in diagnostic criteria over time and the practical concerns with accurately screening large numbers of the general population.² However, international research suggests that the prevalence of narcolepsy lies between 0.16% and 0.66%.² No data is available for the Australasian population. Although the aetiology of narcolepsy remains largely unknown, a genetic component has been established initially focusing on the human leukocyte antigen (HLA) complex.³ More recently, it has been suggested that a loss of hypocretin/orexin cells in the hypothalamus can lead to narcolepsy, as these cells play a major role in the ability to maintain wakefulness.⁴

The primary symptoms of narcolepsy—excessive daytime sleepiness and cataplexy—have a major and widespread impact on the quality of life of sufferers.⁵ Individuals with narcolepsy have attributed poor grades at school, impaired job performance, interpersonal problems, and a greater susceptibility to driving accidents to their symptoms.⁶

Recent studies have reported the average time between the onset of symptoms and diagnosis as being at least 16 years,^{7,8} and narcolepsy is particularly susceptible to mis-diagnosis.^{9,10} Since the first symptoms of narcolepsy usually appear during late adolescence,¹¹ most individuals with narcolepsy are diagnosed too late in life to prevent the dramatic impact of the disease on their personal and professional development.

A provisional diagnosis of narcolepsy is straightforward if all of the aforementioned symptoms are present, however more often the symptoms of dissociated rapid eye movement sleep (REM; cataplexy, hallucinations, paralysis) are mild and up to 50% of patients do not present with all symptoms.¹² The only validated diagnostic test consists of an overnight polysomnography (PSG) followed immediately by a Multiple Sleep Latency Test (MSLT).¹³ The overnight PSG ensures an objective measurement of total sleep time and sleep quality on the night before the MSLT, and also allows the exclusion of other possible or additional causes of excessive daytime sleepiness such as obstructive sleep apnoea.

A diagnosis of narcolepsy is confirmed if the MSLT shows a mean sleep onset latency of <8 minutes, and in particular, the occurrence of two or more sleep onset REM periods.^{1,14} Guidelines state that the PSG/MSLT protocol is highly desirable but not mandatory for a diagnosis, and therefore it is possible for patients to receive a diagnosis of narcolepsy based on clinical review, particularly in the presence of cataplexy.¹

Once a diagnosis is confirmed, individuals with narcolepsy can benefit from pharmacological treatment/s to control daytime sleepiness, cataplectic attacks, disrupted sleep, hallucinations and/or sleep paralysis. Current practice parameters list stimulants (modafinil/armodafinil, sodium oxybate, amphetamine, methamphetamine, dextroamphetamine, methylphenidate) and anti-depressant agents (selegiline, ritanserin, tricyclic antidepressants, selective serotonin reuptake inhibitors, venlafaxine and reboxetine) as potentially effective treatments of narcolepsy.^{15,16}

Of these, modafinil and sodium oxybate have become the treatments of choice internationally due to lower tolerance (tolerance to a drug refers to a diminished physiological response whereby a patient requires a larger dose for the same effect), longer half-life and lower potential for abuse compared with the sympathomimetic stimulants,^{17,18} however modafinil has no effect on cataplexy¹⁷ and therefore REM-suppressant anti-depressants are often prescribed alongside modafinil despite there being little evidence of their efficacy for this purpose.¹⁹ The combination of drugs prescribed must be tailored for each individual and long-term follow-up is crucial in order to monitor the response to treatment as well as potential dependence, tolerance, abuse or side-effects.^{16,20}

Not only is the prevalence of narcolepsy in New Zealand unknown, but to our knowledge there has been no attempt to survey New Zealanders with narcolepsy to

determine their pathway to diagnosis, symptoms, treatment, or the effects of symptoms on quality of life.

This study aimed to address each of these areas using a comprehensive questionnaire, compiling descriptive information, and comparing measures of daytime sleepiness and quality of life to data available for the general New Zealand population.^{21,22}

Methods

Ethics—Ethical approval was granted by the Multi-Region Ethics Committee prior to commencement (MEC/05/02/020).

Questionnaire development—The questionnaire was developed by the researchers with input from members of the Narcolepsy Support Group of New Zealand, and included 6 sections:

- Demographic information (age, gender, body mass index (BMI), ethnicity, education and qualifications, paid and unpaid employment);
- Daytime sleepiness, sleep quantity and quality (Epworth Sleepiness Scale (ESS),²³ and questions regarding total sleep time, napping, arousals and subjective sleep quality);
- General health and wellbeing (questions relating to ability to concentrate, get things done, cope with problems, relationships with family/friends, physical wellness and general quality of life, as well as the complete SF-36 Health Survey²⁴);
- Diagnosis and treatment of narcolepsy (modified/extracted from the Australian Narcolepsy Survey (D Bruck, personal communication, 2005), and two questions from the Stanford Centre for Narcolepsy Sleep Inventory²⁵ relating to the method of diagnosis);
- Symptoms of narcolepsy (occurrence and age of onset of vivid dreams, sleep paralysis and cataplexy);
- Effects of narcolepsy on quality of life (questions relating to whether narcolepsy has caused difficulties in everyday activities and communicating/socialising).

Recruitment and questionnaire distribution—Questionnaires were sent to every current member of the Narcolepsy Support Group of New Zealand via an intermediate person in order to maintain confidentiality. To identify individuals with narcolepsy who were not members of this group, sleep physicians, neurologists and sleep laboratories throughout New Zealand were also supplied with questionnaires to distribute to all patients who had confirmed or suspected narcolepsy.

Each questionnaire was assigned a code to ensure that the origin of each returned questionnaire could be identified (support group member, sleep physician, neurologist or laboratory patient). Respondents were able to complete the written questionnaire and return it in a reply-paid envelope, or they could call a toll-free number to complete the questionnaire verbally. When a reply by either method was not received after six weeks a reminder letter was sent, and after a further six weeks remaining non-responders were sent a second copy of the questionnaire.

Data handling and statistical analysis—All questionnaire data were double-entered and analysed using SPSS (Version 13.0; IL, USA) software. Continuous data were analysed for normality of distribution and homogeneity of variance where appropriate. Correlations were analysed using Pearson or Spearman tests. Between-group data were analysed using independent-samples t-tests or Mann-Whitney U tests. Categorical data were analysed using Pearson Chi-squared tests. All statistical tests were considered significant when $p \leq 0.05$.

Results

Response rate—92 questionnaires were distributed—68 (74%) were sent via the Narcolepsy Support Group; the remaining were sent via neurologists or sleep physicians. No respondents were identified through a sleep clinic/laboratory.

A response was obtained from 67 individuals (70%), and of these, 54 questionnaires were complete or near-complete (59% of the total surveys sent, or 81% of those who responded). Two questionnaires were completed verbally over the phone.

Forty-one of the complete/near-complete questionnaires (76%) came from members of the Narcolepsy Support Group. The return rates for the two groups were markedly different: 75% of the Narcolepsy Support Group members surveyed responded, whereas only 38% of individuals approached through a specialist responded.

Descriptive characteristics of the study sample are contained in Table 1. There was no significant difference in age between males and females ($p>0.05$). The respondents from the Narcolepsy Support Group were significantly older than those who responded via a specialist (58 ± 16 and 44 ± 22 years respectively; $p=0.01$).

The majority of respondents were of New Zealand European ethnicity, and less than half were in paid employment.

Table 1. Descriptive characteristics of the study sample

Descriptive characteristic		Number (\pm SD or % or range)
Number of females (n=54)		34 (63.0%)
Mean age (years) (n=54)		54.7 \pm 18.3; range 18-86
Mean BMI (kg/m ²) (n=47)		28.2 \pm 5.8
Ethnicity (n=54)	NZ European	50 (92.6%)
	Māori	1 (1.9%)
	Other	3 (5.5%)
Completed high school (n=46)		45 (97.8%)
If yes, obtained further qualifications (n=45)		32 (71.1%)
Receive income (n=54)	Paid work	25 (46.3%)
	Superannuation / Pension	18 (33.3%)
	Government benefits	14 (25.9%)
Mean hours of work per week in primary job (n=25)		40.4 \pm 20.3
Mean hours of work per week in all other jobs (n=17)		3.1 \pm 9.7

Daytime sleepiness, sleep quantity and quality—As shown in Table 2, the average level of daytime sleepiness was high, although respondents scored along the full range of ESS scores. The mean ESS of our study sample (16.4 ± 5.4) was significantly higher than the mean of 6.0 ± 3.9 reported in the general population of non-Māori New Zealanders ($p<0.0001$),²² and 4.6 ± 2.8 in an Australian sample of normal sleepers ($p<0.0001$).²⁶

Three-quarters of our sample reported napping during the day and excluding those who did not nap, the mean number of naps per week was 8 ± 5.7 (range 2-28).

There was a significant relationship between difficulty falling asleep and frequency of waking during the night ($r_s=0.441$, $p<0.001$), as well as difficulty getting back to sleep if one woke during the night ($r_s=0.680$, $p<0.001$). There were no significant relationships between napping during the day and any of the sleep variables measured, including waking feeling refreshed (Spearman's correlations, all $p>0.05$).

Table 2. Daytime sleepiness, sleep quantity and quality

Sleepiness variable		Number (\pm SD or % or range)
Mean Epworth sleepiness scale (/24) (n=53)		16.4 \pm 5.4 (range 1–24)
Mean total sleep time per 24-hrs (hours:mins) (n=53)		7:57 \pm 02:00 (range 4:30–16:00)
Mean bed-time (n=54)		10:26pm
Mean rise-time (n=54)		6:50am
Mean time in bed overnight (hours:mins) (n=54)		08:26 \pm 01:32 (range 00:50–12:30)
Mean sleep latency after lights out (hours:mins) (n=52)		0:08 \pm 00:13 (range 00:00–01:00)
Mean number of naps per week (n=54)		6.3 \pm 6.1 (range 0–28)
Mean duration of typical nap (hours:mins) (n=41)		00:40 \pm 00:32 (range 00:01–02:00)
Have difficulty falling asleep (n=54)	Never / Rarely	43 (79.6%)
	Often / Always	11 (20.4%)
Awakenings per night (n=54)	Never / 1–2 times	20 (37.0%)
	3+ Nights	34 (63.0%)
Wake feeling refreshed (n=53)	Never / Rarely	30 (56.6%)
	Often / Always	23 (43.4%)

General health and wellbeing—Most respondents reported as ‘good’ or ‘excellent’ their ability to concentrate (69.8%), get things done (62.3%), cope with minor problems (84.9%), relationships with family and/or friends (88.7%), physical wellness (69.8%) and general quality of life (69.8%) (Table 3).

Table 3. General health and wellbeing

In general, how would you rate yourself on the following (based on the last 4 weeks)...	Poor (N; %)	Fair (N; %)	Good (N; %)	Excellent (N; %)
Ability to concentrate (n=53)	4 (7.5%)	12 (22.6%)	30 (56.6%)	7 (13.2%)
Ability to get things done (n=53)	4 (7.5%)	16 (30.2%)	24 (45.3%)	9 (17.0%)
Ability to cope with minor problems (n=53)	1 (1.9%)	7 (13.2%)	28 (52.8%)	17 (32.1%)
Relationships with family and/or friends (n=53)	1 (1.9%)	5 (9.4%)	26 (49.1%)	21 (39.6%)
Physical wellness (n=53)	8 (15.1%)	8 (15.1%)	25 (47.2%)	12 (22.6%)
General quality of life (n=53)	5 (9.4%)	11 (20.8%)	25 (47.2%)	12 (22.6%)

Table 4 summarises the eight SF-36 subscales compared with data reported elsewhere for the general New Zealand population²¹; patients with narcolepsy reported significantly lower scores on all sub-scales except bodily pain and mental health.

Respondents who had received an objective evaluation of narcolepsy, including overnight PSG and daytime MSLT, scored significantly lower on vitality (34.13 \pm 25.26 versus 48.04 \pm 23.54; p=0.05) and mental health (68.04 \pm 18.75 versus 79.46 \pm 14.22 p=0.02) compared to those who had not.

Table 4. SF-36 Health Survey responses compared with data from the general New Zealand population

SF-36 subscale	Mean±SD Current Study	Mean±SD NZ Data ²¹	P value (1-sided)
Physical Functioning (/100) (n=54)	68.6±32.4	86.0±26.3	<0.01
Role Physical (/100) (n=53)	51.4±40.3	80.7±52.6	<0.01
Bodily Pain (/100) (n=53)	73.3±26.0	77.9±35.0	0.17
General Health (/100) (n=52)	59.7±13.7	73.8±26.3	<0.01
Vitality (/100) (n=52)	41.8±24.8	65.6±26.2	<0.01
Social Functioning (/100) (n=53)	72.6±23.0	86.6±26.4	<0.01
Role Emotional (/100) (n=51)	69.9±37.8	85.0±43.5	<0.01
Mental Health (/100) (n=52)	73.8±17.4	78.0±26.2	0.12

Diagnosis and treatment of narcolepsy—Survey respondents reported their first symptomatic experience of narcolepsy as occurring, on average, in their early 20s. Diagnosis was on average 12 years later, with this diagnosis coming from a range of medical practitioners (sleep specialists 33%, general practitioner 18.5%, other specialist/consultant 11.1%, neurologist 7.4%). Less than half of those who had a diagnosis of narcolepsy had undergone a daytime sleep study (MSLT) as part of their diagnostic procedure.

The majority of members of the Narcolepsy Support Group had not undergone a sleep study of any description (70.7%), although 55.2% of this group take stimulant medication. On average, the study sample had discussed their symptoms with 2.2 other doctors prior to receiving a diagnosis (range 1–23 doctors), within a wide range of specialties—neurology (including epilepsy, chronic fatigue syndrome and neuralgia), psychology, psychiatry, endocrinology, and cardiology.

Table 5. Diagnosis and treatment of narcolepsy

Diagnosis / Treatment Variable		Number (±SD or % or range)
Mean age of symptom onset (years) (n=52)		20.7±9.7 (range 12-68)
Medical diagnosis (by any means) received (n=53)	Yes	47 (88.7%)
	If so, age (years) (n=41)	33.4 (13.8%)
Previously discussed symptoms with medical professional (n=48)	Yes	42 (87.5%)
	If so, number of times (n=46)	2.2 (4.0%) (range 1-23)
Undergone overnight sleep study (e.g. PSG) (n=53)		24 (45.3%)
Undergone daytime sleep study (e.g. MSLT) (n=52)		21 (40.4%)
Symptoms experienced at diagnosis	Sleepiness (n=53)	51 (96.3%)
	Loss of muscle power (n=53)	35 (66.0%)
	Sleep paralysis (n=53)	23 (43.4%)
	Hallucinations (n=53)	18 (34.0%)
	Disturbed dreams (n=53)	31 (58.5%)
	Frequent awakenings (n=53)	31 (58.5%)
	Loud snoring (n=51)	13 (25.5%)
	Inability to breathe during sleep (n=53)	7 (13.2%)
	Sleeping for >12 hours at a time (n=53)	11 (20.8%)
Other (n=54)		13 (24.1%)
Other diagnosed sleep disorder (n=52)		3 (5.8%)
Other long-term illness (n=53)		19 (35.8%)
Taking medication/s for narcolepsy or other medical condition (n=54)		44 (81.5%)

All respondents who were diagnosed via a sleep study reported experiencing daytime sleepiness, tiredness or fatigue at the time of diagnosis. All respondents who had their diagnosis without an overnight sleep study reported an inability to breathe while asleep during the four weeks prior to diagnosis with narcolepsy.

Three respondents (5.8%) had been diagnosed with obstructive sleep apnoea and had received treatment with either continuous positive airway pressure (CPAP) or surgery. Other long-term illnesses were reported by approximately one third of respondents, and included asthma, hypertension, cardiac problems, diabetes and mental illness.

Eighty-one percent of respondents were taking medication at the time the questionnaire was completed. Sixty-three percent were taking medication specifically for narcolepsy, while an additional 10 respondents (18.5%) were taking anti-depressant medication (fluoxetine, paroxetine or chlorthalidone/clonidine) without other medication specific to narcolepsy.

Stimulant medication (methylphenidate, dexamphetamine or modafinil) was taken by 29.6% of respondents despite not having received an objective diagnosis of narcolepsy via a sleep study, although all had received a diagnosis of some description.

Comparing respondents who were taking stimulants and those who were not, there were no significant differences in ESS, time spent in bed, bedtime, rise time, difficulty falling asleep, frequency of waking at night, difficulty getting back to sleep, waking refreshed, napping during the day, number of naps per week and nap length (all $p > 0.05$). There were also no significant differences in any of the General Health & Wellbeing questions (Table 3), Symptoms of Narcolepsy questions (Table 6) or Quality of Life questions (Table 7).

Those taking stimulants scored significantly lower than the rest of the sample on the SF-36 bodily pain subscale (68.09 ± 25.89 and 82.63 ± 24.12 respectively, $p = 0.05$) and vitality subscale (36.21 ± 25.19 and 51.58 ± 21.48 respectively, $p = 0.03$).

Symptoms of narcolepsy—Over half of the sample had experienced cataplexy when laughing and vivid dreams during the four weeks prior to completing the questionnaire. Other triggers of cataplexy were less common. Average onset of sleep paralysis, cataplexy and/or vivid dreaming was similar for all three symptoms at 22-26 years of age (see Table 6).

Table 6. Symptoms of narcolepsy

Symptom variable		Number (\pm SD or % or range)
Experienced vivid dreams (based on last four weeks) (n=51)		30 (58.8%)
Experienced sleep paralysis (based on last four weeks) (n=51)		8 (15.7%)
Experienced cataplexy when... (based on last 4 weeks)	Laughing (n=53)	29 (54.7%)
	Angry (n=53)	14 (26.4%)
	Excited (n=53)	18 (34.0%)
	Stressed (n=53)	14 (26.4%)
	Other (n=53)	13 (24.5%)
Mean age at first episode of cataplexy (years) (n=39)		25.8 \pm 11.6 (range 9-56)

Effects of narcolepsy on quality of life—Just over half of the study sample indicated that narcolepsy prevented them from doing everyday activities or caused difficulty in doing everyday activities (Table 7). A higher percentage indicated that narcolepsy prevented them from communicating, mixing, or socialising with others. Almost a third of respondents had left school or a job due to narcolepsy, and nearly three quarters had injured either themselves or someone else as a result of narcolepsy. Both of these results were independent of age or whether a participant had received an objective diagnosis via a sleep study (both $p > 0.05$).

Table 7. Effects of narcolepsy on quality of life

Quality of life variable		Number (%)
Narcolepsy causes difficulty/prevents...	Everyday activities (n=54)	28 (51.9%)
	Communicating/socialising (n=54)	37 (68.5%)
	Other activities (n=51)	22 (43.1%)
Other health problem causes difficulty/prevents...	Everyday activities (n=54)	14 (25.9%)
	Communicating/socialising (n=54)	10 (18.5%)
	Other activities (n=53)	10 (18.9%)
Dropped out of school or left a job due to narcolepsy (n=54)		17 (31.5%)
Participant or someone else injured due to narcolepsy (n=52)		12 (23.1%)
Failed to do what was normally expected due to narcolepsy (n=42)		13 (31.0%)

Discussion

The diagnosis of narcolepsy in New Zealand appears to be performed for the most part via clinical review by a wide range of medical professionals, with slightly less than half of the respondents in our sample undergoing a sleep study (overnight PSG and/or MSLT).

In our sample, symptoms appeared on average at 21 years of age although the mean age of diagnosis was 33, and the most common initial symptoms were daytime sleepiness, loss of muscle power, disturbed dreams and frequent awakenings. This is consistent with the international literature.^{5,11,27}

Our data indicates that the quality of life of individuals with narcolepsy in New Zealand is poor. The mean ESS was very high compared with that reported by non-Māori New Zealanders²² and the general Australian population,²⁶ despite a healthy duration of sleep reported overnight (mean 7:57±2:00 hours) and a high number of naps reported per week. This is consistent with the description of narcolepsy.²⁸

Although most respondents reported 'good' or 'excellent' on a number of general health and wellbeing questions, health-related quality of life was substantially reduced compared with that of the New Zealand population.²¹ Less than half of our sample was in paid employment, and nearly a quarter of respondents had left school or a job due to narcolepsy.

The results of this study highlight some important points regarding diagnosis and treatment of narcolepsy. Only 63% of the respondents were taking medication for

narcolepsy, despite the fact that the mean ESS of the study sample indicated excessive daytime sleepiness. Thirty percent of respondents were taking prescription stimulant medication despite not having received an objective evaluation of narcolepsy. Interestingly, all of the respondents who had been diagnosed by means other than a sleep study reported an inability to breathe while asleep during the month prior to diagnosis, which suggests that these respondents may have been suffering from obstructive sleep apnoea, either instead of or in addition to narcolepsy.

There are clearly a number of differences between the diagnostic pathways for narcolepsy in New Zealand and best-practice guidelines,¹³ and inconsistencies in treatment pathways in the sample studied.^{15,16} This is likely due to an absence of physicians specialising in sleep in general and narcolepsy in particular at the time the respondents were diagnosed (on average, over twenty years ago). Although services have improved markedly over the last few decades in this relatively young field of medicine, there is still significant variability in sleep services between the different District Health Boards in New Zealand.

A limitation of this study was the relatively small sample size, due to both the small target population and the reasonably low questionnaire completion rate (59% complete or near-complete questionnaires received), which raises the likelihood of non-responder bias. In order to maintain patient confidentiality we were reliant on physicians mailing out a large portion of the questionnaires, and these physicians were also responsible for following up individuals who did not respond.

Many physicians did not have a database or method to track the patients under their care who had been diagnosed with narcolepsy. Additionally, we did not target patients who were diagnosed by a general practitioner. Hence, the majority of our sample was made up of members of the Narcolepsy Support Group (76%), and our results therefore reflect a group who are probably more motivated and pro-active than those in the general population with narcolepsy.

Narcolepsy is under-diagnosed,²⁷ and results in a high economic burden due to both direct costs (hospital admission, treatment, ambulatory care) and indirect costs (sick leave, shortened working hours, early retirement).²⁹ An increase in public and medical awareness of narcolepsy is vital so that up-to-date diagnostic and treatment pathways are available and utilised.

Our data indicate an excessive level of daytime sleepiness and poor health-related quality of life of individuals with narcolepsy in New Zealand, and suggests that best-practice guidelines for the diagnosis and treatment of narcolepsy^{13,15,16} are not closely followed.

In recent years medication to treat narcolepsy has evolved and will likely continue to evolve. Regular follow-up and education of all patients with narcolepsy is therefore crucial in order for physicians and patients to make appropriate choices to improve the quality of life of individuals with narcolepsy in New Zealand and elsewhere.

Competing interests: None.

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Patterns of prescription drug misuse presenting to provincial drug clinics

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Abstract

Aim To survey new patients, presenting to three drug clinics, on the patterns, usage and costs of prescription pharmaceuticals.

Method Consecutive patients seen by the medical staff for assessment had a 7-day history recorded for prescription drug (PD) usage, and the associated costs of these from street sources.

Results There were 37 patients (26 males) with a mean age of 34 years (21–51). Ten reported using only intravenous (IV) morphine, at a median dose of 105 mg/day (40–600), at a mean cost of 56 cents/mg. Another 12 reported methadone as their sole opioid at a median dose of 50 mg/day (27–70), at a mean cost of 81 cents/mg. A further 11 used a mixture of opioids, predominantly morphine and methadone but also dihydrocodeine (3), oxycodone (1), tramadol (1) and codeine (2). Seventeen reported also using hypnotosedatives, but did not report high doses of these. The overall weekly expenditure on PDs was \$367/week (0–2100).

Conclusions Morphine and methadone remain the predominant street opioid PDs in this region. Street prices have reduced, perhaps reflecting greater drug availability in accordance with increased national prescribing of opioids. There is continuing diversion of PDs to the street which is an ongoing Public Health issue requiring coordinated responses, including improved prescribing training, pain guidelines, drug clinic policy and actions by Medsafe, Police and regulatory bodies to contain this problem.

Prescription drug abuse has been a long-standing issue in New Zealand,¹ which in the past has been overshadowed by imported heroin, cannabis, and more recently by methamphetamine.

Regarding opioids, New Zealand was placed in an unusual situation 30 years ago when the “Mr Asia” supply ring was contained, and imported heroin became increasingly uncommon and virtually unavailable for “street” supply. In this situation prescription opioids became a prime source, and the misuse of buprenorphine was a notable initial consequence, followed by morphine and methadone.^{2–4}

A number of transient “New Zealand innovations” emerged: firstly the “homebake” phenomenon of producing morphine/heroin from over-the-counter codeine (combination) products,⁵ secondly the development of oral opium ingestion from poppyseed tea,⁶ and perhaps, more recently, increasing misuse of codeine itself from prescribed or over-the-counter combination sources.⁷

However continuing prescription drug abuse is of ongoing concern in New Zealand,⁸ and also particularly in the United States.^{9–11}

We were prompted to evaluate the pattern of current drug use in patients presenting to our drug clinics, as there has not been a recent survey.

Methodology

This study was restricted to new patients (new episode of care) who presented to three provincial clinics, and who were seen by medical staff, usually in the course of assessment for opioid substitution treatment, but also for withdrawal management.

In the course of taking the drug history, the medical officer completed a form on prescription drug use over the previous 7 days. This included the drug(s), daily dose, route of administration, and source (street or prescription). There was a further section to estimate the total costs of each street-sourced drug for the previous 7 days.

This form did not collect information about alcohol, cannabis or tobacco.

This information was collected from October 2009 until April 2010 on consecutive new patients seen by the medical staff.

Results

There were 37 assessments comprised of 12 from Hawke's Bay, 15 from Taranaki and 10 from Palmerston North.

There were 26 men and 11 women, with a median age of 34 years (21–56 range).

Drugs

Opioids—10 patients reported using intravenous morphine as their only opioid over the previous week. The mean dose/day was 169 mg (median 105), with a range of 40–600 mg/day.

Another 12 reported using intravenous methadone as their sole opioid over the previous week, at a median dose of 50 mg/day, with a range of 27–70 mg/day.

There were a further 11 patients who reported using a mix of opioids over the previous 7 days. Ten of those reported using morphine, which was combined with methadone in seven. Two morphine users were prescribed Dihydrocodeine tartrate (DHC Continus) at doses of 180 mg and 240 mg daily, and another reported street-sourced DHC Continus at 60 mg/day.

One patient reported using a mixture of morphine, methadone and oxycodone, the latter at a mean dose of 70 mg daily. Two patients reported regular use of codeine at 190 mg/day in addition to morphine or methadone. One patient reported regular use of opium in addition to morphine. No patients reported poppyseed tea or codeine-sourced “homebake” morphine use.

We included 2 subjects who reported using over-the-counter codeine combinations, as codeine can be a prescription drug. The doses of codeine were 260 mg/day and 800 mg/day. One patient reported problematic use of prescribed tramadol.

Stimulants—There was one presentation due to street-sourced methylphenidate.

Hypnotics—Seventeen patients reported use of hypnotics over the previous week. These were prescribed to five patients, and street-sourced in the remainder. This included a wide range of benzodiazepines, and zopiclone in one case. These drugs were all reported being used at usual therapeutic dosages, and often taken irregularly.

Cost

The patients who reported using morphine stated a street price range from 25 cents to \$1 per mg—mean 56 cents/mg.

The patients who reported methadone use stated a street price range of 20 cents to \$1 per mg—mean 81 cents/mg.

The one patient using oxycodone was paying 50 cents/mg.

For benzodiazepines prices ranged from 40 cents to \$6 per tablet, and zopiclone \$1 per tablet.

The weekly expenditure on street pharmaceuticals was tabulated at the time of assessment. Excluding those with pharmacy codeine purchasing, and those who were prescribed drugs, the mean cost was \$367/week (0–2100) n=31.

Discussion

This survey, although confined to small numbers in a selected group of drug users, confirms our clinical experience over the last 15 years that the principal “street” opioids continue to be long-acting morphine tablets, and methadone solution. In the absence of evidence of morphine importation, it is likely this is mostly diverted by patients prescribed for chronic pain and malignancy; and for methadone from that prescribed by drug clinics for opioid dependence, particularly via “take-home” doses. There may also be some contribution from pharmacy/warehouse break-ins, prescription forgery, internet purchases, or even healthcare employees.

The amounts of morphine reported are likely to be accurate, as users buy the long-acting morphine in specific tablet sizes, in contrast to methadone takeaways which may be diluted by the seller. The amounts and proportions of methadone and morphine are comparable to those reported in a 1995–96 survey.⁴

Despite limited and sporadic supplies of imported heroin over the last 30 years, opioid misuse has continued, as judged by the increased numbers on opioid substitution treatment for dependency. These numbers have increased from 650 in 1990 to more than 4,000 currently. This probably reflects the considerable increase in prescriptions of morphine, methadone and other Class B opioid pharmaceuticals drugs over the last decade, of which a proportion is diverted to the street market.

Although there was only one patient reporting oxycodone use, it is anticipated that a range of pharmaceutical opioids will emerge onto the streets. In the US in 2002 opioid analgesics accounted for 9.85% of all drug abuse notifications, being oxycodone, morphine, hydromorphone and fentanyl, compared to 5.75% in 1997; heroin accounted for 7.7% in 2002.¹²

The cost of street opioids is considerable, but may be relatively lower than in the past. Adamson and Sellman⁴ found the average expenditure on opioids was \$882 per week for patients on the methadone waiting list in Christchurch 15 years ago. It is acknowledged that there may well be regional variation in drug use patterns and price. Reasons for the apparent lower current street price of opioids may include the increased provision of opioid treatment services with relatively less opioid dependent persons not in treatment, as well as the increased prescribing of opioids.

Adamson and Sellman also confirmed the frequent association with crime to fund the drugs for opioid dependent persons.⁴ In addition to the social and legal issues generated by street pharmaceutical costs, injecting drug use is associated with bloodborne virus transmission and infection, sepsis, and opioid overdose. Reith et al¹³ have reported on opioid-related deaths in New Zealand noting the significant contribution of both morphine and methadone in unintentional deaths. They raised the issue of take-home methadone doses as a contributor.¹³ A Scottish study found fewer methadone-related deaths despite increasing prescriptions due to improved clinical systems around methadone provision and compliance with guidelines.¹⁴

Internationally there is awareness of methadone diversion by patients on maintenance opioids through drug clinics.¹⁵ This is of concern as it brings clinics into disrepute through paradoxically providing drugs to the very market they are attempting to treat. There is tension around perceived overly restrictive treatment conditions making programmes unattractive to drug users, versus the risks of diversion via take-home doses, which require careful individual consideration depending on clinical progress.

The factors determining “takeaway” policies are complex and include local heroin availability, treatment availability, access/waiting lists, improved retention in treatment, and employment facilitation.¹⁶ In addition Zador reflected on the high mortality from overdose in patients discharged from substitution treatment by “strict” drug clinics.¹⁷

We believe prescription opioid abuse is an important and ongoing Public Health issue in New Zealand. However it would appear to have been a lower priority for Police compared to methamphetamine, cannabis and cocaine. It received scant attention in the National Drug Policy (2007–2012). However, the proposed review of the Misuse of Drugs Act indicates an intention for better controls to prevent prescription “drug-seeking”. Some measures have been introduced, including a 10-day maximum dispensing for Class B drugs, and controlled drug prescriptions being recorded on an electronic database by Medicines Control which will improve monitoring of prescribing. There appears to be less activity regarding Drug Abuse Containment publications and propagation of the “restricted persons” schedule.

There are relatively few prosecutions by regulatory bodies or restrictions on Controlled Drug prescribing rights of doctors or other Health Practitioners for improper prescribing. To date responses from various authorities and prescribers of opioids have failed to contain this problem. Sheridan and Butler have provided an in-depth menu of strategies for reduction of prescription drug misuse in New Zealand.⁸

If there can be a positive in this matter, there was a recent speculative opinion proffering an alternative view that prescription drug abuse may be advantageous in harm-reduction terms, compared to heroin. Not all prescription drugs lend themselves to the injection route and doses are pharmaceutically defined. This reduces overdose risks, costs to users and acquisitive crime. Systems of unsanctioned quasi medical opioid substitution emerge, which may offer some public health benefits.¹⁸

Finally, it is acknowledged the balance between prescribing for adequate analgesia versus the risks of diversion of drugs to the street for non-therapeutic purposes is a complex issue needing research avenues.¹⁹ Attempts have been made to address these issues by a combined Australasian Medical Colleges publication on Prescription

Opioid Policy.²⁰ There is a need for better information and support for doctors on analgesic-ladder prescribing, guidelines for opioid treatment of chronic non-malignant pain, and strategies to monitor patient compliance with medication.

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Controlled intoxication: the self-monitoring of excessive alcohol use within a New Zealand tertiary student sample

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Abstract

Aims Drawn from a study aimed at exploring students' drinking behaviour and attitudes, this article focuses upon findings that revealed how heavy-drinking students monitored and managed their experiences of alcohol intoxication.

Methods 819 students residing within three university student residences were invited to participate in three phases of data collection. Utilising a combination of qualitative and quantitative research methods, a total of 15 focus group interviews and 18 indepth interviews were undertaken, and 501 students (61%) completed a written survey questionnaire.

Results Sixty percent of students agreed with the statement "I usually know beforehand if I am going to get drunk". One-half of male drinkers and one-third of female drinkers reported they were intoxicated on a weekly basis. When drinking to intoxication, the majority of students monitored a range of drinking effects (a total of 14 were identified) which they considered were signals for the need to either slow down or stop drinking.

Conclusions The majority of drinkers in this study who consume alcohol with the intention of getting intoxicated, typically drink to a predetermined level of intoxication, and maintain that level by monitoring a range of drinking effects—this behaviour has been termed controlled intoxication. Future harm-minimisation strategies could be developed that encourage heavy-drinkers to adopt 'safer' drinking-effect signals as indicators to slow down or stop drinking.

Excessive alcohol use is common amongst New Zealand drinkers¹ and this is particularly so amongst New Zealand tertiary (university and polytechnic) student drinkers.^{2,3} Students living in student residential accommodation (dorms, colleges, and halls of residence) often consume larger amounts of alcohol than their non-resident student peers.^{4,5} Student drinking is associated with high levels of alcohol-related harms.^{3,6}

The current study was undertaken as a component of a doctoral research project that investigated the drinking behaviour and attitudes of campus-resident student drinkers, with a view to developing harm minimisation strategies. This paper details findings that reveal the dynamics of excessive drinking behaviour and uses the term 'controlled intoxication' to identify the process by which some heavy-drinking students monitor and manage their predetermined level of intoxication.

Method

Sample

The research population was drawn from three student residential facilities, situated on-campus, at a medium-sized New Zealand university, in 2006. Sixty-two percent of students were female and 38% male. The majority of students were aged 18–19 years (59%), followed by 20 years (15%) and 21 years (9%). Fifty-three percent of students identified as New Zealand European, 16% Asian, 12% New Zealand Māori, and 5% Pacific people (e.g. of Samoan origin).

Data collection

A combination of qualitative and quantitative research methods⁷ was used during three phases of data collection. The use of focus group interviews and indepth interviews during the first phase of the data collection process allowed the researchers to explore the topic with students, and to gather data to assist in the development of the questionnaire schedule. The use of a quantitative questionnaire survey during the second phase of the data collection process enabled the researchers to collect and analyse data on a large proportion of the campus-resident student population and identify patterns of behaviour and attitudes. The inclusion of focus group and indepth interviews during the third and final phase of the data collection process enabled the researchers to gain students' understandings of the questionnaire survey results and allowed the opportunity to continue refining concepts arising from the data.

Phase 1—Phase 1 involved nine focus group interviews and 12 indepth interviews. Information sessions outlining the research project were held at each of the three residences, and interested students were provided with an invitation letter, information sheet, and a consent form (which provided the researcher with a range of demographic details). The principles of grounded theory⁸ were used to guide the collection and analysis of the qualitative data. Using the grounded theory principle of 'theoretical sampling', students were selected for the Phase 1 interviews to include a diversity of gender, ethnicity, age, and drinking levels. The focus group interviews (consisting of 5–7 students) and the indepth interviews involved semi-structured discussion exploring student drinking behaviour and attitudes.

Phase 2—The second phase of data collection assessed students' drinking behaviour and attitudes utilising a written survey questionnaire, which was completed by 501 students, representing 61% of the research population (respondents provided a good representation of the research population). To ensure the questionnaires were completed privately, and to minimise peer influence, the anonymous questionnaire was administered within a private area of the residence dining room, under the supervision of the researcher.

Phase 3—Phase 3 of the data collection process consisted of six focus group interviews and six follow-up indepth interviews (selected from the original 12 indepth interviewees). The Phase 3 interviewees were provided with a summary of the Phase 2 questionnaire survey results and semi-structured discussion was initiated to elicit student understandings of the survey results. Again, the principles of grounded theory informed the selection of the Phase 3 interview participants and analysis of the qualitative data.

Measures

A 'drinking' student was defined as one who had consumed alcohol during the previous 12 month period. A 'standard' alcoholic drink was defined as the equivalent of 10 grams of ethanol.⁹ The experience of 'intoxication' was self-assessed by students; earlier focus group discussions revealed that students were confident in self-assessing experiences of alcohol intoxication.

Ethics

All data collection procedures, as a component of a PhD research project, were approved by the institution's ethics committee.

Results

Intoxication

Fifty-one percent of male drinking students (44% of all male students) and 36% of female drinking students (30% of all female students) reported that they were intoxicated on a weekly basis.

Premeditated intoxication

Sixty percent of drinking students agreed with the statement 'I usually know beforehand if I am going to get drunk', 14% of students disagreed with the statement and 26% neither agreed nor disagreed (no significant gender differences were found in students' replies to this statement). Further analysis revealed that those students who agreed with the statement, were also more likely to consume more drinks on a typical drinking occasion (Spearman's $r_s = 0.202$, $p < 0.001$).

This finding was supported by interview data, with many heavy-drinking students reporting that they typically planned their big drinking occasions in consultation with peers, purchased a predetermined volume of alcohol from an off-licence retailer prior to commencing drinking (to consume in residence or at a friend's flat [apartment]), and also pre-planned which on-licensed premise they would be attending later in the evening.

Attitudes towards intoxication

Sixty-three percent of students agreed with the statement 'It is okay to get drunk as long as it is not every day'; 17% disagreed, and 20% neither agreed nor disagreed (no significant gender differences were found in students' responses to this statement). Those students who agreed with the statement were also more likely to consume more drinks on a typical drinking occasion (Spearman's $r_s = 0.253$, $p < 0.001$).

The majority of students associated alcohol use with desirable social experiences including having a good time with friends, relaxation, fun, meeting new people, increased social confidence, and for some, an increased likelihood of sexual activity.

Controlled intoxication

Discussion with students during the first phase of interviews revealed that the majority of students who regularly drank to intoxication were very clear in commenting that they did not consume alcohol in an uncontrolled manner. These students reported that they typically consumed alcohol to achieve a pre-determined level of intoxication.

Students stated that they achieved their preferred level of intoxication by monitoring a range of alcohol effects to signal the need to either slow down their drinking or to stop their drinking. For example, four students participating in a Phase 1 focus group, who had varying thresholds of controlled intoxication, discussed how they knew when they had had enough to drink:

Student 1—Whenever I get tipsy [a little drunk] or happy, as soon as I get to that stage. As soon as I am happy, more confident, and louder.

Student 2—I know if I am going to drink anymore I am going to make a total arse of myself, so I stop.

Student 3—I usually just keep going. I am usually still in control when I am smashed to a certain degree. I just keep topping it up. Once I hit smashed, then I will slow down a bit, come back down a bit and then keep topping up to that point.

Student 4—Mine is kind of weird. If I kind of shake my head from side to side and the room moves slower than my head, then I know I have had a bit. So then I probably slow down when my vision can't keep up with my movement.

Drinking students monitored their level of intoxication for reasons of personal safety and control, to minimise the impact of alcohol-related harms, and to ensure that they could gain entry into on-licensed premises. During the Phase 1 interviews and piloting of the questionnaire schedule, students identified 14 drinking effects that were commonly used to monitor intoxication.

As significant gender differences were found in drinking-students' responses to 12 of the 14 drinking effects, male students' responses are shown in Table 1 and female students' responses in Table 2.

The two drinking effects that did not show any significant gender difference were 'when the night is boring and/or no fun' and 'when my friends tell me to stop drinking'.

Table 1. Male drinking-student responses to 14 drinking effects

Variables	I will usually stop my drinking	I will usually slow my drinking	It does not influence my drinking	Not applicable to my drinking
Drinking effect	%	%	%	%
When I start to feel like vomiting	64	26	5	4
When I vomit	61	17	10	12
When I feel extremely tired and want to sleep	57	20	17	5
When my head or the room starts spinning	53	26	11	10
When I know I am very drunk/wasted	48	26	17	8
When I start to lose the ability to walk properly	41	29	21	9
When the night is boring and/or no fun	41	25	28	6
When my friends tell me to stop drinking	39	31	11	19
When I start to lose the ability to talk properly	29	32	23	15
When I know I am drunk	28	40	27	5
When I start to feel aggressive or angry	22	16	25	36
When I start to get too emotional	16	17	23	43
When I reach my limit of counted drinks	15	20	25	39
When I start to feel a little drunk and tipsy	8	44	46	2

Table 2. Female drinking-student responses to 14 drinking effects

Drinking effect	I will usually stop my drinking	I will usually slow my drinking	It does not influence my drinking	Not applicable to my drinking
	%	%	%	%
When I start to feel like vomiting	76	10	1	12
When I vomit	71	8	1	19
When I know I am very drunk/wasted	70	13	6	10
When I feel extremely tired and want to sleep	69	19	8	3
When my head or the room starts spinning	61	24	4	10
When I start to lose the ability to walk properly	61	17	7	14
When I start to lose the ability to talk properly	51	21	11	17
When the night is boring and/or no fun	48	20	27	5
When I know I am drunk	48	35	12	5
When I start to get too emotional	44	25	11	20
When my friends tell me to stop drinking	39	39	8	13
When I start to feel aggressive or angry	39	15	8	37
When I reach my limit of counted drinks	28	19	23	30
When I start to feel a little drunk and tipsy	12	59	28	1

Access into on-licence premises—It was a feature of student drinking behaviour that once students commenced drinking in residence (or at a friend’s flat) they would typically move on to an on-licensed premise to continue drinking. Although students pre-loaded with alcohol (as it was cheaper) prior to arriving at on-licensed premises, they typically limited their level of intoxication to ensure they could gain entry.

Alcohol-related harms—Many students reported that in the previous six-month period they had experienced alcohol-related harm as a consequence of their drinking. The most common harms included vomiting (58%), missing an academic class (58%), physically hurting themselves (57%), and passing out (32%). A feature of interview discussions with students was the high level of tolerance that some students held towards many alcohol-related harms. These students commented that ‘some’ alcohol-related harm was an expected and acceptable consequence of their drinking behaviour.

Discussion

In the current study, the majority of students who consumed alcohol with the intention of getting intoxicated, practised ‘controlled intoxication’ through monitoring a range of drinking effects as signals to either stop or slow down their drinking.

Research evaluating the drinking behaviour of English pub-patrons found similar drinking behaviour, describing it as ‘controlled loss of control’ and explaining that this behaviour allowed drinkers to experience the pleasures of intoxication, while at the same time minimising the associated risks to personal safety and health.¹⁰

Similarly, research assessing the intoxication levels (using a breathalyser machine to test blood-alcohol levels) of bar drinkers in a major European city reported that most intoxicated drinkers’ level of intoxication did not vary by the hour of the survey,

suggesting that drinkers reached a certain level of intoxication which they then endeavoured to maintain throughout the course of the evening.¹¹

The New Zealand Alcohol Advisory Council's 2005 survey of New Zealanders' drinking behaviour reported that of the 22% of New Zealand adults who were assessed as binge drinkers, just over one-half (12%) were categorised as 'constrained-binge drinkers'.¹ Constrained-binge drinkers were defined as consuming binge drinking amounts of alcohol but limiting their total volume of alcohol due to concerns about health, work, and family.

Although students practising controlled intoxication were typically endeavouring to limit their experience of alcohol-related harm, many of these students still continued to accept a certain level of alcohol-related harm as an expected and acceptable cost of their excessive drinking behaviour. This tolerant attitude towards alcohol-related harm is consistent with the literature on risk-taking behaviour by young people, and highlights the challenges facing education-based alcohol harm-minimisation strategies that target student drinkers.

Of the 14 drinking effects that students reported using to monitor controlled intoxication, only one effect, 'friends instructing friends to stop drinking', was external to a student's direct control. In 2007, the New Zealand Ministry of Transport launched a social-marketing campaign based upon 'peer-relationships' that encouraged friends not to allow an intoxicated friend to drink and drive.¹²

The New Zealand Alcohol Advisory Council has also utilised similar peer-based messages in its recent 'Ease Up on the Drink' campaign.¹³ The underlying messages of these campaigns is that 'friends take care of friends' and 'friends talk to friends about their drinking behaviour'. The findings of the current study support these peer-based harm-minimisation campaigns.

The propensity of bar drinkers to limit their level of pre-loading to ensure bar entry highlights the importance of bar management and bar door-staff in setting the intoxication-threshold that some students, and arguably some of the general population, drink to prior to their arrival at on-licensed premises. The current findings support initiatives aimed at reducing the intoxication-threshold that licensed premises enforce—as a strategy to reduce pre-loading alcohol consumption amongst bar patrons.

Strengths of the study include the combined use of qualitative and quantitative research methods to provide insight into, and triangulation of, the research data.⁸ The findings of the current study are to an extent limited by the restricted demographics (i.e. resident student drinkers) and small size of the research population.

The research was primarily undertaken as an exploratory study and further research is required, utilising a large general population sample, to assess whether the 14 drinking effects identified in the current study are also used by other drinkers.

If further research determined that certain drinking effects were found to be more commonly used for monitoring and managing alcohol intoxication, then harm-minimisation strategies could be developed that encourage heavy-drinkers to adopt 'safer' drinking-effect indicators as signals to either slow down or stop their alcohol consumption.

Competing interests: None.

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Alcohol's harm to others: self-reports from a representative sample of New Zealanders

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Abstract

Aim There is a lack of research, internationally and in New Zealand, on the harms experienced as a result of drinking by others. Such effects have often been neglected in policy development and in estimates of the economic burden associated with alcohol consumption. This study describes the broad range of harms reported by New Zealanders due to the drinking of someone else.

Method A representative national survey was conducted using Computer Assisted Telephone Interviewing with New Zealanders aged 12 to 80 years (N=3068) in 2008/2009 (response rate – 64%). Harms experienced due to the drinking of others were reported along with demographic variables.

Results One in four respondents indicated that they had at least one heavy drinker in their life. Most of these respondents indicated they had experienced a range of harms because of this person's drinking. Further, 17% of respondents with children reported that their children experienced harm because of the drinking of someone else. Seventy-one percent of those sampled reported experiencing at least one harm because of the drinking of a stranger.

Conclusion A large proportion of New Zealanders report the experience of physical, social, economic, and psychological harms because of the drinking of others. These harms should be considered in the discussion of alcohol policy.

Alcohol has negative effects for people other than the drinker; however, these are less well documented than the impacts experienced by the drinkers themselves. These less well measured effects are relevant to the formulation of alcohol policy and might, if carefully established, be influential in the policy debate in the way that passive smoking contributed to tobacco policy debates.

The limited data available on the effects on those other than the drinker have largely been confined to the effects of traffic crashes and foetal alcohol syndrome, both of which have been established well enough to be included in measures of alcohol's contribution to the Global Burden of Disease.¹

In New Zealand, 40% of traffic crash injury is experienced by those other than the drinking driver.² Other injury has also been related to the effect of others' drinking with a survey showing 5.7% of 12–65 year old New Zealanders reported being physically assaulted by someone who had been drinking, and 5.3% reported being sexually harassed under the same circumstance.³

Research conducted on the effects of alcohol among university students in New Zealand showed that 84% of survey respondents experienced at least one harm because of other students' drinking within the month prior to the survey. For instance,

one-third of students reported being insulted or humiliated; 15% being pushed, hit or otherwise assaulted; and 28% reported experiencing unwanted sexual advances.⁴

The effect of alcohol on the families of heavier drinkers has been investigated with research showing that alcohol is related to greater family disruption and lower family functioning scores,⁵ and stress.⁶ The effects on children begin prenatally with drinking during pregnancy associated with Foetal Alcohol Syndrome.⁷ In longitudinal studies parental alcoholism has been associated with increased risk of attention deficit hyperactivity disorder, substance abuse, conduct disorder, mood and anxiety disorders,⁸ and teacher rated behavioural problems.⁹ Other studies show a relationship between parental heavy drinking and lower academic achievement.¹⁰

While there has been research on alcohol's involvement in workplace injuries or deaths,¹¹ it has been without specific reference to the issue of the effect of a worker's drinking on others. Similarly, absenteeism due to drinking has been studied in New Zealand,¹² as elsewhere, but the impact on the wellbeing and productivity of workmates has not been researched.

Some research has assessed the specific harms reported by survey respondents due to the drinking of strangers (for instance, being afraid of intoxicated people on the street). Research conducted in the Nordic countries found that the percentage of respondents who reported experiencing three or more such harms ranged from 10–22% across gender in the four countries studied (Denmark, Finland, Norway and Sweden). The most common harm reported for men and women was being harassed by intoxicated people in a public place (in the Swedish sample).¹³

This paper reports findings from a national sample of New Zealanders who were asked about their exposure to heavy drinkers. Analysis of respondents' reports of their personal wellbeing and health status has shown significant relationships with level of exposure to heavy drinkers.¹⁴ This analysis reports the prevalence of specific experiences related to the heavy drinking of people in different relationships to the respondent. It provides the first comprehensive measurement of a wide range of alcohol's effects on those other than the drinker in New Zealand.

Method

Data collection—Data was obtained from a representative national sample of 3068 New Zealanders, aged 12–80, who were living in private residential dwellings with a connected landline telephone in New Zealand in 2008/9. The survey was conducted using Computer Assisted Telephone Interviewing (CATI).

Landline phone numbers were generated randomly and distributed in proportion to the usually resident population across strata area, which cover the whole country when combined. Once recognised as a residential line, households were called at different times of the day and days of the week at least ten times in order to reach a respondent. All eligible people in the household were enumerated from which the computer selected one respondent at random. If the selected respondent was not available for interview a call back was arranged (if the selected respondent was away for the duration of the survey they were counted as a non-response).

As with any telephone-based interviewing method, people without landline telephones are excluded from the sample. Telephone coverage in New Zealand however is comparatively high. In 2006, 97.9% of households in New Zealand had accessible landline telephones.¹⁵ Certain sectors of the population are under-represented among those with access to a landline telephone, however previous analysis using similar New Zealand survey data has found that does not affect population level estimates.¹⁶

Response rate—The response rate of the survey was 64% [(number of eligible responding / the number of eligible responding + number of eligible non-responding + estimated number of eligible from the unknowns) × 100].

Eligible respondents were defined as those aged 12–80 years who had lived in the country for at least 12 months. Eligible non-responders were made up of those who were contacted but declined to take part. The estimated number of eligible from the unknowns was an estimation of the respondents that would have been eligible in the households that were unable to be reached.

Response rates are declining internationally and in this context 64% is a high response rate, particularly taking into account that the sampling frame included unpublished telephone numbers (providing a more representative sample but reducing response rates). This response rate is within the range of 50%–80% found to produce accurate and unbiased estimates of health related behaviour.¹⁷

Sample weighting—The sample was reasonably representative of the NZ population aged 12–80 years old at the time of the 2006 Census [see for example Statistics New Zealand^{18–20}] however weighting was carried out to correct for unequal household selection probabilities.

The data were weighted in four stages. The first stage was to correct for dwelling unit or household selection probabilities. The second stage was to match the strata back to Census data – (because there were slightly larger numbers in the Auckland Region). The third stage was to match the survey weights to New Zealand 2006 Census (Access to a Landline Telephone only) population distributions using Rim Weighting, an iterative procedure to match the sample to the known population marginal, upon a set of major variables, in this case for groups based on gender, age and ethnicity. The final stage involved a standardization to match the weighted sample size back to the initial survey size.

Measures—Respondents were asked a number of questionnaire items about the drinking of those around them. Initially they were asked whether there “are any people in your life whom you consider to be fairly heavy drinkers or who sometimes drink a lot”. Those who answered affirmatively were then asked to specify the number of heavy drinkers in their life and their relationship to these people. The respondent next identified the person whose drinking had the most significant negative impact on them over the last 12 months. Subsequently, questions were asked about the occurrence of specific adverse events associated with this person’s drinking.

Respondents with children aged under 18 living in the household were asked about the effects of someone else’s drinking on these children. Another set of questions addressed the specific harms experienced by the respondent because of the drinking of a stranger. The survey also included items on the respondent’s demographic information. The questionnaire items were developed in collaboration with researchers who conducted a similar study in Australia.²¹ The responses to the questions were such that other members of the household were not able to tell what the respondent was reporting.

Ethics—The project was been reviewed and approved by the Massey University Human Ethics Committee.

Results

Twenty-eight percent of respondents indicated that at least one person with whom they had some relationship was a “fairly heavy drinker or sometimes drinks a lot” (as shown in Table 1). Female respondents were significantly more likely to report having a heavy drinker in their life (31%) than males (25%) (Chi-squared=12.8, df=1, p=0.0003).

In order, respondents reported that the heavy drinker was a relative or partner not in the household (this included boyfriends, girlfriends, and ex-partners) (13%), a friend (13%), household member (6%) and finally a coworker (2%). Women were more likely to report heavy drinking among household members and relatives or partners outside the household (22%; males 12%) (Chi-squared=31.9, df=1, p≤0.0001).

Table 1. Percentages of those in various relationships to the respondent reported as heavy drinkers by the respondent

Variables	Female	Male	Total
(N)	1836	1232	3068
Household member*	8	3	6
Relative or partner (not in the household)**	16	9	13
Friend	12	14	13
Coworker†	1	2	2
Total reporting one heavy drinker	31	25	28

*Partner/spouse, mother, father, children, sibling, wider family member who live in the same household as respondents; **Includes boyfriends, girlfriends, and ex-partners; †Work colleague, employer, and employee;

Respondents who indicated the presence of at least one heavy drinker in their life were subsequently asked to identify the person whose drinking had most negatively affected them in the last 12 months (see Table 2). The description of these people in terms of relationships was generally proportionate to the reports of heavy drinkers in that category (Table 1).

Table 2. Percentages of those the respondent reports as the heavy drinker whose drinking most negatively affected them

Variables	Female	Male	Total
(N)	492	268	760
Household member	23	11	18
Family members not in the household	43	33	39
Friend	31	48	38
Coworker	3	7	5
Other	1	1	1

Effects from the drinking of the person who most affected them—Of the respondents who indicated the presence of at least one heavy drinker in their life, 85% identified the one person whose drinking most negatively affected them. These respondents were then asked a series of questions about the specific adverse experiences and situations they had encountered in the last 12 months due to this person’s drinking (see Table 3).

Eighty-four percent of these respondents indicated that they had experienced at least one of the adverse impacts listed. Among younger respondents (12–29 years) over 95% of those who reported one person who most negatively affected their life reported at least one specific harm because of this person’s drinking.

The most commonly reported experience (reported by over half of respondents) was that the other person’s drinking meant that the drinker failed to do something they were being counted on to do. Around a third or more of the respondents indicated that the other person’s drinking meant they had felt emotionally hurt and neglected, had a serious argument with the person, had to stop seeing the person, had to drive them somewhere, had to clean up after them, had to take on extra caring responsibilities, and that the other person was not there for them and interested in them.

In regards to the more serious harms asked about, 7% of respondents reported being physically hurt by the other person and 9% reported having money stolen by them.

There was no significant gender difference in the percentage of those reporting experiencing at least one harm (Chi-squared=1.08, df=1 p=0.300); however, there were differences in the specific harms reported. The largest differences were that females were more likely to report being emotionally hurt or neglected (51%) (Chi-squared=17.3, df=1 p<0.0001) or feeling threatened and scared (30%) than males (35% and 22% respectively) (Chi-squared=4.0, df=1 p=0.046).

Table 3. Percentage of respondents who reported being affected in the following ways by the drinker who most negatively affected them (n=760)

Harms	Female	Male	Total (% of the total sample)
<i>How many times in the last 12 months, because of their drinking:</i>			
Were you emotionally hurt or neglected by them?	51	35	44
Did you have a serious argument (not including physical violence)?	51	47	49
Did they fail to do something they were being counted on?	57	52	55
Did you have to stop seeing them?	32	28	31
Did you have to take them somewhere?	33	39	35
Was there not enough money for the things you needed?	19	11	15
Did you have to clean up after them?	38	41	39
Did you feel threatened or scared by them?	30	22	26
Were you physically hurt by them?	8	7	7
Did you feel at risk in the car when they were driving?	18	11	15
Were you forced or pressured into sex or something sexual?	2	3	3
Did they break or damage something that mattered to you?	27	19	23
Did you have to take on extra responsibilities caring for children or others?	30	31	30
Could you not bring friends home?	17	17	17
Did you have to leave home to stay somewhere else?	17	10	14
Did you avoid seeing other friends/family because you were embarrassed?	26	18	22
Were you injured in a car accident?	2	1	1
Were you less able to do your paid employment, or have to take time off?	12	15	13
Were meals not cooked?	19	14	16
Was there no transport to and from places?	19	21	20
Have they not shown much interest in you?	45	38	42
Have you not seen them when you wanted to?	44	40	42
Has your money been stolen by them?	8	10	9
Have you gone without food?	6	5	5
Total respondents experiencing at least one adverse impact	85	83	84

Effects on respondents' children—Respondents who had identified having at least one heavy drinker in their life were asked whether they had any children under 18 years old living in their household. Those who did were asked about specific harms experienced by the children because of someone else's drinking (this could have included another parent or caregiver of the child) (see Table 4).

Seventeen percent of respondents with children in the household indicated the children were negatively affected by the drinking of someone else in the last 12 months. Eleven percent of those with children living in the household indicated that the child had been yelled at or verbally abused because of someone else's drinking.

Seven percent of respondents with children in the household reported that children had witnessed serious violence in the home because of someone else's drinking.

Table 4. Harms experienced by under-18 year old children living in the household of the respondent (n=334)

Harms	Female	Male	Total
<i>Because of someone else's drinking how many times in the last 12 months:</i>			
Were children yelled at, criticised or verbally abused?	13	9	11
Did children witness serious violence in the home?	6	7	7
Were children left in an unsupervised or unsafe situation?	6	5	5
Were children physically hurt?	4	1	2
Was a protection agency or family services called?	2	2	2
Was there not enough money for the things the children needed?	4	5	5
Total respondents with children reporting at least one impact	18	15	17

Effects from a heavy drinking coworker—Of those respondents who reported having a heavy drinking coworker, 39% reported experiencing at least one specific harm (see Table 5). Forty-four percent of these reported that they had experienced reduced productivity because of a colleagues' drinking.

Table 5. Harms experienced by respondents with coworkers identified as heavy drinkers (n=61)

Harms	Female	Male	Total
<i>Because of your coworker's drinking how many times in the last 12 months:</i>			
Were you involved in an accident or a close call at work?	4	0	2
Have you had to work extra hours?	29	24	26
Did you have to cover for them?	47	21	31
Was your productivity at work reduced?	34	50	44
Total percentage of respondents who indicated they had one heavy-drinking coworker reporting at least one adverse impact	44	36	39

Effects from strangers' drinking—Finally, respondents were asked about the harms experienced because of a stranger's drinking. Seventy-one percent of respondents indicated that they had experienced at least one harm due to the drinking of strangers or people they did not know very well. The age group most likely to report experiencing at least one harm was those between 20–29 years old (85%) and least likely were those over 50 (58%), with no differences in the likeliness of reporting at least one harm across genders, although reports of experiencing specific harms varied by gender.

The specific harms experienced by those respondents who indicated one or more adverse event are shown in Table 6. Almost half of those who indicated they had experienced at least one harm because of a stranger's drinking stated that they had gone out of their way to avoid drunk people and places. Nearly one third responded that they had been verbally abused.

Around 15% answered that because of a stranger's drinking they had damage done to their property, had been paid unwanted sexual attention, had been threatened, had felt unsafe and had a serious argument. Fewer respondents indicated that they had been physically hurt (4%), been injured in a car accident (1%), or been forced or pressured to do something sexual (2%).

The largest gender differences were that female respondents were more likely to report feeling unsafe waiting for public transport (18%; males 11%) (Chi-squared=19.3, df=1 p<0.0001) and being paid unwanted sexual attention (18%; males 12%) (Chi-squared=11.7, df=1 p=0.0006) than men, whereas men were more likely to report being annoyed by vomit and littering (70%; females 59%) (Chi-squared=31.4, df=1 p<0.0001) and being verbally abused (31%; females 24%) (Chi-squared=11.7, df=1 p=0.0006) due to the drinking of others.

Table 6. Respondent-reported alcohol-related harms in the community. Harms experienced by respondents indicating at least one harm because of the drinking of a stranger (N=2142)

Harms	Female	Male	Total
<i>We would now like to ask you about strangers or people you don't know very well. Because of their drinking, in the last 12 months, how many times have you:</i>			
Avoided drunk people/places?	47	44	45
Been kept awake or disturbed at night?	51	46	49
Been annoyed by vomit, urination or littering?	59	70	64
Felt unsafe waiting for public transport?	18	11	15
Felt unsafe in a public place?	21	19	20
Experienced trouble or noise related to a licensed venue?	10	11	11
Been verbally abused?	24	31	27
Been physically hurt?	3	5	4
Been threatened?	15	18	16
Been involved in a serious argument?	14	18	16
Been injured in a car accident?	1	1	1
Had damage done to your house, car or other property?	13	15	14
Been forced or pressured to do something sexual?	2	2	2
Been paid unwanted sexual attention?	18	12	15

Discussion

This research records the self-reported harms experienced because of the drinking of others by a representative sample of New Zealand respondents. The range of harms experienced is wide: from physical violence to emotional hurt and neglect to lower work productivity.

Previous research has tended to look at the effects on the immediate family of heavy drinkers. Our research shows that the drinking of people in various relationships can also have negative effects on others. One in four of our sample reported experiencing at least one adverse impact during the previous 12 months due to the drinking of someone they knew.

While there were significantly more women than men reporting heavy drinkers in their lives, reports of the specific adverse effects experienced because of the drinking

of someone known to them were similar between men and women. Similar numbers reported taking on extra responsibilities caring for children or others, that they had money stolen and had gone without food.

As many men as women also reported having been physically hurt and forced or pressured into sex. However, some gender difference existed in the harms experienced with women more likely to report having felt threatened or scared, having felt at risk in a car and having something that mattered being broken or damaged. They also were more likely to have felt emotionally hurt or neglected.

Children have long been recognised as being particularly vulnerable to the impacts of others' drinking, particularly that of their caregivers. A New Zealand literature review for the Families Commission concluded that most studies show harmful impacts on the health and wellbeing of children in families with caregivers who are heavy drinkers.⁷

Two percent of the respondents in this study who had children living in their household reported that child protection services had been called because of someone else's drinking. However, this is a smaller proportion than those who reported both verbal and physical abuse of children due to someone else's drinking. The considerable impacts on children found in this study fit with previous research that indicates that heavy alcohol use in the family is a risk factor for child abuse.²²

While only small numbers reported heavy drinking coworkers, more than a third of these reported some adverse effects, including having to cover for their workmates and their own productivity being reduced. While this is a relatively under researched area, estimates of economic costs from alcohol attribute considerable costs to lost productivity in the workplace.^{11,12}

Seventy percent of our sample reported at least one adverse event that was attributed to the drinking of a stranger. These findings support other evidence of the relationship of alcohol with public nuisance and disorder. Internationally, United Kingdom data show that almost 75% of residents in London Boroughs reported experiencing crime, disorder, nuisance or anti-social behaviour that they attribute to the drinking of others²³ and one-quarter of respondents in an Irish sample report feeling unsafe in their local area at night.²⁴

Overall, these results are similar to those found in a comparable Australian study. In Australia, 28% percent of respondents reported being negatively affected by the drinking of someone they knew and 75% of respondents reported at least one harm of a public nuisance sort that they attributed to the drinking of strangers.²¹

Establishing causal relationships in cross-sectional research such as this is difficult. In this analysis data from items in which the respondents themselves attribute the harm experienced to the drinking of others is reported. At the least, these data show the extent and range of harms that people attribute to the drinking of others.

A large proportion of New Zealanders report harm from the drinking of others. New Zealand adults experience these impacts in homes, workplaces and in the community. These effects from others' drinking are often overlooked in alcohol policy development, and yet their common occurrence suggests that these harms should be

considered in economic costings of alcohol consumption and in discussion of policies to reduce alcohol-related harm.

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The benefits and harms of alcohol use in New Zealand: what politicians might consider

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Abstract

The New Zealand Government is currently considering ways to reduce alcohol-related harm, following on from a detailed report by the Law Commission. To inform discussions we briefly summarise the benefits and harms of alcohol use in this country. The most substantive benefits to society are probably pleasure to users and economic benefits (largely to industry). The most substantive harms are probably those to mental and physical health, harm to society (e.g. from crime) and adverse net economic impacts. Overall the picture is suggestive that New Zealand society would be likely to achieve a large net benefit from reducing heavy and binge drinking, and shifting alcohol consumption towards a pattern of smaller amounts. The substantial harm to non-users is a key argument for democratic governments to use regulations and taxes to minimise harm from alcohol.

Alcohol presents a relatively complex policy problem, partly because there is a range of benefits and harms that accrue at individual, community and national levels. Nevertheless, national and local politicians are in a position to use laws and regulations to maintain the benefits of alcohol use while further reducing the harms. This year (as of April 2011) a Parliamentary Select Committee has been considering ways to reduce alcohol-related harm, following on from a detailed report by the Law Commission.¹ The international context for this includes good evidence that interventions to reduce alcohol-related harm are cost-effective,² and even cost-saving to government (e.g. alcohol taxation and advertising restrictions).^{3,4}

In an attempt to identify some of the major issues, we generated a very brief summary of benefits and harms of alcohol use in the Table below. These findings suggest to us that New Zealand society would be likely to achieve a large net benefit from reducing heavy and binge drinking, and shifting alcohol consumption towards a pattern of smaller amounts.

Given the issues raised in Table 1, we also suspect that politicians from different parts of the political spectrum might generally favour a shift towards stronger regulation and to higher alcohol taxes. For example, politicians on the “political right” may particularly favour reducing the cost to taxpayers from alcohol-related harm. They may also focus on protecting the rights of individuals who might be harmed by drinkers (e.g., particularly those harmed by alcohol-related violence, crime, and car crashes) and reducing the proportion of police resources used to deal with alcohol-related harm.

Politicians of the “political left” may particularly consider the overall societal benefits from enhanced alcohol regulation and the benefits to disadvantaged New Zealanders. Possibly the only political groupings to oppose any regulatory advances are those of

the “far right” of the political spectrum, if they have an ideological opposition to all new government regulation. But even this grouping may be conflicted, if some members recognise that poorly regulated alcohol imposes an extra burden on taxpayers and imposes harms on non-drinkers, including children.

While major political perspectives are probably consistent with improved regulatory responses to reduce alcohol-related harm, the alcohol and retail industries will tend to oppose enhanced new regulatory or fiscal controls. Overall, these industries have historically voiced opposition to new measures that limit total sales and the size of the discount alcohol market (e.g., which appears to be used to attract shoppers to supermarkets in New Zealand). It is therefore critical for New Zealand politicians to consider industry arguments with appropriate scepticism, and to dispassionately weigh the benefits and harms of alcohol according to the evidence available.

Table 1. The benefits and harms of alcohol use in New Zealand, a brief summary

Aspect	Benefits to drinkers and society	Harms to drinkers, others and society
Psychological / mental health aspects	Pleasure to alcohol consumers which may relate to: beverage taste, the impact of alcohol on enjoying food, satisfaction with being a connoisseur or home brewer, and the pharmacological effects (e.g., pleasure via endogenous morphinergic mechanisms ⁵).	<i>Drinkers:</i> At varying levels, alcohol can cause stress associated with poor judgement. For example, among NZ university students the occurrence of “unsafe, unhappy and unwanted sexual experiences” is increased with alcohol misuse and young age of drinking onset. ^{6 7} There is also stress associated with being dependent on alcohol (especially where this is contributing to financial hardship and/or harming others). Other harms are more obvious e.g., suffering from withdrawal symptoms, hangovers and disrupted sleep after heavy and binge drinking. Alcohol can also cause mental health disorders including depression, anxiety and psychosis and is often present in the blood of those who attempt or complete suicide. <i>Other NZers:</i> There are multiple adverse psychological impacts on family and friends associated with alcohol dependence and adverse consequences of use (e.g., violence, crime, exacerbation of poverty, physical health problems etc – see below). E.g., one study estimated that in NZ, “more than 62,000 physical assaults and 10,000 sexual assaults occur every year which involve a perpetrator who has been drinking.” ⁸ NZ data indicate that when people are exposed “in your life” to heavy drinkers, this adversely impacts on their personal wellbeing and health status. ⁹
Physical health	At low and regular consumption levels (1-2 drinks/day) there are almost certainly benefits from a reduction in cardiovascular disease (CVD) risk for middle-aged and older people. ^{10 11} But this benefit, in terms of disability-adjusted life years (DALYs) gained, appears to be outweighed by other harms in all age-groups from injury and other health problems (e.g., Rehm et al estimated the CVD benefit as being only	<i>Drinkers:</i> The most recent study estimated that 3.9% of annual deaths in NZ were attributable to alcohol consumption (approximately 1037 deaths). ¹³ Even when considering likely health benefits (heart disease prevention) there was a net annual loss of almost 12,000 years of life. In a separate analysis, an annual net loss of 26,000 DALYs was estimated. ¹³ This high overall health burden is consistent with international estimates. ¹⁴ Major adverse impacts are in terms of: injuries, cirrhosis of the liver, selected cardiovascular diseases (hypertensive disorders, cardiomyopathy and haemorrhagic stroke) and cancer (liver, breast, colorectal, pharynx, and larynx). ^{12 15 16} An Emergency Department (ED) study in NZ found that the risk of sustaining an injury was over two times greater when alcohol involved. ¹⁷ <i>Other NZers:</i> There is harm to the fetus from alcohol use in pregnancy and some pregnant women in NZ continue to drink during pregnancy (e.g., 11% of those with unplanned pregnancies have 4-20 drinks per week at some point during their pregnancy). ¹⁸ Children can be harmed where there is alcohol-related violence, sexual assault and poverty. But all age-groups can

Aspect	Benefits to drinkers and society	Harms to drinkers, others and society
	3.3% of the lost DALYs from harm to health from alcohol ¹²).	suffer alcohol-related assault, vehicle crashes, and treatment delays (e.g., in EDs and hospital wards overloaded with patients with alcohol-related problems). One NZ study found “that more than 40% of alcohol-related crash injuries in New Zealand are suffered by people who have not themselves been drinking.” ¹⁹ ... “Most innocent victims are car passengers, and this includes almost all children who are injured by drink driving.” ... “Using official cost figures, alcohol-related injuries to innocent victims cost the country more than half a billion dollars per year.”
Health inequalities (ethnic & gender)	Nil benefits identified.	<i>Society:</i> Ethnic inequalities in health are exacerbated by the relatively high burden of harm to Māori from alcohol. ¹³ As for Māori, Pacific peoples are also more likely to have patterns of hazardous alcohol use (relative to European NZers). ²⁰ Similarly, gender inequalities in life expectancy are also exacerbated by alcohol with 76% of DALYs lost from alcohol in NZ being from men. ¹³ Of note is that low socioeconomic status in childhood is itself a risk factor for alcohol dependence in adulthood according to longitudinal data from NZ. ²¹
Societal functioning	<i>Society:</i> At low and moderate levels alcohol may act as a “social lubricant” and can be valued in some cultural settings (e.g., in celebrations).	<i>Society:</i> A NZ survey found 40% of people had experienced harmful effects on friendships or social life “due to someone else’s alcohol use”. ²² Alcohol-related violence and crime are serious social harms e.g., in NZ at least 31% of violent offences involved an offender who had consumed alcohol before committing the offence. ¹ The Law Commission also reported “a disturbing level of anti-social behaviours from abusive and offensive language, intimidation, sexual harassment, graffiti and vandalism to urinating, excreting and vomiting in public places”. Drink driving and disorderly conduct in youth increased after liberalisation of alcohol laws in NZ in 1990s to early 2000s. ²³ Social capital is also reduced from the adverse health effects (see above) and economic impacts (see below).
Economy	<i>Society/industry:</i> There are economic benefits to alcohol producers and retailers. There is also the benefit to tourism from alcohol contributing to NZ cuisine. It is hard to quantify these benefits at the margin (e.g., the value of a vineyard over other agricultural production, especially when “wine glut” situations occur in NZ ²⁴).	<i>Drinkers:</i> There is adverse economic impact to individuals who buy alcohol when suffering economic hardship. An estimated 147,500 adults (6% of the NZ population) reported having at least one day off work or school in the last 12 months as a result of their alcohol use. ²² <i>Other NZers:</i> A NZ survey found 10% of people had experienced harmful effects on their financial position “due to someone else’s alcohol use”. ²² There is a cost to taxpayers from costs due to health harms, crime, and costs to the education sector (children harmed directly by alcohol [e.g., fetal alcohol syndrome], or where learning is impaired by alcohol-exacerbated poverty). There is also lost tax revenue from lower worker productivity (i.e., there are an estimated 392,800 work days per year in NZ that are lost to alcohol). ¹ Local government (funded by rate payers) pays for some crime-related costs and the costs from alcohol-related litter and broken glass, graffiti and cleaning costs (e.g., from vomit and urine on streets).
Government revenue	<i>Society:</i> Alcohol excise tax provides the government with revenue and so may slightly off-set the need for higher levels of other taxes (e.g., higher income taxes and GST).	<i>Drinkers:</i> The excise tax means that drinkers have to pay extra for alcohol, albeit in proportion to the amount they buy. From an overall alcohol-harm reduction perspective the excise tax is beneficial in reducing heavy drinking, but from an individual drinker perspective it is an added cost, and may contribute to financial hardship, especially in dependent drinkers. <i>Industry:</i> Due to the impact of the higher prices on domestic sales, the alcohol industry will have slightly reduced profits.
Summary	The most substantive benefits to society are probably pleasure to users and economic benefits (largely to industry).	The most substantive harms are probably those to mental and physical health, harm to society (e.g., from crime) and adverse net economic impacts. There appears to be substantial harm to non-users, which provides a key argument for democratic governments to use regulations and taxes to minimise harm from alcohol.

Competing interests: While we do not consider it a “competing interest”, we note that two of the authors (FI and NW) have previously performed work on alcohol issues for health sector agencies. All the authors are regular/occasional alcohol consumers.

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A case with refractory wheezes

Hsiao-Li Lo, Sheng-Hsiang Lin

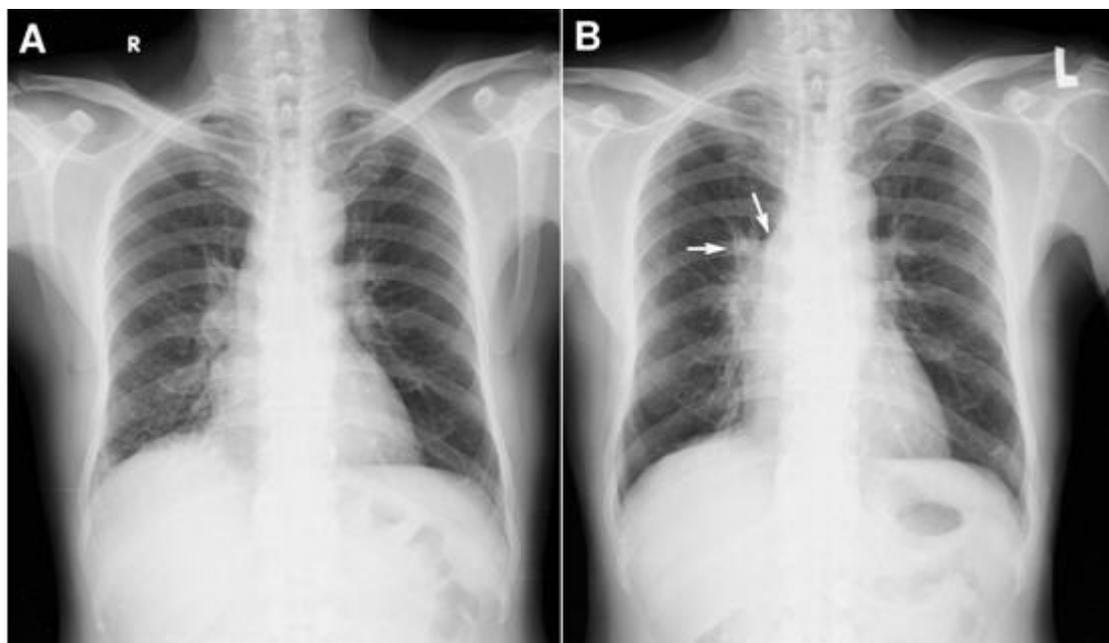
Clinical

A 62-year-old man who had smoked one pack per day for more than 40 years, and had a history of chronic obstructive pulmonary disease (COPD), complained of cough with blood-tinged sputum, dyspnoea and progressive exercise intolerance for 1 week.

On physical examination, expiratory wheezes were heard bilaterally with a stethoscope. Blood tests for haemogram and biochemistry were all unremarkable. The chest radiography showed emphysematous changes and subtle alterations in the right mediastinal and hilar contours (Figure 1).

He was treated for presumed acute exacerbation of COPD with oxygen supplementation, nebulised bronchodilators, intravenous glucocorticosteroids and antibiotics. In the following 3 days, the respiratory symptoms were aggravated and wheezes persisted.

Figure 1. Chest radiography performed 2 years ago (A) and on arrival (B). Abnormalities in the right mediastinal and hilar contours (arrows) were detected by comparison between the two films

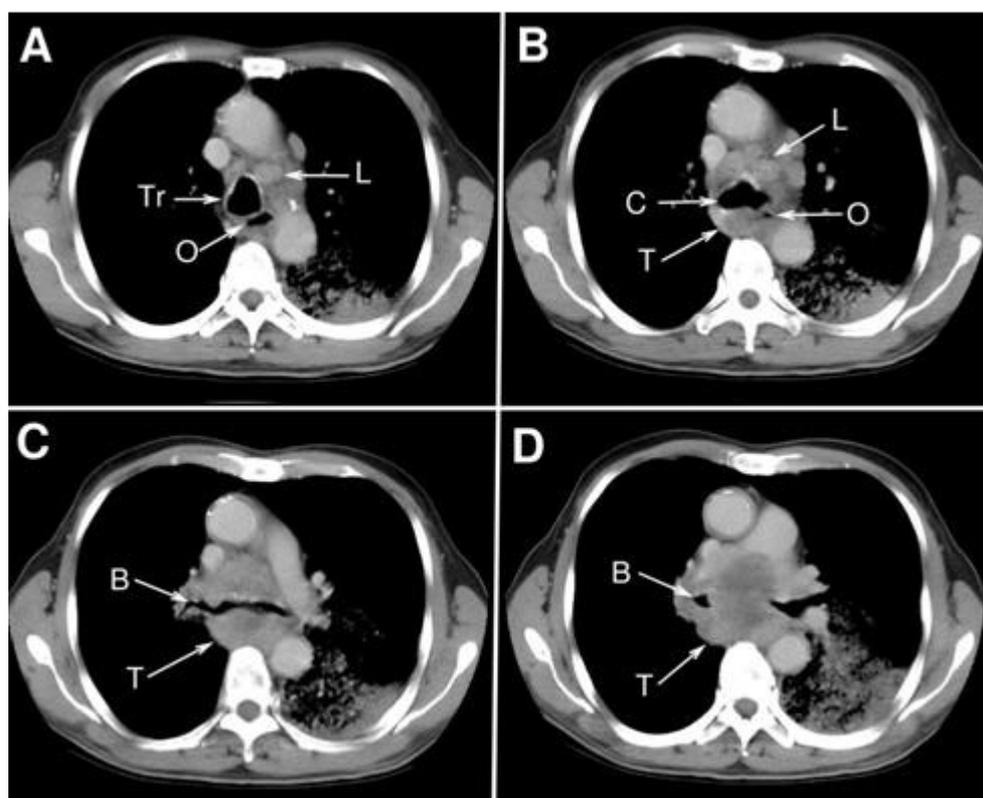


What is the diagnosis?

Answer

Stenosis of bilateral main bronchus secondary to lung cancer. Enhanced computed tomography (CT) of the chest revealed enlarged mediastinal lymph nodes and a large mediastinal tumour with extensive central low attenuation causing bilateral main bronchus encasement and stenosis (Figure 2). The diagnosis of lung cancer was further confirmed by bronchoscopic biopsy.

Figure 2. Enhanced computed tomography of the chest at the levels of trachea (A), carina (B), main bronchus (C), and subcarina (D)



Tr=trachea; O=oesophagus; L=lymphadenopathy; C=carina; B=bronchus; T=tumour.

Discussion

Wheezes are continuous adventitious lung sounds that could be produced in a variety of conditions associated with narrowed airway calibre, such as bronchospasm, mucosal oedema, endobronchial tumour, foreign body, external compression, or dynamic airway collapse.¹ It has been emphasised that all that wheezes is not asthma² and an alternative diagnosis should be considered in all patients with wheezes who do not respond to treatments for asthma or COPD.

In our case, diffuse wheezes were generated by the narrowed main bronchus and the diagnosis was readily established after CT imaging. Although bronchoscopy allows direct visualisation of the airway, CT scanning is a reliable noninvasive assessment of

the central airway. Furthermore, recent advances in multidetector CT may provide a more comprehensive imaging of the tracheobronchial tree.³

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Bilateral diffuse cystic, cavitory lung metastasis of adenocarcinoma

Talha Dumlu, Bekir S Karapolat, Ümran Yıldırım, Adem Güngör, Leyla Y Aydın

Clinical

A 35-year-old female patient admitted to hospital with complaints of cough, chest pain and weight loss.

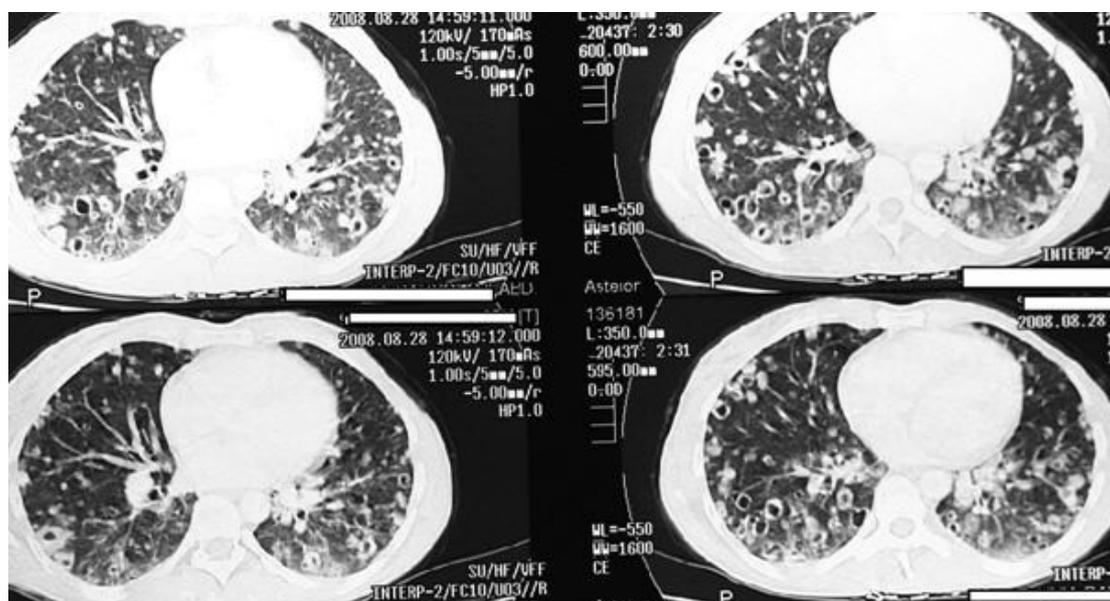
Chest X-ray revealed bilateral diffuse nodular-cavitory and cystic lesions (Figure 1).

Figure 1. Posterior anterior chest X-ray revealed bilateral diffuse nodular-cavitory and cystic lesions



Computed tomography demonstrated bilateral diffuse multiple cavitory and cystic lesions (approximately 1–1.5 cm in diameter) (Figure 2). Cytological examinations of bronchoalveolar lavage were reported as benign cytology. Pathologist recognised neither malignancy nor benign cystic lung disease on transbronchial biopsy material.

Figure 2. Thoracic computerised tomography demonstrated bilateral diffuse multiple cavitary and cystic lesions



During follow-up, the patient developed abdominal pain and jaundice, so we performed endoscopic retrograde cholangiopancreatography. Tubulovillous adenocarcinoma of the duodenum was reported in the duodenal biopsy specimen. Transthoracic open lung biopsy was done for cystic cavitary lung lesions to clarify adenocarcinoma metastasis. Pathological examination of lung biopsy material was identical to the adenocarcinoma metastasis.

Discussion

A wide spectrum of chronic diseases—including usual interstitial pneumonia, desquamative interstitial pneumonia or respiratory bronchiolitis, interstitial lung disease, lymphocytic interstitial pneumonia, emphysema, lymphangioleiomyomatosis, and Langerhans cell histiocytosis—show pulmonary cystic lesions.¹⁻³

Pathogenesis of cavitary-cystic metastasis is not known exactly. They may be either caused by excavation of nodular tumour through discharge of necrotic material inside, or by infiltration of malignant cells into the walls of a pre-existing benign pulmonary bulla. Thirdly, it may be caused by infiltration of malignant cells into the walls of air sacs formed by cystic distension of small airways through the ball-valve effect of the tumour.⁴

In conclusion, we suggest that diffuse bilateral cystic-cavitary pulmonary lesions could be metastasis of malignant diseases.

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Lifetime cardiovascular risk

In his 27 May 2011 *NZMJ* editorial entitled *Improving cardiovascular risk assessment* Harvey White asserts that “lifetime risk is favoured” supported by a reference to his own work. He points out that men have a lifetime cardiovascular risk of 50%.

We of course all have a 100% lifetime risk of death (life is a fatal disease). What Dr White does not even consider is that if people do not die of cardiovascular disease they will die of something else.

The PROSPER study¹ concluded that men over 75 had a decreased mortality from cardiovascular disease, but that all-cause mortality was unchanged: they died of cancer instead of cardiovascular disease.

I for one would rather die of a heart attack than metastatic cancer. Lifetime risk of cardiovascular disease may be of interest to cardiologists but as a GP I think it is a useless parameter to discuss with patients.

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Important questions about healthcare expenditure and the allocation of resources in the last year of life

Dear Sir,

The authors of the recent article *Healthcare services funded by Counties Manukau District Health Board for people in the last year of life* are to be congratulated for a balanced and considered discussion that raises important questions about healthcare expenditure and the allocation of resources in the last year of life.¹

New Zealand, like many industrialised countries, faces the challenge of a rapidly aging population. Combined with a declining workforce, the question of how healthcare resources are allocated when those resources are finite (basically who gets what and why) must be seriously addressed if our health care system is to continue to protect and promote the health of patients and the public.² Of course there will be those who view any such questioning as a thinly veiled attempt to seek to implement rationing driven by a variety of measures—age being the most obvious and contentious.

The authors however make a compelling point: current trends in health care expenditure are unsustainable in the long term in New Zealand. Furthermore, adverse outcomes such as physical distress can be associated with aggressive management in individuals whose prognosis is poor. Whilst they claim that high-cost interventions provided near the end of life ought to be considered in light of several factors—clinical, cost of utility, and patients' expectations, it is focusing on the latter that would seem particularly fruitful in helping determine what is appropriate for individuals and their families near the end of life.

One area that merits serious consideration is promoting discussions with individuals around their preferences for medical treatment near the end of life. This is perhaps best situated in the primary care setting where patients and their general practitioner have a strong trusting relationship, often developed over a number of years. Such a setting may also be more comfortable for patients than the hospital environment. A patient may feel less pressured and more relaxed talking with their GP than a discussion with a clinician in hospital—especially if their admission was sudden and unexpected. Furthermore, initiating such discussions at the primary care level allows individuals to include their partners and other family members in decision—making if they so wish.

However, promoting such discussions must be seen in a much wider context of greater financial and moral support for primary care. Encouraging individuals to think about what they may want for themselves in the context of their own health towards the end of life cannot happen productively in a 15-minute consultation.³ It is perhaps self evident to comment that the medical preferences of individuals must be included in their medical records so that both wanted and unwanted interventions are clearly acknowledged and outlined.

Far from being an exercise in rationing healthcare resources, the opportunity to engage with individuals about what is important and valuable to them at the end of life truly reflects what lies at the heart of the patient doctor relationship: that of respect and trust.

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Use of Asthma Control Test (ACT) affects New Zealand primary care doctors' perception of asthma control

The Asthma Control Test (ACT) is not routinely used in New Zealand primary care. An audit of asthma control using ACT was undertaken by 11 primary care doctors in 3 medical practices in New Zealand, in order to assess the level of control in patients with asthma and to determine if the results of the ACT score corresponded to the doctor's previous assessment of their patients asthma control.

Asthma affects around 1 in 6 New Zealanders, causing the third largest number of years lost to disability.¹ The Asthma Control Test is a recently validated questionnaire, consisting of 5 simple questions.²⁻⁵ A score of 20–25 indicates well or completely controlled asthma, 15–19 indicates somewhat controlled asthma and a score of 5–14 identifies patients with poorly or uncontrolled asthma.

The practices undertaking the audit were based in a large city, a mid-size town and a rural area with a larger Māori population. Each practice, which had not previously used the ACT score, audited approximately 50 consecutive adult asthma patients and the audit consisted of:

- Collecting demographic information
- ACT score
- Whether patient had had a severe exacerbation in the previous year (defined as an out-of-hours or A&E visit, hospital admission or course of oral corticosteroid)
- Patient treatment group (short-acting beta-agonist (SABA) only, inhaled corticosteroid (ICS), or ICS & long-acting beta-agonist (LABA))
- A question regarding whether the ACT score led to a different assessment of the patient's asthma control than previously appreciated

The patients had a mean age of 50 (range 15–86), 38% were male, and 70% were New Zealand European, 20% Māori, 6% Asian and 4% other ethnicity. 19% were current smokers, 28% were former smokers and 53% had never smoked. 40% had had a severe exacerbation in the previous year and 17% were taking SABA only, 40% were taking ICS and 42% were prescribed both ICS and LABA.

ACT score results are presented in Table 1.

Table 1. ACT score results

Variables		ACT score (mean)	% ACT score >19	% ACT score 15–19	% ACT score <15
Overall	N=152	18.9 (range 6–25)	53%	28%	19%
Gender	Male	19.2	47%	42%	11%
	Female	18.7	56%	22%	23%
Age	<30	18.8	48%	35%	17%
	30–49	18.2	51%	20%	29%
	50–69	19.4	56%	28%	16%
	>69	19.3	53%	34%	13%
Ethnicity	New Zealand European	19.6	58%	28%	14%
	Māori	17.7	41%	28%	31%
	Asian	19.0	63%	25%	13%
	Other	13.7	17%	17%	67%
Smoker	Current smoker	17.4	36%	36%	29%
	Former smoker	18.4	46%	27%	27%
	Never smoked	19.8	62%	25%	13%
Exacerbation in previous year	Yes	17.9	44%	33%	23%
	No	19.6	58%	25%	16%
Treatment	SABA only	20.4	67%	37%	7%
	ICS	19.0	58%	22%	20%
	ICS & LABA	18.6	44%	37%	19%

The mean ACT score was 18.9%, corresponding to somewhat controlled asthma. 53% had well or completely controlled asthma, 28% had somewhat controlled asthma and 19% had poorly or uncontrolled asthma. ACT scores were slightly lower in Māori, smokers, patients taking more treatment and patients who had had a severe exacerbation, but no important differences were seen with respect to gender or age.

Of particular interest was the finding that 18% of patients had an ACT score indicating asthma that was better controlled than previously appreciated, 36% having an ACT score indicating asthma less well controlled than previously appreciated, and 45% having an ACT score indicating asthma control that was as expected.

Therefore the main finding of this audit was that in around half of patients, the ACT score was different to that anticipated by the doctor, being worse than expected in around 2/3 of cases and better than expected in around 1/3 of cases.

The use of the ACT score by primary care doctors may lead to a more accurate assessment of asthma control.

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Advocacy for General Practice

Is there anyone in the New Zealand medical system who is advocating for the General Practitioner (GP)—the medically trained primary health worker who most people still actually consider as “their” primary doctor; and the initial and frequently final source of advice, treatment and information regarding their health and wellbeing?

Electronic newsletters report that clinical leaders are being nurtured, selected, groomed and dare I suggest indoctrinated for a brave new world of medical care provision but there seem to be very few reports advocating for a fair and equitable provision of funding, locums, and support across the board for the GP *per se*.

The provision of a professional body for rural hospital generalist doctors suggested there were about 120 rural hospital doctors in New Zealand in 2007.¹ The Medical Council of New Zealand in its 2009 workforce survey had 2970 doctors in general practice as their main site of work, with a further 124 in accident and medical practice.² The majority of GPs work in small group collectives, and many practices work from houses that have been converted into medical rooms. The majority of GPs work among city or urban populations of a wide range of ethnicities and socioeconomic status.

There appears to be a perception among the public, administrators and some clinical leaders and educators that the full scope of real general practice is only experienced or provided by practitioners working in remote or rural areas. That would suggest there are only about 120 real GPs in New Zealand. Those who chose to work in those areas need to be congratulated, remunerated and supported, but so do GPs working in urban areas and provincial New Zealand who may not have a well defined cohesive population that can be succinctly classified as remote, low decile, migrant, indigent or refugee.

The average GP has 20 children born into the Practice every year and will have 10 to 12 deaths per year. They will have 1 new case of prostate cancer every 4 years and 2 deaths from cervical cancer every 50 years. Their patients will have 4 to 6 abortions a year. The GP will likely have over 100 patients on statins and an undetermined number on anti-hypertensives. In the over-65s there will be 3% of the population who have had a melanoma excised, 6% who will have had a squamous cell carcinoma removed, and 12% will have had a basal cell skin cancer histologically diagnosed.

About 10% of GP consultations will be for accidents and attract a patient subsidy from the Accident Compensation Corporation (ACC). Following from the two ACC-endorsed provider network trials in 2000 and 2003, time-based payments that have been indexed to the CPI have been provided to Accident and Medical (A&M) Clinics and rural practices.³

In 2000, the average ACC consultation fee was \$48.57. These payments have generally increased the ACC subsidy toward patient fees by up to 100% over the regulated fees which were last set in 2005. A review of the ACC payments for 2009 to

the Whanganui Accident and Medical Clinic showed an average contribution per ACC consultation at the clinic was \$79.40 plus GST.^{4,5}

Under the present cost of treatment regulations the ACC fee paid for a GP consultation is \$29.15 + GST.⁶ The ACC subsidy for a first specialist consultation for a non operative (talk) specialist is \$88.88 (plus GST).⁷ From 1 October 2010 the ACC payment to a GP for a closed reduction of a fractured finger is \$39.65 (incl GST) and for a specialist to do the same thing is \$97.77 (incl GST).

In Wanganui, the commonly advised standard ACC patient copayment fee is of the order of \$20.00—\$25.00 because the A&M Clinic has an advised fee of \$10.00 for accidents for patients being seen from 8am to 9pm. These levels of remuneration are unsustainable, anticompetitive, unfair and discriminatory.

Does ACC recognise vocational registration of GPs? There should be a financial recognition. Do the general practice advocates recognise the anomalies here and the institutionalised discrimination against general practice ? What advocacy has there been since 2004 when the time payment rates for A&M Clinics was extended to the rurals?

Urban General Practice also has expenses and their patients should be entitled to the same rate of subsidy. If the problem can be equally managed in general practice as an A&M Clinic why the huge difference in patient entitlement and subsidy? Do the fees review committees take cognisance of the cost constraints due to preferentially funded A&M Clinics in their fee determinations on practices?

The National Party Government has determined to open up workplace accident insurance to private insurers. Will there be an alignment of patient and medical fees to give a level playing field for the accident victim, employer and service providers?

Is there a role for the Commerce Commission to investigate anti-competitive behaviour in the accident treatment market place? Is there a role for the Human Rights Commission to look at why some patients are financially discriminated against by where they live and what subsidy distortions are in place?

There is definitely a role for the General Practice leaders and advocates to lobby for a fair and equitable payment and remuneration system for GPs and their patients.

Bill Douglas
Wanganui

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Can many anterior cruciate ligament (ACL) ruptures heal without surgery?

As a doctor who has spent 18 years working in accident medicine I was intrigued to read the letters by Drs Cooper and Hooper with regard to ACL reconstructions. I have had considerable exposure to knee injuries and am familiar with the routine use of ACL reconstructions.

I was surprised by Dr Cooper's claims as I assumed that such a routine procedure must be based upon sound evidence.

The 1995 paper states that 40% of ACL ruptures heal completely with conservative management. Professor Hooper states that these injuries "rarely heal" without surgery. These assertions are mutually exclusive.

In a subsequent letter Prof Hooper provided an unreferenced and largely irrelevant missive in which he made no comment as to the validity of the 1995 paper. He asserts that a conservative vs operative trial would be unethical. This—assuming all of the above to be true—is nonsense.

It is an important issue for three reasons.

- First—the incidence of iatrogenic osteoarthritis resulting from the procedure may well exceed that arising from conservative management.
- Second—it diminishes trust in an authority which appears to have a casual attitude to careful analysis.
- Third—ACC dollars are rare and there is an urgent need to spend them wisely.

Dr Andrew Montgomery
Remuera, Auckland

Revenge of the ACL

In 2005 the Cochrane Collaboration reviewed thousands of articles on ACL injuries.¹ They found only two quasi-randomised studies that addressed the basic question as to whether ACL ruptures should be operated on or not. The methodological quality of both these trials was rated poor, and the surgical techniques undertaken are not current practice.

One quasi-randomised trial of 167 patients with complete ACL rupture found no difference in return to sports activity.² The other trial of 157 patients with ACL injury found no difference in long-term functional outcome, but surgery associated with longer recovery, postoperative complications and less knee instability.³

Professor Hooper's claims of overwhelming literature support for ACL reconstruction are not supported by the facts. Although I have presented him evidence to the contrary, Professor Hooper clings to the orthopaedic myth that ACL ruptures rarely heal. This is simply not true in my opinion, as with most damaged structures in the body the ACL heals with time.⁴⁻⁷

I agree with Professor Hooper that getting ethical approval now for an RCT comparing conservative and surgical treatment of ACL ruptures would be difficult. It is hard to imagine an ethics committee approving a trial that involved surgery to a damaged structure that was going to heal anyway.

Professor Hooper's justification for ACL reconstruction by comparison to the "success" of total hip joint replacement couldn't have been more mistimed. This week the *Lancet* has reported one of the biggest disasters in orthopaedic history; the pain and disability caused by upwards of 93,000 ASR hip joint prostheses and the dubious ethics of the orthopaedic surgeons involved in the promotion of the device and the attempted cover-up of its failure.⁸

At present, standard treatment for a ruptured ACL in an otherwise healthy young knee is to reconstruct it.⁹ Not only is this surgery unnecessary as the ACL will usually heal anyway but it delays recovery of the injury.³ For those few ACLs that don't heal, there is no evidence that a reconstruction leads to a better outcome.^{2,3} Furthermore the long-term consequences of reconstruction surgery remain unknown.

The millions of dollars that ACC spends on ACL reconstruction each year are at best wasted, but more than likely will lead to more expense for ACC from medical misadventure claims.

It is time for the Ministry of Health to proscribe this procedure. It seems to me to be of no benefit to the patient, so why is it being done?

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Consequences of a flawed epidemiological approach to air quality regulation

In posing their question "Does health evidence support or undermine our regulatory approach to air quality?" Longley and Hales,¹ after having read our papers²⁻⁴ and individual responses⁵⁻⁷ to an accompanying editorial,⁸ are gracious enough to concede that the existing approach is "far from perfect". However, they then proceed to reveal an inherent bias by stating that our views "are based on a selective and incomplete understanding of the epidemiological evidence and do not justify major changes to current policy". I disagree and would respectfully suggest that the latter viewpoints are a matter of opinion still to be resolved once and for all.

Given that "low level" air pollution epidemiology is a relatively new, inherently controversial, branch of science requiring of necessity a multidisciplinary approach, all the relevant evidence should be weighed objectively before firm conclusions are drawn by anyone. As it is, the New Zealand Government has adopted a decidedly internally conflicted regulatory policy to the detriment, I believe, of science and the public generally.

Regarding the observed "'persistence' of all the world's leading authorities" concerning their presumption of equal toxicity for the same mass of fine particulate matter [PM₁₀, PM_{2.5}, etc], such behaviour surely is a matter of regulatory expediency. That the intrinsic unhealthiness/toxicity of air-borne particulate matter varies according to source, composition, exposure, etc. is not unexpected.^{2,4,9} Consequently, for WHO to decry—as summarised under "...these guidelines are neither standards nor legally binding criteria..."¹⁰—formal standards written around a generic index such as PM mass seems entirely appropriate in the circumstances.

Concerning the purported absence of suggestions pertaining to "quantitative solutions likely to provide greater effectiveness or efficiency compared to the current regime" this ignores well-documented efforts over several years by ourselves to this end. Meanwhile, potentially useful debate has undoubtedly been stifled by the authors/sponsors of the 2005/2007 HAPINZ reports deciding not to correct errors and other shortcomings identified subsequently.^{11,12}

For Longley and Hales to maintain that methodological flaws are completely absent is, based on evidence already in the public domain, simply unsustainable. Furthermore, the government's publicised intention to update the HAPINZ report[s] signifies that all is not well therein, albeit without providing any clear indication of the extent to which the proposed review will address the key scientific and related issues that arise..

For Longley and Hales to claim that the mortality effects attributed to PM₁₀ by both Kingham and the HAPINZ study are viewed as being overstated when "a recent cohort study suggests that in New Zealand, the long-term effects of air pollution on mortality are similar to those found in overseas studies", misrepresents my position. Primary data from both long-term ["chronic"] and short-term ["acute"] exposure to air

pollution typically are obtained in the form of error-bound mortality associations corresponding to stated increments of measured PM air pollution. To the extent that they may be informative per se, such associations are often accepted at face value.

Where I take issue in particular and where Kingham and the HAPINZ study go astray in my opinion is when the original results are reinterpreted to yield precise numbers of lives lost/saved e.g. per year due to changed levels of air pollution notwithstanding that the aforesaid associations are tenuous/small in size compared to the main cause[s] of death as officially recorded. Also, whereas in the latter case the circumstances surrounding the demise of individual people are identified in considerable detail, in the former case since the enumerated deaths are derived statistically, such questions are of little account

Finally, as alluded to in my paper,³ ascribing significant positive \$ values to changed public health circumstances attributable to reduced levels of air pollution when, as in the present case, the lives 'saved' are those of elderly people 65 years and older, is highly contestable. This is because, rather than a benefit as normally understood, such ageing needs to be regarded as a net cost expressed in monetary terms.^{13,14} For this reason alone, expenditure justified by conventional [i.e. positive value of life saved/lost-type] benefit-cost analysis¹⁵ related to enforced control of air pollution deserves to be regarded as fiscally disadvantageous overall.

John Hoare
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Nicotine replacement therapy in grocery stores; but wait, there's more

In their letter—*Promotion of nicotine replacement therapy and smoking cessation services at grocery stores*—Williman et al suggest selling nicotine replacement therapy (NRT) through small convenience stores to make it more accessible.¹ Agreed; any measure that can switch people away from smoking should be considered and several measures should be attempted. However there are some barriers, under the current scheme, that would need to be overcome.

In order to provide NRT cheaply under the current scheme, retailers would have to become Quit Card providers. At present, only registered health professionals can become Quit Card providers; this programme would need to change. Quit Card providers also need to be trained in smoking cessation, and finally Pharmac would need to approve a scheme to allow a funded medicine to be dispensed through general retail stores. Pharmacists might oppose this. One answer is to sell NRT through retail at full mark-up; though this could make it less appealing to the smoker.

Another option, and one with potential advantages for smokers and retailers alike, is the sale of non-pharmaceutical products. Retailers may benefit from selling products that can compete with cigarettes for sales and are healthier alternatives to smoking. Smokers would then be presented with alternatives to smoking in the comfortable environment where the majority of them normally purchase cigarettes.

Additionally, the widespread sale of tobacco alternatives might provide a real opportunity to force a reduction of the number of cigarettes that can be sold and could become a lead up to a total ban on cigarette sales.

Swedish snus and e-cigarettes are two non-pharmaceutical products worth considering. The use of these products is straightforward and there would be no need to train retailers as expert dispensers.

Swedish snus is wet-cured tobacco supplied in small tea-bag like sachets. Nicotine from Swedish snus is absorbed through the oral mucosa, much like using nicotine gum. Swedish snus is pasteurised and this process reduces tobacco-specific nitrosamines that are known carcinogens. It should be noted that, due to the uptake of snus, rates of smoking and cardiothoracic disease among males in Sweden are among the lowest in the Western World.^{2,3}

E-cigarettes are a small tube that have the appearance of a cigarette and can deliver heated nicotine vapour to the user. E-cigarettes also deliver propylene glycol, which provides white mist to the otherwise invisible nicotine. The e-cigarette does not produce any smoke. Nicotine and propylene glycol are considered safe for human use and are approved by most OECD governments.

Swedish snus and e-cigarettes are not pharmaceuticals and therefore the range of evidence for their efficacy and safety is somewhat limited. There is evidence that the continued use of nicotine beyond smoking may increase the risk of pancreatic cancer.⁴

This evidence, and a suggestion that these products will appeal as a gateway for young people to smoke, has led to a reticence to legitimise them. These risks need to be weighed against the known harms of continued cigarette smoking. The Ministry of Health has recently endorsed e-cigarettes by saying they are “far safer than cigarettes”, and there is no doubt in many scientific minds that Swedish snus and e-cigarettes will save lives.

The last New Zealand Tobacco Use Survey suggests that an estimated 650,000 adults continue to smoke and put their health in jeopardy; this includes fifty percent of the Maori female population.⁵ Every possible measure, including wider access to pharmacotherapies and smokeless tobacco alternatives, should be considered and enacted as soon as practicable.

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Editorial. Our scale of fees

Published in NZMJ 1912 May;42(11):131-3.

In the present age, and particularly at the present time, there is a world-wide difficulty being faced—a question which is at the root of the great industrial upheaval in the Old Country, as also of our own little strikes in New Zealand—which has for its basis the question of adequate pay for work done. Each side is struggling to get the best terms for itself—the one the best work for the least pay, and the other the best pay for the least work.

We, as a profession, are, in the main, only spectators in this great struggle, but we feel the effects of it in the resulting greater increase in the cost of living. We feel this directly and indirectly in the greater cost in running our practices and in the lessened capacity of the patients to pay adequate fees, yet it is an undisputed fact that in spite of all this, the tendency in our professional fees is to a general lowering, more especially in regard to surgical work. The reason for this, in our opinion, is not fait to seek, and lies in the common cause of all our complaints of a professional nature: our own apathy and carelessness.

There are few, we think, who do not consider that midwifery is a poorly paid branch of our work. And the fact that it is so badly remunerated is often a remote reason that it is not always conducted as it should be. All midwifery has practically dropped to three or four guinea cases, irrespective of the time involved and the trouble and anxiety that often ensue. We are of opinion that this fee should be the minimum for the night attendance at a normal labour and through the normal puerperium. Yet though we are all unanimous that it is badly paid work, we are equally of one mind that it is now impossible to raise the fee.

We have cited this as an example of what is now a process with regard to most of our fees. Certain operations are becoming so common which in themselves differ little or no more than do cases of midwifery with each other—at any rate as far as the public can tell—and there is the same tendency to group all such similar operations alike, just as with midwifery and also similarly to lower the fee to a common inadequate one. For, after all, it is the public who will eventually decide the fee for us if we do not first do so ourselves; for just as when doctors disagree the patients usually suffer, so when the doctors are careless about their own affairs the patient suffers.

Take, for instance, the operation for appendicitis. A dozen cases may be operated on by as many surgeons, and probably in no two instances would the charges be the same. The surgeon never knows what the others charge, but provided those patients meet each other, as we know they do—on the "birds of a feather" rule apparently—they all compare notes, especially regarding the fee. As far as they know the conditions were the same, but their expenses vary considerably, perhaps quite properly so; but as often as not the one who has been operated on by a comparatively unknown surgeon has been charged a higher fee than has he who engaged a leading surgeon. There has been no wilful undercutting, but only the inevitable result of

there being no standard, scale of fees which is of any practical use, and partly often because the better recognised surgeon can now afford to take a less fee than his fellow-practitioner, though ignorant of the fact that he is doing so and not thinking of, or unaware of the effect his action has upon his fellow practitioner and upon the succeeding generations of his profession. The same might be said of most recognised operations—hernia, carcinoma of the breast, hysterectomy, etc., which usually run a course comparable to the course of a midwifery case.

The sum total of all this is a tendency to lower the fees unnecessarily, and this is obtaining throughout all our work. Even distance is being annihilated mainly by reason of the motor, and our radius of 10/6 visits is now so wide that we will soon have a legitimate protest by those living near by against being charged the same fee for a visit as those a mile or more away.

We consider we have done our duty in raising this question before the position becomes so involved that nothing can be done, and we are of opinion that the Council of this Branch should immediately set up a committee to investigate the matter and collect information and opinions with a view to an early revision of our scale of fees and the provision of a scale that would be more useful and applicable than our present one, which is so vague that each one has to be a law unto himself.

One very great advantage would accrue from having a somewhat standard scale of fee for any stated operation in that the surgeon of pre-eminence could, notwithstanding, charge a much larger fee, as we think he should do, leaving the average adequate fee to be charged by the average surgeon. For the public often gauges the skill of the surgeon by the size of the fee, and many a one has lost some respect for charging a small fee for what, in the patient's mind, was a great operation, and for which he was expecting to pay at his own valuation.

Alcohol consumption and cardiovascular disease

Opinions on this topic vary widely and this paper reports on a study of the specific question—is alcohol consumption associated with a reduced risk of multiple cardiovascular outcomes? This meta-analysis reviews 88 studies which compare cardiovascular outcomes in alcohol drinkers versus non-alcohol drinkers. Their results showed that light to moderate alcohol consumption is associated with a reduced risk of multiple cardiovascular outcomes. A companion paper by the same authors reports on the effect of alcohol use on circulating biomarkers associated with risk for coronary heart disease.

This analysis of 44 studies shows that moderate alcohol consumption produced favourable changes for selected biomarkers, significantly increasing circulating levels of high density lipoprotein cholesterol and adiponectin and decreasing levels of fibrinogen. For the record light to moderate is defined as 1–2 standard drinks per day.

BMJ 2011;342:671 & BMJ 2011:342:636.

Risk factors for dementia

This paper reviews the relationship between several lifestyle factors and various medical conditions and the subsequent development of dementia. Smoking increases the relative risk of developing dementia twofold or more and obesity is also an appreciable risk factor. Physical activity and cognitive reserve (i.e. intelligence, occupation and education) are associated with a lower risk.

Somewhat surprisingly alcohol consumption matches these by being associated with a relative risk of 0.74. Midlife hypertension, hyper-cholesterolaemia, diabetes and previous stroke were all very significant in an association with development of dementia.

Intervention by treating hypertension probably resulted in a decreased risk but treatment with vitamin B12, folate or statins was not effective. So, keep fit and mentally active, lose weight if you are obese, stop smoking and keep on drinking alcohol with moderation.

BMJ 2011;377:1019–31.

Traumatic brain injury with increased intracranial pressure—a role for decompressive surgery?

This paper from Australia reports upon a prospective study which sets out to clarify whether decompressive craniectomy improves the functional outcome in patients with severe traumatic brain injury and refractory raised intracranial pressure. 155 patients with intracranial hypertension caused by traumatic head injury were randomised to decompressive surgery or standard care, having failed to respond to first line treatment.

They report that decompressive craniectomy decreased intracranial pressure and the length of stay in the ICU but was associated with more unfavourable outcomes. Very disappointing as presumably the researchers expected the opposite conclusion.

N Engl J Med 2011;364:1493–502.

US nurse practitioners to the rescue?

Thirty-two million Americans become eligible for treatment within the next few years under healthcare reform passed last year by the Obama administration. That's good. However this extra weight-load is combined with a forecast shortage of 40,000 family doctors by 2020. This has led to about 28 states considering changes to their licensing requirements to allow US nurse practitioners to expand their role at the workplace, viz—make diagnoses, to order tests and referrals, and to write prescriptions.

These proposals have been supported by senior policy advisors of the American Nurses Association and also received an important endorsement from the *Journal of Family Practice*, which is read by many practising family physicians. Not unexpectedly, the more conservative Academy of Family Physicians say that the proposal does not adequately address the scope of patient safety.

We await further developments with interest as a similar situation is brewing closer to home.

Lancet 2011;377:625–6.

The downside of medical tourism

Medical tourists are people who cross international borders for the exclusive purpose of obtaining medical services. The reasons for such tourism include rising health costs in developed countries, cheaper costs in third world countries and ready air travel facility. This paper from Thailand points out that their country provides medical services for more than 400,000 such people each year.

In Thailand, medical tourism has both positive and negative effects. For the Thai economy, medical tourism generates a value added approximately equal to 0.4% of the GDP. The downside is that providing these services has exacerbated the shortage of medical staff by luring more workers away from the private and public sectors towards hospitals catering for foreigners. This has raised costs in private hospitals substantially and is likely to raise them in public hospitals and in the universal healthcare insurance covering most Thais as well.

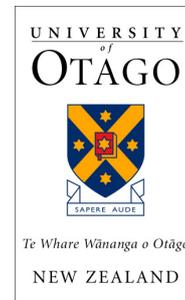
The “brain drain” may also undermine medical training in future. A very serious downside which may also be affecting other medical tourist sites in Asia.

Bull World Health Organ 2011;89:336–44.

**University of Otago Faculty of Medicine
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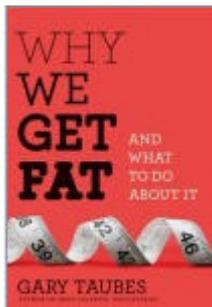
Applications close on **15 July 2011** with the Department Manager, Department of Women's & Children's Health, Dunedin School of Medicine, PO Box 913, Dunedin 9054, from whom further details may be obtained (wch.admin@otago.ac.nz)



Why we get fat—and what to do about it

Gary Taubes. Published by [Alfred A Knopf/Random House Inc](#) (New York), 12/2010. ISBN 9780307272706. Contains 272 pages. Price US\$24.95

The author has written extensively on diet for *Science*. This book shows that our thinking about being overweight is based on misconceptions and should be required reading for all healthcare professionals.



It covers why we get fat and the dietary causes of heart disease and diabetes. I was taught the hypothesis that high saturated fat is a cause of raised cholesterol and therefore heart disease, so a low saturated fat diet is required. The implication of a low saturated fat diet is a high carbohydrate diet. However, the Pima Indians, Inuit and Tokelauans all lived on a high-fat, high-protein diet and did not get fat or develop diabetes. With the introduction of sugar and flour into their diet, they became fat and developed diabetes.

The simple answer of why we get fat is carbohydrates make us fat, but protein and fat do not. The low saturated-fat diet is known not to work and perhaps we have been giving patients the wrong advice.

Some eat more than many others, yet do not gain weight, others eating the same amount would gain weight. We tell our patients that to reduce weight they should eat less and exercise more. If we overeat by 20 calories a day (a bite of a croissant) then the result would be to gain 1 kg per year. According to the author there is little evidence to support that the number of calories we eat has any effect on our weight. If we believe people get fat because they overeat then we are blaming their mental state and leaving out physiology. Therefore Newton's first law of thermodynamics regarding conservation of energy does not apply to calories in/calories out.

The belief that saturated fat causes heart disease, led to the idea that carbohydrates prevent it. The arguments against a low-carbohydrate diet are:

- It is a fad and calories in/calories out counts but we know that physics has nothing to do with diet;
- It is unbalanced, and there is no nutriment in refined carbohydrates;
- The brain requires glucose but it is natural for the brain to use ketones as it does every night.

The official embrace of the low-fat, high-carbohydrate diet has coincided with the recent epidemics of obesity and diabetes. The MRFIT low-fat saturated trial failed to reduce heart disease. A 2001 Cochrane collaboration stated that modification of dietary fat has shown inconclusive evidence to reduce heart disease. The Women's Health Initiative showed that the women who cut total and saturated fat by 25% lowered their total and LDL cholesterol slightly, with no benefit on heart disease, cancer, and fat accumulation.

Body fat is exquisitely regulated. The absorption of a carbohydrate load stimulates insulin release. Insulin has a direct lipogenic effect on adipose tissue: it activates lipoprotein lipase on fat cells; promotes entry of glucose into fat cells; and inhibits release of fatty acids. Insulin allows muscle to use glucose; liver cells to convert glucose to fatty acids; fat cells to get fatter; and fat stays in fat cells until insulin level drops. The only way to get fat out of fat tissue, so as to burn it, is to lower insulin. Secretion of insulin will store fat, low insulin will allow mobilisation of fat.

If you are predisposed to get fat and wish to remain lean then you have to restrict carbohydrates to keep the blood glucose and insulin low. Some people secrete more insulin in response to the same carbohydrate load and store more fat. Insulin resistance requires more insulin to be secreted, so more fat is stored. It is well known that fat cells are more sensitive to insulin than muscle cells; therefore mobilisation of fat requires low insulin.

You do not lose weight because you reduce calorie intake but because you reduce the foods that make you fat. Calorie-restricted diets fail and increased exercise programmes fail, because the resultant hunger leads to failure.

To reduce body fat, carbohydrates need to be restricted.

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