The Northern Territory's Living With Alcohol Program, 1992-2002: revisiting the evaluation

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ABBREVIATIONS

ABS:	Australian Bureau of Statistics
AIHW:	Australian Institute of Health and Welfare
ASGC:	Australian Standard Geographical Classification
ASR:	Age Standardised Rate
CPI	Consumer Price Index
ERP	Estimates of Residential Population
ICD:	International Classification of Diseases
LWA:	Living With Alcohol program
NAIP:	National Alcohol Indicators Project
NDRI :	National Drug Research Institute
NDSHS:	National Drug Strategy Household Survey
SLA:	Statistical Local Area
WHO:	World Health Organisation

EXECUTIVE SUMMARY

INTRODUCTION

This report documents the results of a new analysis of the Northern Territory's comprehensive program to reduce alcohol consumption and alcohol-related harms known as the *Living With Alcohol* (LWA) program. The LWA program was introduced in 1992 and was initially funded by the imposition of a small levy on all alcoholic beverages sold in the Northern Territory (NT) containing 3% alcohol by volume or greater. The LWA Levy effectively raised the retail cost of these beverages by about 5 cents per standard drink. As a direct result of a High Court ruling, the LWA Levy was removed in 1997 which in turn resulted in a fall in the real price of alcoholic beverages with more than 3% alcohol by volume. Nevertheless, LWA programmes and services continued to operate to 2002 and were funded from redirected taxes collected by the Commonwealth.

The public health, safety and economic impact of the LWA program was initially evaluated by Stockwell and colleagues (2001) who found that during its first 4 years of operation, the LWA program was associated with a significant cost saving to the Northern Territory (NT). However, the evaluation was unable to determine:

(i) the relative impacts of the LWA programmes and services versus the Levy on levels of alcohol-related harms;

(ii) whether the positive impacts lasted beyond the first 4 years; and,

(iii) whether macro economic factors other than the Levy may have been responsible for part or all of the cost savings.

The current study improves upon the earlier evaluation by using a more stringent methodology, a longer time series of data and providing comparisons between Indigenous and non-Indigenous NT residents.

STUDY DESIGN

Retrospective deaths data between 1985 and 2002 were used to evaluate the impact of both the LWA program and the Levy that occurred in the NT between 1992 and 2002. The study design and the timing of these two interventions allowed three comparisons;

- alcohol-attributable death rates 'before' versus 'during' the introduction of the combined Levy and LWA program (Levy/LWA program);
- alcohol-attributable death rates 'during' versus 'after' the introduction of the combined Levy/LWA program; and,
- alcohol-attributable death rates 'before' and 'during' the full length LWA program, including a period when the Levy was removed.

Both internal and external control series were included in the analyses. Simultaneous trends in non-alcohol related deaths in the NT were used to control for any changes 'internal' to that region that might have affected overall death rates but which were unrelated to either of the interventions. Trends in alcohol-attributable death rates in a control region (northern Queensland and Western Australia) were used to adjust for any macro changes non-specific to the NT. Two other possibly confounding events were also accounted for in the analysis; the NT Cask wine Levy and the implementation of a 0.05mg/ml legal blood alcohol level for driving. Alcohol-attributable death rates were calculated using year and region specific population alcohol aetiologic fractions (Chikritzhs *et al.*, 2002). The impact of the LWA program and the Levy were considered in relation to acute (e.g. road injury, violent assault) and chronic deaths (e.g. alcoholic liver cirrhosis, cancer) as well as Indigenous and non-Indigenous NT residents.

SUMMARY OF MAIN FINDINGS

- This investigation has confirmed the results of an earlier evaluation (Stockwell *et al.*, 2001) suggesting that the LWA program resulted in significantly reduced alcohol-attributable deaths and financial cost savings to the NT.

- The combined impact of the LWA program and Levy resulted in an immediate reduction in *acute* alcohol-attributable deaths in the NT.
- In the absence of the Levy, the LWA program alone did not show a significant impact on *acute* alcohol-attributable deaths.
- There was no evidence of a short-term effect of the LWA program and Levy on numbers of *chronic* alcohol-attributable deaths.
- There was evidence of a longer term impact of the LWA program and/or Levy on *chronic* alcohol-attributable deaths.
- Both Indigenous and non-Indigenous residents of the NT experienced declines in *acute* alcohol related harms during the LWA program and Levy period.
- *Chronic* alcohol-attributable deaths among Indigenous NT residents did not appear to benefit from the LWA program or Levy but, Indigenous deaths identified as 'unrelated' to alcohol consumption declined significantly.

CONCLUSION

The results of this study present a strong argument for alcohol taxes combined with comprehensive programs and services designed to reduce the harms from alcohol. There are both short and long term benefits to be had from this combination of strategies. What is more, the benefits accrued due to a reduction in acute harms, are equally evident among Indigenous populations.

INTRODUCTION

The Northern Territory (NT) has a history of having the highest rates of alcohol related problems throughout the nation (Chikritzhs *et al*., 1999; Chikritzhs *et al*., 2000; Gray *et al*., 1999). At approximately 15 litres of pure alcohol per adult (15+yrs), per capita alcohol consumption in the Territory is about twice the national average (d'Abbs, 1993; Gray & Chikritzhs, 2000).

This report provides a new analysis of a comprehensive program to reduce serious alcohol related harm in the NT known as the *Living With Alcohol* (LWA) program. An earlier evaluation (Stockwell *et al*., 2001) estimated that there had been substantial reductions in the health and economic costs of alcohol misuse over the program's first four years. It was unclear the extent to which these apparent benefits may have been caused by the program, the special tax (Levy) on alcohol which funded the program or other prevailing economic factors. The present report is able to shed light on this issue by a) employing a longer time series of data including a period *after* the Levy was removed and (b) re-analysing the impacts of both the program and the taxes using adjoining areas of northern Queensland and Western Australia as control regions.

BACKGROUND

The Living With Alcohol (LWA) program was introduced in 1991, and officially implemented in April 1992 as a comprehensive strategy for the reduction of alcohol related harm in the NT. The main objective of the project was to bring NT levels of alcohol consumption and related harm down to national levels by 2002 (d'Abbs, 2004). This was to be achieved by multiple strategies including education, increased controls on alcohol availability and expanded treatment and rehabilitation services (d'Abbs, 2004).

The LWA program was originally funded by the imposition of a Territory specific Levy on the sale of alcohol products with more than 3% alcohol by volume. The Levy raised the price of a standard drink by 5 cents.[1] Levy funds were primarily used for various

[1] Increases by beverage type were: beer and pre-mixed spirits $0.20 per litre; wine and cider $0.48 per litre; spirits and fortified wines $1.60 per litre.

harm reduction programs and the implementation of new services including; drink driving advertising campaigns, several community education programs; night patrols for some areas; and, the addition of new sobering up shelters. Several changes to the NT Liquor Act (1978) and the Liquor Commission's liquor policy were also implemented during the life of the program including; reductions in prescribed hours for off-premise alcohol sales, increased power for licensees to remove intoxicated persons from licensed premises; and, known heavy drinkers who have caused injury to self or others or been arrested more than 3 times in the last 6 months to be prohibited from entering licensed premises.

Although the primary focus of the LWA program was to reduce alcohol related problems through focused harm reduction programs and services, changes in alcohol taxation that affect the real price of alcoholic beverages have been found, in their own right, to be a highly effective means of reducing aggregate alcohol consumption and related harms (Godfrey, 1997; Ludbrook *et al*., 2002; Osterberg, 2001). Hypothecated taxes such as the LWA Levy, i.e., specific taxes earmarked for expenditure on prevention and treatment programs, have been found to be particularly popular among the general public (Beel & Stockwell, 1993).

Unfortunately, a 1997 High Court ruling prompted by a dispute in New South Wales over state tobacco taxes prohibited states and territories from raising license fees and additional taxes on alcoholic beverages, tobacco and petrol. As a direct result, the LWA Levy was removed in August 1997 which in turn resulted in a fall in the real price of alcoholic beverages with more than 3% alcohol by volume. The Commonwealth continued to fund the LWA program by collecting an equivalent amount to that provided by the Levy which was then re-directed to the NT government. In this way, although the Levy was removed, the LWA programmes and services continued to operate as usual until the year 2000. After this time, LWA funds were dispersed directly to the existing programmes and services (d'Abbs, 2004).

The earlier Stockwell, Chikritzhs *et al*. (2001) evaluation used times series data to estimate changes in consumption, and alcohol-attributable mortality and morbidity in the NT over the first four years of the program, i.e. 1991/92 – 1995/96. Estimates of the numbers of lives saved and hospitalisations prevented were based on comparisons with

trends in non-alcohol-attributable conditions occurring in the NT during the same time period. The authors concluded that during the four years immediately following LWA implementation there were significant declines in; per capita alcohol consumption, rates of road fatalities and serious road injury, alcohol-attributable deaths and hospitalisations for acute conditions, as well as alcohol-attributable hospitalisations for chronic conditions. In dollar terms, a net saving of over A$124.3 million was attributed to the LWA program during the first four years of operation.

Importantly however, the authors qualified their conclusions by noting that during the time when the LWA programmes and services were being introduced, the Levy, which had increased the real price of alcohol in the territory, was also in operation. Thus, although a significant saving occurred immediately after the program began, it was not clear to what degree the reductions in alcohol related harm were related to the LWA program itself as opposed to the Levy that was also in operation at the time. It was also possible that economic factors unrelated to the LWA had impacted specifically on rates of alcohol consumption and related harm.

This report presents the results of a re-evaluation of the LWA program and builds on the previous study in several ways;

- The previous evaluation examined trends in consumption and related harms for the four years immediately following the implementation of the LWA program, i.e. from 1991/92 to 1995/96. The current evaluation extends the evaluation period to 2002.

- The longer time series includes several years after the Levy was removed – but during which time, the LWA programmes continued to operate, i.e. August 1997 – December 2002. This allows a comparison of the impact of the combined Levy/LWA program versus the LWA program only to be made.

- In order to verify an association between declines in rates of alcohol-attributable harm, the previous evaluation included an 'internal' control series, i.e. non-alcohol related deaths in the NT. The current evaluation includes an 'internal' control series as well as an 'external' control region, i.e. northern Queensland combined with northern Western Australia. This enhances the reliability of the

results by controlling for changes in rates of harm that may have been occurring throughout the rest of the country and which were not specific to the NT and the LWA program

- The alcohol-attributable death rates examined in the current evaluation were based on population alcohol aetiologic fractions specific to the NT with prevalence estimates derived from the 2001 National Drug Strategy Household (NDSH) survey (control region alcohol aetiologic fractions were also region specific).

- The current evaluation includes a comparison of the combined impact of the Levy/LWA program and the LWA program only on Indigenous versus non-Indigenous NT residents.

DESIGN OVERVIEW

This study used retrospective deaths data between 1985 and 2002 to evaluate the impact of both the LWA program and the Alcoholic Beverage Levy that occurred in the NT between 1992 and 2002. The study design and the timing of these two interventions allowed three comparisons;

- alcohol-attributable death rates 'before' versus 'during' the introduction of the combined Levy and LWA program;
- alcohol-attributable death rates 'during' versus 'after' the introduction of the combined Levy/LWA program; and,
- alcohol-attributable death rates 'before' and 'during' the full length LWA program, including a period when the Levy was removed.

Both internal and external control series were included in the analyses. Simultaneous trends in non-alcohol related deaths in the NT were used to control for any changes 'internal' to that region that might have affected overall death rates but which were unrelated to either of the interventions. Simultaneous trends in alcohol-attributable death rates in the control region were used to control for any macro changes non-specific to the NT. Alcohol-attributable death rates were calculated using year and region specific population alcohol aetiologic fractions (Chikritzhs *et al*., 2002). The impact of the program and the Levy were both considered in relation to acute and chronic deaths.

METHODS

DATA SOURCES

Information on deaths occurring in the NT between 1985 and 2002 were sourced from the Australian Bureau of Statistics (ABS) Mortality Datafile. The ABS Mortality Datafile codes age at death, sex, date of registration of death, date of death, cause of death and place of residence by statistical local area (Australian Standard Geographic Classification) and Indigenous status. For 1985 to 1997, causes of death (both primary diagnosis and any applicable external causes of death, E-codes) were recorded according to the *International Classification of Diseases 9th revision, Clinical Modification* (ICD-9-CM), deaths occurring between 1998 and 2002 were recorded using the *International Classification of Diseases and Related Harm Problems, 10th Revision* (ICD-10).

Estimates of residential populations (ERP) for men and women in five year age cohorts (0-4yrs, 5-9yrs…85yrs+) for the NT and control regions were obtained from the Australian Bureau of Statistics ERP.

ESTIMATING ALCOHOL-ATTRIBUTABLE DEATHS

English *et al*., (1995) and more recently, Ridolfo and Stevenson (2002) have identified diseases and injuries for which there is sufficient evidence to conclude that risky or high risk alcohol consumption is a contributing cause (about 40 different conditions). Each death recorded in the ABS Mortality Datafile is accompanied by several ICD codes assigned according to the underlying causes of death. Primary diagnostic and primary external cause ICD codes for each individual death were used to identify deaths from conditions that were either wholly or in part attributable to alcohol. Chikritzhs and colleagues (2002) have provided a list of all conditions currently known to be attributable to alcohol as well as their corresponding ICD-9 and ICD-10 codes (see Appendix A).

Following identification, alcohol related deaths were additionally aggregated according to age (0–14yrs, 15–29yrs, 30–44yrs, 45–59yrs, 60–74yrs, 75yrs+) and sex.

POPULATION ALCOHOL AETIOLOGIC FRACTIONS

Routinely collected mortality data provide no information about each individual's alcohol consumption. For a population, one method of estimating the total magnitude of alcohol-attributable illness or injury is to multiply the number of people with each particular condition by the alcohol population aetiologic fraction specific to that condition, then to sum the results. The population aetiologic fraction for a particular condition that is attributable to drinking is the proportion of cases with that condition in the population that can be attributed to such drinking

For conditions such as *alcoholic* liver cirrhosis, *alcohol* dependence and *alcoholic* psychosis, the alcohol population aetiologic fractions are 1. This is because such conditions, are by definition, wholly attributable to alcohol. For many other forms of injury and disease, such as assault, road crashes and stroke, the alcohol population aetiologic fractions are less than 1, because they are only partially attributable to alcohol. When a condition is not wholly attributable to alcohol (i.e. the population aetiologic fraction is less than 1), the population alcohol aetiologic fraction is calculated as a function of both the strength of the causal relation between a particular level of drinking and the condition (i.e., the 'relative risk'), **and** the population prevalence of the drinking levels of interest.

Moreover, the value of a relative risk depends on which alcohol consumption levels are chosen as 'exposed' (e.g. risky and high risk drinkers or all drinkers) and 'unexposed' (e.g. low risk drinkers or abstainers). Following the recommendations from Chikritzhs *et al*., (2002) this study has applied population alcohol aetiologic fractions based on comparisons between *abstainers* and risky/high risk drinkers.

According to Chikritzhs and colleagues (2002) it is crucial that alcohol aetiologic fractions reflect levels of alcohol consumption in the population to which they will be applied. Using a method described in the WHO Guidelines on alcohol monitoring (2000), population alcohol aetiologic fractions specific to the NT were based on drinking prevalence estimates from the 2001 NDSHS. These were then adjusted according to annual changes in per capita alcohol consumption – thus, the aetiologic fractions applied were both NT and year specific.

To estimate alcohol-attributable deaths, the numbers of alcohol related deaths in each age, sex and condition category were then multiplied by their year, age, sex and condition specific population alcohol aetiologic fraction.

ACUTE AND CHRONIC CONDITIONS

According to Chikritzhs and colleagues (2001; 2002), alcohol related conditions can generally be divided into two groups; '**acute'** and '**chronic'**. Conditions that tend to result from episodes of drinking to intoxication are referred to as 'acute' and are characterised by injuries (e.g., assault, road injury, drowning). Chronic conditions are those that tend to develop over many years of alcohol misuse (e.g., oropharyngeal cancer, chronic gastritis) and reflect degenerative disease states. The WHO guide (2000) highlighted the importance of distinguishing between acute and chronic alcohol-attributable conditions when monitoring and reporting on alcohol related problems. The National Health and Medical Research Council (2001) drinking guidelines also differentiate between short-and long-term drinking patterns in a similar way.

For this report, data were analysed separately for acute and chronic conditions with groupings largely following classifications identified by the WHO (2000). However, as Chikritzhs *et al.* (2001) have noted, few conditions are entirely acute or chronic in nature. They identified stroke and suicide as being particularly difficult to specify in terms of acute or chronic and their solution was to create a third category referred to as 'mixed'. For this study it was not feasible to create a third category for analysis. As an alternative, suicide was assigned to acute conditions (together with the majority of all other injuries); haemorrhagic stroke was also assigned to acute conditions while ischaemic stroke was assigned to the chronic group (See Appendix A for list of chronic and acute conditions).

INTERNAL AND EXTERNAL CONTROL SERIES

The analyses that follow consider changes in alcohol-attributable deaths rates in the NT in order to evaluate the impact of the LWA program and Levy on levels of such deaths. However, variations in the deaths rates may merely reflect variation in overall mortality

rates or the influence of factors other than the interventions being consideration (e.g. macro economic changes). In order to examine whether or not this was the case, both an 'internal' and 'external' control series have been included:

- trends in non-alcohol related deaths in the NT between 1985 and 2002 (internal control); and,
- trends in alcohol-attributable deaths in a 'control region' including northern Queensland and northern Western Australia between 1985 and 2002 (external control).

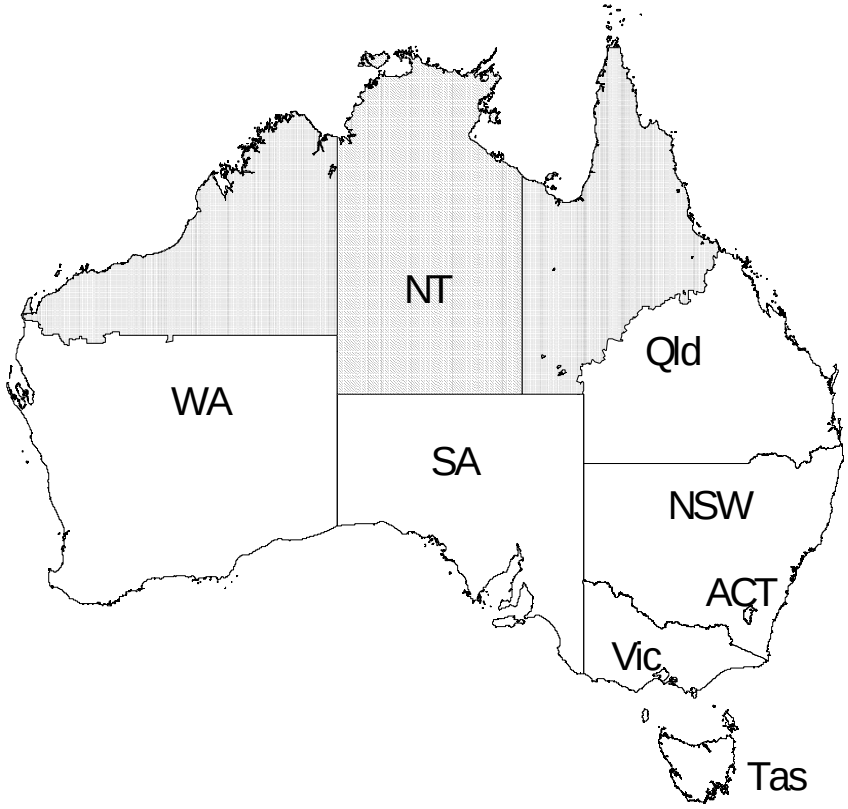
NT non-alcohol related deaths

For hospitalisations, it is possible to identify large numbers of admissions for conditions that are unrelated to alcohol consumption (e.g. Chikritzhs *et al.*, 2000; Jonas *et al*. 1999). However, alcohol is attributable to a large proportion of serious conditions that result in death, by comparison, conditions which have been identified as specifically non-alcohol related rarely result in death (e.g. genital prolapse, dental caries, constipation). What is more, the small numbers of deaths from conditions unrelated to alcohol limit their application. Therefore, non-alcohol related deaths have been designated as all deaths for which the population alcohol aetiologic fraction is zero (i.e. all remaining deaths), with the exception of several conditions known to be moderately or strongly related to tobacco consumption (trends in tobacco consumption may confound related trends). Lists of alcohol-attributable conditions, tobacco-related conditions and non-alcohol-related conditions can be found in Appendix A.

Control region alcohol-attributable deaths

In addition to an internal control, a simultaneous time series from a similar region was included that controlled for changes influenced by factors external to the NT such as may occur in a national economic downturn. The region was selected in order to be as similar as possible in climactic, demographic and geographic respects to the NT. An aggregate of the northern regions of Queensland and Western Australia (i.e., the remainder of the northern part of Australia) was identified as the most appropriate control region. Alcohol-attributable death rates for the control region were based on

region and year specific population alcohol aetiologic fractions, i.e., Western Australia and Queensland. Map 1 provides a geographical description of the control region.



Map 1

NT and control regions

Comparability between the NT and the control region

As shown in Table 1, the age and sex distributions of the NT and control region followed generally similar trends between 1986, 1992 and 2002, i.e. increasing proportions of older people and declining numbers of younger people. However overall, the NT indicated somewhat larger proportions of young people and fewer older people than the control region. It should be noted that all rates presented in this report have been age standardised in order to adjust for this discrepancy.

Table 1
Age and sex distributions in the NT and Control region, 1985, 1992 and 2002

	NT			Control region (northern Qld & WA)		
	1985	**1992**	**2002**	**1985**	**1992**	**2002**
Sex (%)						
Males		52.4	52.6		52.1	51.7
Females		47.6	47.4		47.9	48.3
Age (% of total)						
0–14 yrs	29.5	27.8	25.5	26.8	24.9	23.1
15–29 yrs	30.1	28.3	24.5	27.3	25.9	22.0
30–34 yrs	25.8	26.8	25.9	22.8	24.8	24.5
45–59 yrs	10.3	12.5	17.2	12.9	14.1	18.1
60–74 yrs	3.5	3.9	5.5	7.9	7.6	8.6
75+ yrs	0.7	0.8	1.3	2.4	2.6	3.6

As shown in Figure 1, Australian Bureau of Statistics (2003) estimates of changes in the Consumer Price Index (CPI) indicated that the capital cities of the NT (Darwin), Western Australia (Perth) and Queensland (Brisbane) experienced marked declines in CPI from 1991 to 1992 – this downward trend was not atypical to these regions but was experienced throughout the nation. In fact, the economic trend was similar for all capital cities between 1991 and 1998. Unfortunately these CPI estimates are limited to capital cities but should serve as a rough guide for the remainder of the state/territory. It is possible however, that the northern regions of Western Australia and Queensland experienced economic trends unlike that of their capital cities.

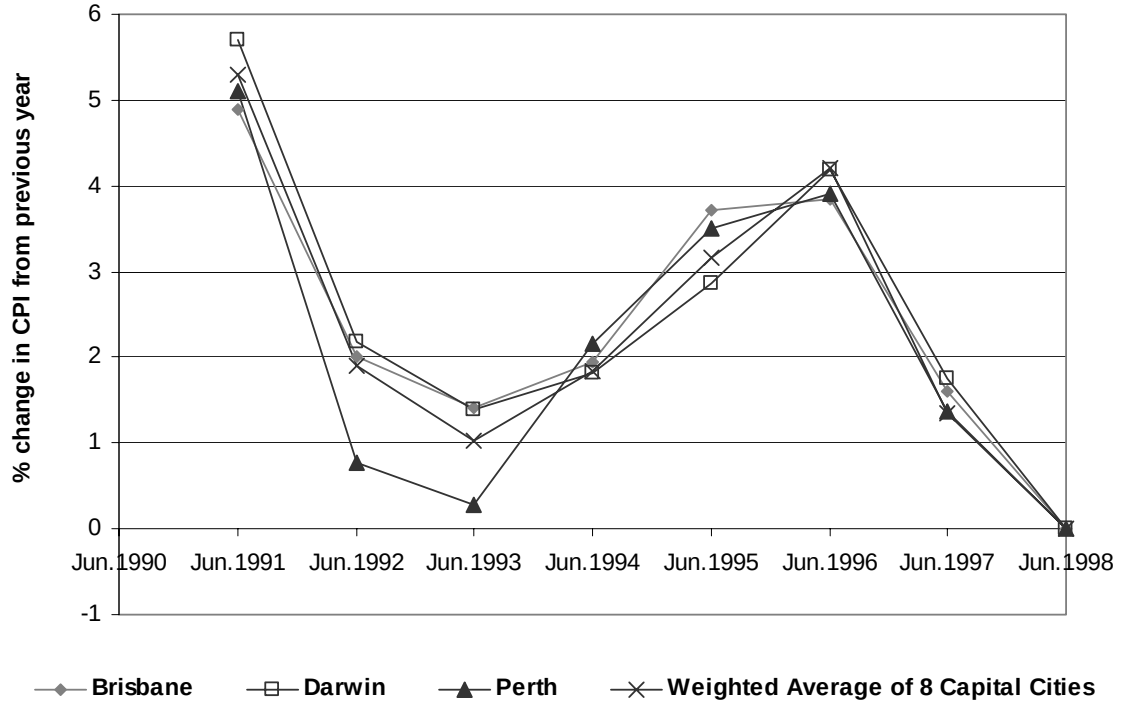
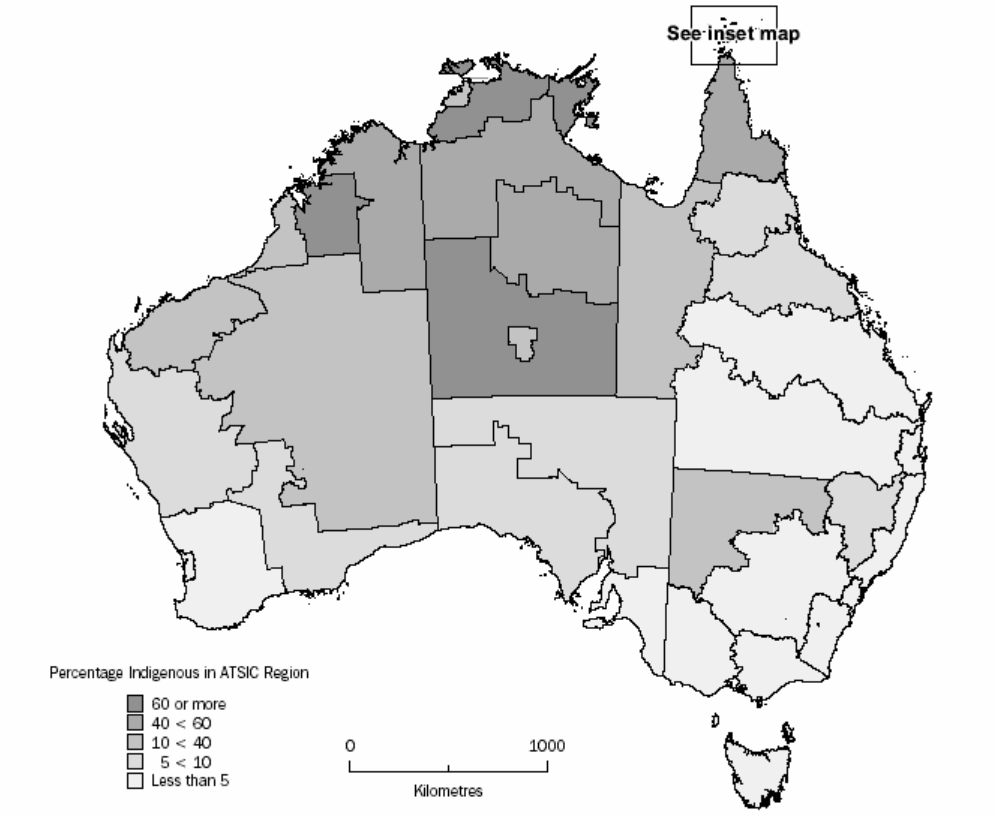


Figure 1
Consumer Price index changes for Darwin, Brisbane, Perth and the nation, June 1992 – June 1997
(Source: ABS, 2003)



Map 2
Proportion of regional population that are Indigenous, 1996
(Source: ABS Cat No. 4704.0)

When compared to the southern regions of Australia, more residents living in northern areas identify themselves as Indigenous. Map 2 shows that as a proportion of the regional population, Indigenous residents are most concentrated in the NT, and the northern areas of Western Australia and Queensland.

POPULATION RATES

Quarterly crude rates of alcohol-attributable deaths were calculated by dividing the number of cases by the estimated residential population (ERP) for that region. Population rates were generated per 10,000 adult persons. However, crude rates do not account for the age-structure within a population (which has been shown to affect levels of alcohol consumption and harm) and are therefore not suitable for inter-regional comparisons (Rothman, 1986).

To counter this, crude rates were used to estimate age-standardised rates (ASRs) for both the NT and control regions. Using the direct method for age standardisation (Rothman, 1986), the 2002 control region age structure was designated as the reference population. Rates were then weighted depending on the degree and direction of disparity between these distributions and finally multiplied by 10,000. In this way, differences in the age distribution between defined populations were removed, allowing direct comparisons between regions.

ARIMA TIME SERIES ANALYSIS

ARIMA was used to investigate the association between quarterly alcohol-attributable death rates due to risky/high risk drinking in the NT and the periods during which the LWA program and the Levy were operational. The dependent variable was the age standardised and de-seasonalised quarterly rate of alcohol-attributable deaths among NT residents between January 1985 and December 2002, known as '*NT alcohol deaths'*. There were three main independent variables of interest;

(i) '*LWA-on'* (dummy variable) defined the period during which only the LWA program operated continuously, i.e., from August 1992 to December 2002;

(ii) '*Levy/LWA-on'* (dummy variable) defined the period during which both the LWA Alcoholic Beverage Levy and the LWA programmes were in place, from April 1992 to August 1997;

(iii) '*Levy-off*' (dummy variable) distinguishes the period after the Levy from the period before. Notably, the period after the Levy and the period before the Levy are dissimilar in that the LWA program continued to operate after the Levy had ceased.

The main control variables included in the analysis as independent regressors were the age standardised and de-seasonalised quarterly trends in; non-alcohol attributable deaths in the NT known as '*NT non-alcohol deaths'* and; alcohol-attributable deaths in the control region abbreviated as '*Control region alcohol deaths'*.

Cask wine Levy and '0.05' minimum legal blood alcohol level

Two other possibly confounding events took place during the study period; (a) the introduction of the NT Cask wine Levy in June 1995, which, like the alcoholic beverage Levy, ceased in August 1997 and; (b) the implementation of a 0.05mg/ml legal blood alcohol level for driving in the NT in December 1994. This legislation remained in place throughout the remainder of the study period. Thus, in order to identify the period of operation of the Cask wine specific Levy, the '*Cask wine Levy'* dummy variable was denoted 1 from the 3rd quarter of 1995 to the 2nd quarter of 1997 and 0 for all earlier quarters. To identify the period of operation of the 0.05 drink-driving legislation, a dummy variable labelled '*05 legislation*' was denoted 1 from the 1st quarter 1995 to the end of the study period (4th quarter 2002).

Analysis

Using the technique for intervention analysis introduced by Box and Tiao (1975), basic ARIMA models were developed for the series occurring before intervention and the independent variables of interest were treated as step functions.

The primary intervention model examined the simple linear association between the intervention period and NT alcohol deaths. The following regressions successively introduced the control variables; NT non-alcohol deaths, control region alcohol deaths as well as the Cask wine Levy and 0.05 Legislation. Initial analyses were performed separately on both acute and chronic death trends and for the LWA and Levy intervention periods. The two final analyses test the associations between the two

intervention periods (LWA-on and Levy-on) and acute and chronic alcohol-attributable death rates in the NT when both are presented simultaneously in the same model.

THE EFFECT OF LWA AND THE LEVY ON INDIGENOUS VERSUS NON-INDIGENOUS ALCOHOL-ATTRIBUTABLE DEATH RATES

More than one in four people in the NT (29%) are estimated to be of Indigenous origin. In all other States/Territories, Indigenous Australians comprise less than 4% of the resident population (ABS, 2001). According to the 1994 *National Drug Strategy Household Survey Urban Aboriginal and Torres Strait Islander Peoples Supplement,* some 82% of all Indigenous current drinkers consume at risky or high risk levels compared to 28% of non-Indigenous drinkers (AIHW, 1996).

This evaluation was also able to compare Indigenous and non-Indigenous alcohol-attributable deaths rates in the NT before and during the LWA program and before, during and after the Levy/LWA program. However, data on Indigenous status is of limited quality and reliability varies markedly between jurisdictions. Queensland data on Indigenous status is particularly problematic and only of acceptable quality after 1998 (Chikritzhs *et al*., 2004). NT mortality data has maintained some of the most reliable data on Indigenous status but is only of acceptable quality from 1989 onwards. Comparisons could therefore be made between Indigenous and non-Indigenous rates of alcohol-attributable deaths within the NT, but it was not feasible to compare these to a control region. Nevertheless, it was possible to include internal control series of non-alcohol related deaths control for Indigenous and non-Indigenous deaths. The same methods described earlier for identifying NT non-alcohol related deaths were applied to Indigenous and non-Indigenous groups.

Small absolute numbers of Indigenous alcohol-attributable deaths also prohibit estimates of quarterly death rates. In order to ensure reliable rates of alcohol-attributable deaths for Indigenous persons, annual estimates have been presented. Thus, annual age standardised death rates for Indigenous and non-Indigenous populations in the NT have been calculated for 14 consecutive years, including 1989 through to 2002.

Since annual rates were used in preference to quarterly rates, the start date for the LWA program (August 1992) was assumed to be 1992. For the Levy, the start date (April 1992) was also denoted as 1992 while the final year of the Levy (August 1997) was denoted as 1997, that is, 1998 was assumed to be the first year when the Levy was not in operation. The use of time series techniques such as ARIMA is inappropriate for short time series and simple comparisons of means were applied.

RESULTS

DESCRIPTIVE RESULTS

Figures 2 and 3 show actual trends in quarterly age standardised alcohol-attributable death rates for acute and chronic conditions respectively in the NT, as well as NT non-alcohol attributable deaths and alcohol-attributable deaths in the control region. Figures 4 and 5 show smoothed trends[2] for the same series.

Table 2 shows mean death rates before, during and after the Alcoholic Beverage Levy was introduced to the NT for acute and chronic alcohol-attributable conditions.

The average rate of acute deaths in the NT during the Levy/LWA program was about 37% lower than before the interventions had been introduced. In fact, as evident from Figure 2, the majority of the decline in acute death rates occurred within the first 2 years of the Levy/LWA program. After the Levy was removed (but while the LWA program continued), acute death rates in the NT remained the same as they had been during the combined Levy/LWA program period. Acute death rates in the control region also declined between April 1992 and August 1997, but to a lesser extent (15.9%). However, unlike the trend seen in the NT, acute death rates in the control region continued to decline after 1997 (20.3%).

For chronic conditions, death rates in the NT declined once the Levy/LWA program was in place (10.0%) but in fact, a much more marked decline occurred after the Levy was removed (26.0%). Similarly, chronic death rates in the control region were lower during the Levy/LWA program period than before (18.2%). Like NT chronic deaths, death rates in the control region also continued to decline after the Levy was removed in late 1997 (15.6%) – although the fall was not as large as that seen in the Territory.

[2] Smoothed values were calculated using SPSS v11 time series smoothing procedure sometimes referred to as T4253H smoothing. Values are based on a compound data smoother starting with a running median of 4, which is centered by a running median of 2. It then resmoothes these values by applying a running median of 5, a running median of 3, and hanning (running weighted averages). Residuals are computed by subtracting the smoothed series from the original series. This whole process is then repeated on the computed residuals. Finally, the smoothed residuals are computed by subtracting the smoothed values obtained the first time through the process.

Table 2
NT and control region (de-seasonalised) mean quarterly age standardised death rates for *acute* and *chronic* conditions, before, during (April 1992 – August 1997) and after the Living With Alcohol Levy period

	Before Levy/LWA (before) (N=29)		Levy/LWA-on (during) (N=21)		Levy-off (after) (N=22)	
	ASR	**(95%CI)**	**ASR**	**(95%CI)**	**ASR**	**(95%CI)**
Acute						
NT	1.25	(1.12, 1.37)	0.79	(0.70, 0.88)	0.79	(0.71, 0.87)
Control region	0.50	(0.46, 0.55)	0.42	(0.38, 0.47)	0.34	(0.30, 0.37)
Chronic						
NT	0.90	(0.79, 0.10)	0.81	(0.68, 0.93)	0.60	(0.50, 0.69)
Control region	0.55	(0.50, 0.60)	0.45	(0.39, 0.50)	0.38	(0.34, 0.43)

Table 3 shows mean death rates before and during the full length of the LWA program (August 1992 – December 2002) for acute and chronic alcohol-attributable conditions. During the LWA program, NT death rates for *acute* alcohol-attributable conditions were 32.6% lower on average than they had been before the program was implemented. Acute alcohol-attributable death rates for the control region also fell during the LWA period, but to a lesser extent (23.6%).

For chronic conditions, there was a decline of about 23.5% in quarterly death rates in the NT during the LWA period however, the control region experienced the same level of decline (23.5%).

Table 3
NT and control region (de-seasonalised) mean quarterly age standardised death rates for *acute* and *chronic* alcohol-attributable conditions, before and during (August 1997–December 2002) the Living With Alcohol program

	Before LWA (before) (N=31)		LWA-on (during) (N=41)	
	ASR	**(95%CI)**	**ASR**	**(95%CI)**
Acute				
NT	1.20	(1.06, 1.33)	0.81	(0.75, 0.86)
Control region	0.50	(0.45, 0.54)	0.38	(0.35, 0.41)
Chronic				
NT	0.85	(0.76, 0.95)	0.65	(0.57, 0.73)
Control region	0.51	(0.47, 0.56)	0.39	(0.36, 0.43)

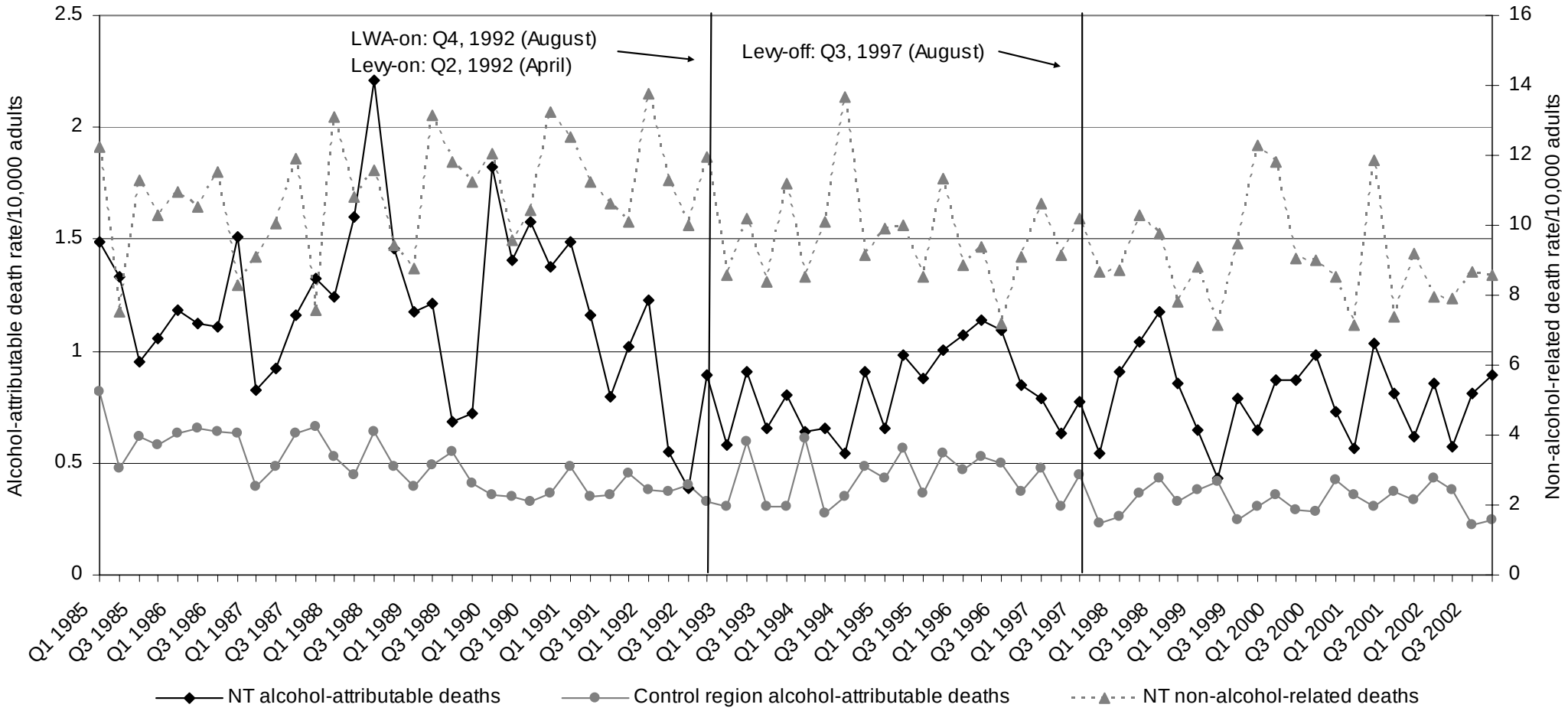


Figure 2
Trends in age standardised, de-seasonalised, quarterly *acute* death rates (per 10,000 adults) in the NT and Control region, 1985-2002

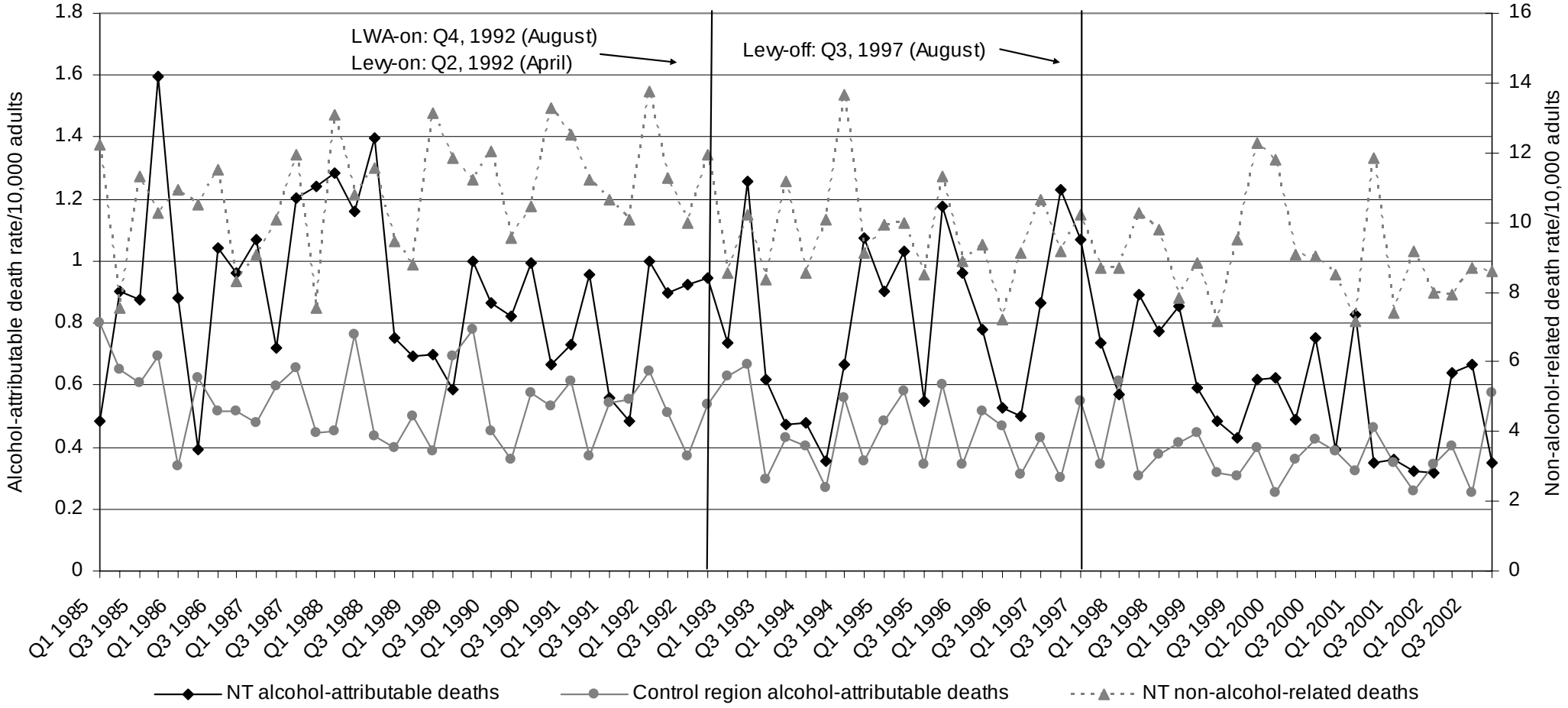


Figure 3
Trends in age standardised, de-seasonalised, quarterly *chronic* death rates (per 10,000 adults) in the NT and Control region, 1985-2002

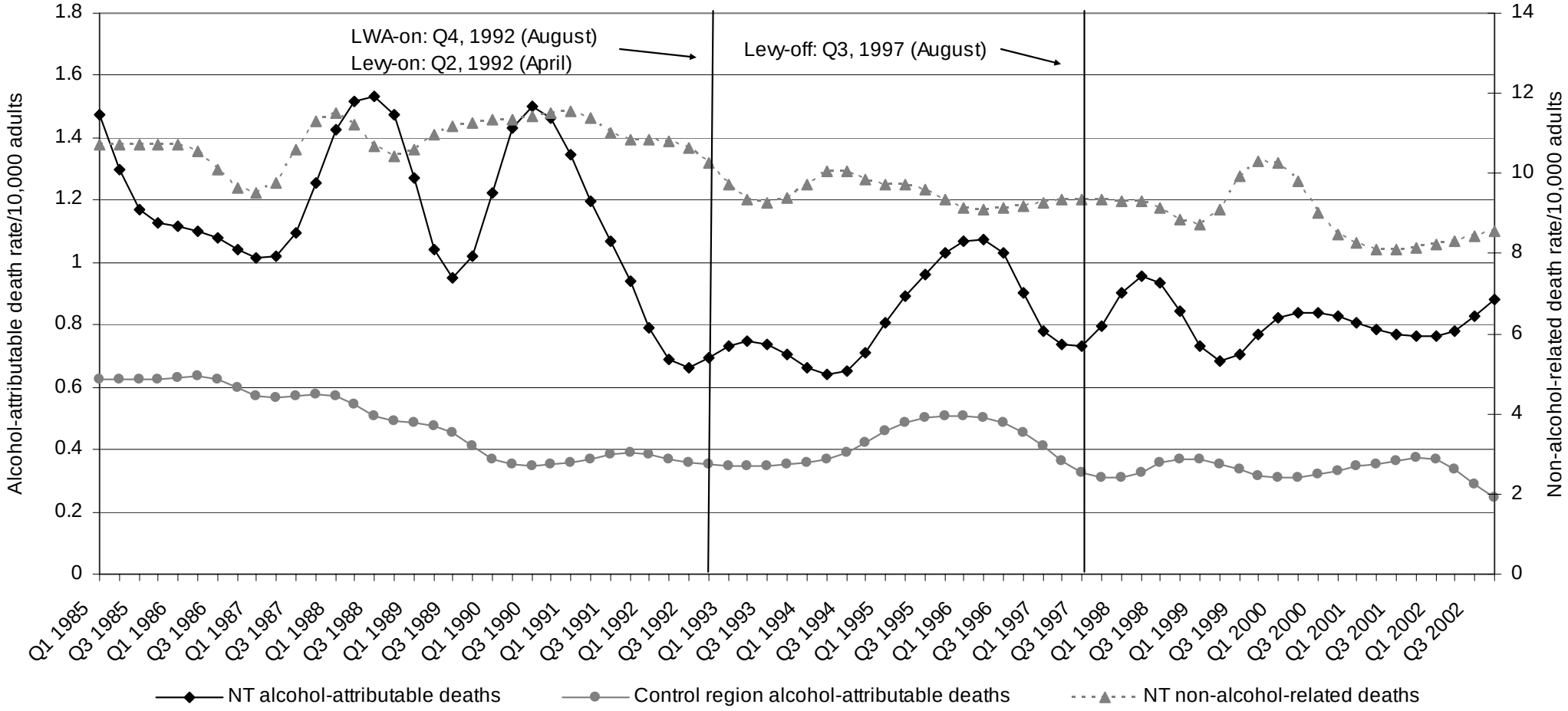


Figure 4
Smoothed trends in age standardised, de-seasonalised, quarterly *acute* death rates (per 10,000 adults) in the NT and Control region, 1985-2002

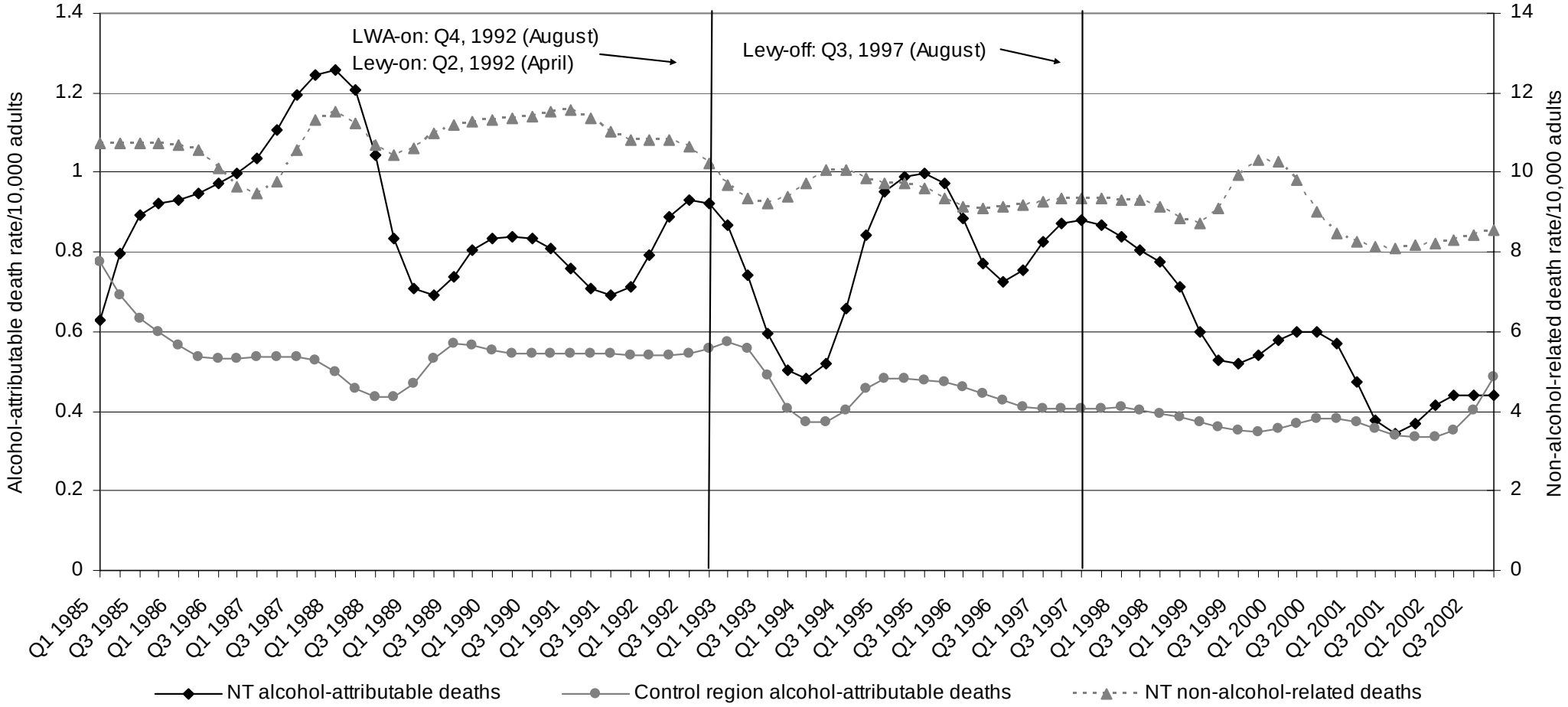


Figure 5
Smoothed trends in age standardised, de-seasonalised, quarterly *chronic* death rates (per 10,000 adults) in the NT and Control region, 1985-2002

ARIMA TIME SERIES ANALYSIS

The effect of the Alcoholic Beverage Levy and Living With Alcohol program on acute alcohol-attributable deaths in the NT

A Durbin-Watson test of serial autocorrelation for the model with dependent variable acute NT alcohol deaths, and independent variable Levy/LWA-on, indicated that the residuals exhibited significant positive autocorrelation (d = 1.209 < $d_{u,\ 0.05}$ = 1.703). Thus, ARIMA was identified as preferable to standard multiple linear regression. The pre-intervention series appeared adequately integrated and was not differenced, although the natural log was taken to stabilise variance. Analysis indicated 1st order serial autocorrelation and autocorrelation plots of errors showed ARIMA (1, 0, 0) to be a parsimonious and adequate model with no spikes evident to the 16th lag (Ljung-Box Q statistic (Q*) = 18.24, ns). Table 4 shows the model statistics for each of the variables in the order which they were entered and their effect on the association between Levy/LWA-on and NT alcohol deaths.

Table 4
The association between NT *acute* alcohol-attributable death rates and the combined Alcoholic Beverage Levy/LWA program period (Levy/LWA-on) including control variables

Variables entered	Q Statistic[a]	New independent variable entered (variable in bold)		Levy/LWA-on independent variable statistics			
		B	Sig.	Beta	S.E. Beta	t-ratio	Sig.
Levy/LWA-on *only*	16.45	-	-	-0.315	0.129	-2.440	0.017*
Levy-off *only*	18.28	0.216	0.097	-	-	-	-
Levy/LWA-on *and* Levy-off	22.15	-	-	-0.473	0.096	-4.490	0.000*
NT non-alcohol deaths *and* Levy/LWA-on, Levy-off	22.96	0.022	0.258	-0.453	0.102	-4.447	0.000*
Control region alcohol deaths *and* Levy/LWA-on, Levy-off	20.53	0.439	0.141	-0.436	0.099	-4.414	0.000*
NT non-alcohol deaths, control region deaths, Levy/LWA-on and Levy-off	21.24	-	-	-0.417	0.103	-4.030	0.000*
Cask wine levy *and* Levy/LWA-on, Levy-off NT non-alcohol deaths, control region deaths	25.32	0.283	0.040*	-0.515	0.105	-4.891	0.000*
05 Legislation *and* Levy/LWA-on, Levy-off NT non-alcohol deaths, control region deaths	28.19	0.264	0.047*	-0.537	0.111	-4.845	0.000*
05 Legislation, Cask wine levy, Levy/LWA-on, Levy-off NT non-alcohol deaths, control region deaths	26.84	-	-	-0.536	0.111	-4.814	0.000*

[a]Q* at 16th lag. *significant at 0.05 level

Taking into account the simultaneous trends in control region alcohol-attributable death rates and NT non-alcohol death rates, the combined Levy/LWA program period was found to be significantly and negatively associated with acute alcohol-attributable deaths in the NT. Although the Cask wine levy and 0.05 legislation both indicated significant independent associations with acute deaths in the NT, they did not show any moderating effect on the association between Levy/LWA-on and acute death rates; moreover, they both appeared to reduce the efficacy of the model.

The effect of the Alcoholic Beverage Levy and Living With Alcohol program on chronic alcohol-attributable deaths in the NT

Durbin-Watson statistic indicated significant positive autocorrelation for the chronic deaths series when regressed on Levy/LWA-on (d = 1.196 < $d_{u,\ 0.05}$ = 1.703). The pre-intervention series was adjusted by natural log and ARIMA (1, 0, 0) was found to be a parsimonious and adequate model with no spikes evident to the 16th lag (Q* = 16.49, ns). Table 5 shows the model statistics for each of the variables entered and their effect on the association between NT alcohol deaths and Levy/LWA-on.

Table 5
The association between NT *chronic* alcohol-attributable death rates and the combined Alcoholic Beverage Levy/LWA program period (Levy/LWA-on) including control variables

	Q Statistic[a]	New independent variable entered (variable in bold)		Levy/LWA-on independent variable statistics			
Variables entered		**B**	**Sig.**	**Beta**	**S.E. Beta**	**t-ratio**	**Sig.**
Levy/LWA-on *only*	16.48	-	-	0.073	0.149	0.491	0.624
Levy-off *only*	18.53	-0.367	0.003*	-	-	-	-
Levy/LWA-on *and* Levy-off	19.41	-	-	-0.110	0.131	-0.837	0.405
NT non-alcohol deaths *and* Levy-on, Levy-off	17.46	0.015	0.562	-0.093	0.135	-0.691	0.492
Control region alcohol deaths *and* Levy-on, Levy-off	19.28	0.128	0.326	-0.095	0.136	-0.699	0.487
NT non-alcohol deaths, control region deaths, Levy/LWA-on and Levy-off	17.55	-	-	-0.085	0.139	-0.610	0.544
Cask wine Levy *and* Levy-off, Levy-on, NT non-alcohol deaths, control region deaths	20.02	0.161	0.220	-0.140	0.137	-1.022	0.310
05 Legislation *and* Levy-off, Levy-on, NT non-alcohol deaths, control region deaths	18.10	0.101	0.602	-0.130	0.163	-0.798	0.427
05 Legislation, Cask wine Levy, Levy-off, Levy-on, NT non-alcohol deaths, control region deaths	20.05	-	-	-0.136	0.155	-0.880	0.382

[a]Q* at 16th lag. *significant at 0.05 level

As shown in Table 5, there was no evidence of a significant association between the combined Levy and LWA program period and chronic alcohol-attributable death rates in the NT. Interestingly, the last five and half years (22 quarters), during which time the Levy had ceased but the LWA program remained in operation (Levy-off), chronic alcohol-attributable death rates declined significantly in the NT.

The effect of the Living With Alcohol program on acute alcohol-attributable deaths in the NT

Durbin-Watson statistic indicated significant positive autocorrelation for the acute deaths series in the presence of Levy/LWA-on and control variables ($d = 1.059 < d_{u,\ 0.05} = 1.703$). Autocorrelation plots indicated ARIMA (1, 0, 0) to be a parsimonious and adequate model for the natural logged pre-intervention series, with no spikes evident to the 16^{th} lag ($Q^* = 24.4$). Table 6 shows the model statistics for each of the variables entered and their effect on the association between LWA-on and NT alcohol deaths.

Table 6
The association between NT *acute* alcohol-attributable death rates and the Living With Alcohol program period (LWA-on) including control variables

Variables entered	Q Statistic[a]	New independent variable entered (variable in bold)		LWA-on independent variable statistics			
		B	Sig.	Beta	S.E. Beta	t-ratio	Sig.
LWA-on *only*	18.51	-	-	-0.330	0.093	-3.535	0.000*
NT non-alcohol deaths *and* LWA-on	21.48	-0.033	0.102	-0.274	0.102	-2.700	0.009*
Control region alcohol deaths *and* LWA-on	18.79	0.602	0.056	-0.257	0.098	-2.620	0.011*
NT non-alcohol deaths, control region deaths and LWA-on	18.74	-	-	-0.215	0.104	-2.064	0.043*
Cask wine levy *and* NT non-alcohol deaths, control region deaths, LWA-on	18.72	-0.104	0.470	-0.241	0.106	-2.280	0.026*
05 Legislation *and* NT non-alcohol deaths, control region deaths and LWA-on	19.43	0.055	0.698	-0.260	0.144	-1.81	0.075
05 Legislation, Cask wine levy, NT non-alcohol deaths, control region deaths and LWA-on	19.31	-	-	-0.273	0.142	-1.917	0.059

[a]Q* at 16th lag. *significant at 0.05 level

Taking into account the simultaneous trends in control region alcohol-attributable death rates, non-alcohol related death rates in the NT, the LWA program was associated with a significant decline in acute alcohol-attributable deaths in the NT. The Cask wine levy and 0.05 legislation both produced non-significant coefficients and did not add to the efficacy of the basic model (i.e. including both control series), but both variables appeared to slightly mediate the t-ratio for LWA-on by a 10% increase and 13% decrease respectively. (According to Rothman, 1986, a 20% change in standardised beta is sufficient evidence of a mediating effect). Adjustment for the effects of both the Cask wine levy and the implementation of 0.05 legislation rendered the association between the full LWA program period and NT acute death rates non-significant.

The effect of the Living With Alcohol program on chronic alcohol-attributable deaths in the NT

For the de-seasonalised series of quarterly chronic death rates in the NT, Durbin-Watson statistic indicated significant positive autocorrelation when regressed on LWA-on (d = 1.384 < $d_{u,\ 0.05}$ = 1.703). Differencing was unnecessary as the pre-intervention series was apparently largely integrated, although natural log transformation was applied to reduce variance instability. For the pre-intervention series, ARIMA (1, 0, 0) was found to be a parsimonious and adequate model with no spikes evident among autocorrelations to the 16th lag (Q* =16.45, ns). Table 7 shows the model statistics for each of the independent variables entered and their effect on the association between NT alcohol deaths and LWA-on.

Table 7
The association between NT *chronic* alcohol-attributable death rates and the Living With Alcohol Program period (LWA-on) including control variables

Variables entered	Q Statistic[a]	New independent variable entered (variable in bold)		LWA-on independent variable statistics			
		B	Sig.	Beta	S.E. Beta	t-ratio	Sig.
LWA-on *only*	14.98	-	-	-0.276	0.120	-2.286	0.025*
NT non-alcohol deaths *and* LWA-on	13.17	-0.276	0.025	-0.230	0.125	-1.995	0.050*
Control region alcohol deaths *and* LWA-on	15.28	0.176	0.320	-0.255	0.125	-2.040	0.045*
NT non-alcohol deaths, control region deaths and LWA-on	13.38	-	-	-0.239	0.129	-1.856	0.068
Cask wine levy *and* NT non-alcohol deaths, control region deaths, LWA-on	13.31	0.123	0.144	-0.295	0.138	-2.133	0.038*
05 Legislation *and* NT non-alcohol deaths, control region deaths and LWA-on	13.68	0.090	0.0186	-0.177	0.186	-0.955	0.343
05 Legislation, Cask wine levy, NT non-alcohol deaths, control region deaths and LWA-on	14.65	-	-	-0.190	0.170	-1.117	0.268

[a]Q* at 16th lag. *significant at 0.05 level

As shown in Table 7, after adjusting for both trends in NT non-alcohol related deaths and control region alcohol-attributable deaths there was no significant association

between LWA and chronic alcohol-attributable deaths in the NT. Interestingly, subsequent adjustment for the effect of the Cask wine levy increased the strength of the relation between the LWA program and NT alcohol-attributable deaths. Conversely, the inclusion of the implementation of the 0.05 legislation had the opposite effect. Thus the two events had strong but opposing mediating effects on the association between NT alcohol deaths and the LWA program. A non-significant association was maintained after adjusting for both events simultaneously. Consequently, taking into account the implementation of the Cask wine levy, the implementation of 0.05 legislation, and the two control series, the LWA program failed to show a significant impact on chronic alcohol-attributable deaths in the NT.

The relative effects of the Living With Alcohol program and the Alcoholic Beverage Levy on acute and chronic alcohol-attributable deaths in the NT

The results presented in Table 8 show that when the impact of the combined but limited Levy/LWA program period was tested against the impact of the full length of the LWA program – and in the presence of the two control series, the Cask wine levy and 0.05 legislation – only the limited period during which both the Levy the LWA program (Levy/LWA-on) were in operation indicated any significant negative association with acute NT alcohol-attributable death rates. Interestingly, adjustment for the introduction of 0.05 legislation had a very large mediating effect on the size of the association between the LWA program and acute deaths (95% reduction).

Table 8
The effect of the combined Alcoholic Beverage Levy and the LWA program period (Levy/LWA-on) on *acute* alcohol-attributable death rates in the NT relative to the effect of the full length LWA program (LWA-on), including control variables

	LWA-on independent variable		Levy/LWA -on independent variable	
Variables entered	**t-ratio**	**Sig.**	**t-ratio**	**Sig.**
LWA-on *only*	-3.535	0.000*	-	-
Levy/LWA-on *only*	-	-	-2.439	0.017*
LWA-on *and* **Levy/LWA-on**	-2.460	0.016*	-1.560	0.123
LWA-on, Levy/LWA-on *and* NT non-alcohol deaths	-1.730	0.088	-1.691	0.095
LWA-on, Levy/LWA-on *and* control region alcohol deaths	-1.582	0.118	-1.853	0.068
LWA-on, Levy-on, NT non-alcohol deaths, control region alcohol deaths	-1.116	0.268	-1.932	0.057
LWA-on, Levy-on, NT non-alcohol deaths, control region alcohol deaths *and* Cask wine levy	-1.538	0.129	-2.773	0.007*
LWA-on, Levy-on, NT non-alcohol deaths, control region alcohol deaths *and* 05 legislation	0.054	0.956	-2.109	0.039*
LWA-on, Levy-on, NT non-alcohol deaths, control region alcohol deaths, Cask wine levy, 05 legislation	0.899	0.372	-3.480	0.000*

[a]Q* at 16th lag. *significant at 0.05 level

When the relative impacts of the combined Levy/LWA program and the LWA program on NT chronic alcohol-attributable death rates were tested, only the longer LWA program period indicated a significant effect. Relative to the effect of the limited Levy/LWA period, the relation between the LWA program and chronic deaths was a negative one – thus, overall, chronic death rates were lower during this longer period (Table 9).

Notably, Table 9 also showed that there was a large decline in chronic deaths in the NT after August 1997 (Levy-off) that is, once the Levy had ceased, chronic death rates actually appeared to improve. As the LWA program extends to 2002 it is therefore inclusive of the apparent fall in chronic death rates. Thus, when the relative impacts of the LWA program period and the Levy period were considered simultaneously, the longer running LWA program period indicated an association with a marked decline in chronic death rates in the NT. This association held true when both the Cask wine levy and 0.05 legislation were controlled for.

Table 9
The effect of the combined Alcoholic Beverage Levy and the LWA program period (Levy/LWA-on) on *chronic* alcohol-attributable death rates in the NT relative to the effect of the full length LWA program (LWA-on), including control variables

	LWA-on independent variable		Levy/LWA-on independent variable	
Variables entered	**t-ratio**	**Sig.**	**t-ratio**	**Sig.**
LWA-on *only*	-2.876	0.005*	-	-
Levy/LWA-on *only*	-	-	0.073	0.624
LWA-on *and* **Levy/LWA-on**	-3.120	0.003*	1.851	0.068
LWA-on, Levy/LWA-on *and* NT non-alcohol deaths	-2.734	0.008*	1.778	0.079
LWA-on, Levy/LWA-on *and* control region alcohol deaths	-2.770	0.007*	1.848	0.069
LWA-on, Levy-on, NT non-alcohol deaths, control region alcohol deaths	-2.540	0.013*	1.783	0.079
LWA-on, Levy-on, NT non-alcohol deaths, control region alcohol deaths *and* Cask wine levy	-3.097	0.003*	2.11	0.038*
LWA-on, Levy-on, NT non-alcohol deaths, control region alcohol deaths *and* 05 legislation	-1.956	0.054	1.82	0.073
LWA-on, Levy-on, NT non-alcohol deaths, control region alcohol deaths, Cask wine levy, 05 legislation	-2.027	0.047*	1.746	0.085

THE EFFECT OF THE ALCOHOLIC BEVERAGE LEVY AND THE LIVING WITH ALCOHOL PROGRAM ON INDIGENOUS AND NON-INDIGENOUS DEATH RATES IN THE NT

Figure 6 shows trends in Indigenous and non-Indigenous annual acute alcohol-attributable and non-alcohol related death rates between 1989 and 2002. Trends in chronic alcohol-attributable deaths are shown in Figure 7.

Most notably, throughout the study period, regardless of the cause of death, Indigenous NT residents had higher death rates than their non-Indigenous counterparts. Indigenous and non-Indigenous death rates for acute alcohol-attributable conditions appeared to decline swiftly during the early 1990's when the LWA program and Levy were first introduced. Conversely, up to about 1998, chronic death rates did not appear to improve for either group – in fact there was a marked increase in chronic alcohol-attributable deaths for Indigenous NT residents in the mid 1990's. Over the last few years of the study period however, chronic alcohol-attributable death rates did appear to

improve for both Indigenous and non-Indigenous NT residents. Most surprisingly however, for Indigenous residents only, there was a large decline in non-alcohol related deaths from about 1992 to 1997 – coinciding with the Levy/LWA period.

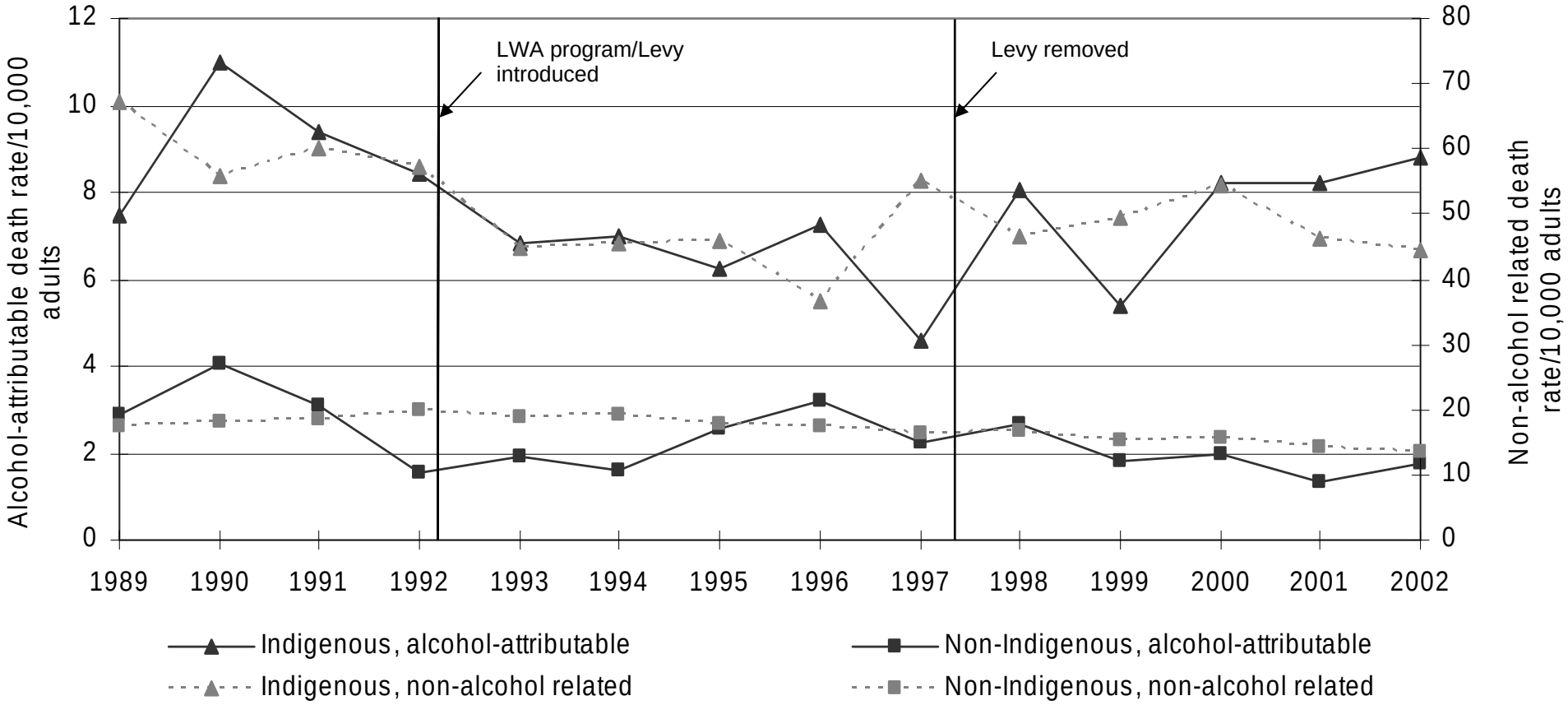


Figure 6
Indigenous and non-Indigenous trends in age standardised, annual *acute* alcohol-attributable death rates (per 10,000 adults) in the NT, 1985-2002

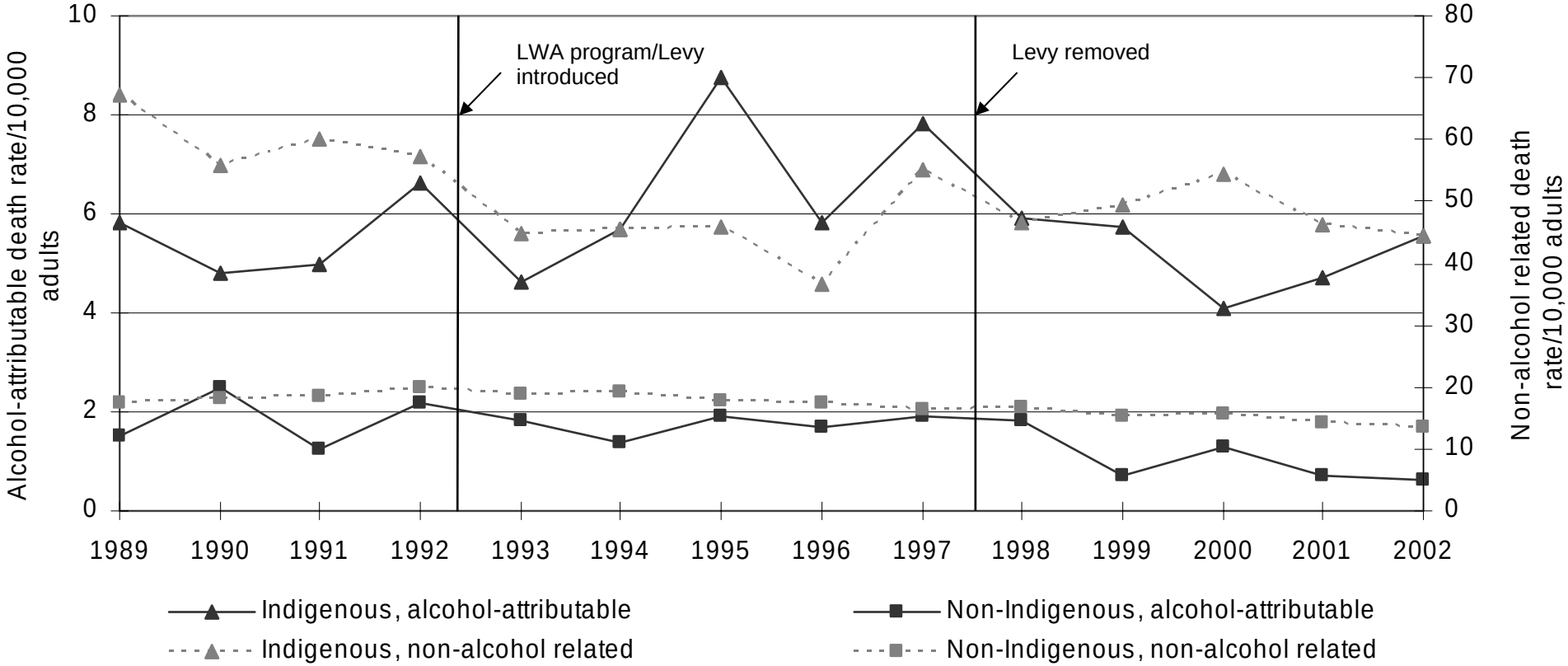


Figure 7
Indigenous and non-Indigenous trends in age standardised, annual *chronic* alcohol-attributable death rates (per 10,000 adults) in the NT, 1985-2002

As shown in Table 10, there was a significant decline in acute alcohol-attributable death rates for both Indigenous and non-Indigenous residents during the combined Levy/LWA period but, no evidence of any significant change in acute deaths after the Levy was removed. Notably, non-alcohol related deaths among Indigenous persons declined significantly during the LWA program period (21.6%); although this was not the case for non-Indigenous non-alcohol related deaths.

Among Indigenous and non-Indigenous NT residents who died from chronic alcohol-attributable conditions, there was no evidence of any significant change during the Levy/LWA period. After the Levy was removed, chronic death rates for non-Indigenous residents showed a significant decline (43.4%), but there was no significant change evident for Indigenous residents (Table 11).

Table 10
Indigenous and non-Indigenous NT mean annual age standardised death rates for *acute* conditions, before, during and after the combined Living Alcoholic Beverage Levy and Living With Alcohol program period (April 1992 – August 1997)

	Before Levy/LWA (N=3)		During Levy/LWA (N=6)		% Change from before to during (t-value)	After Levy (N=5)	% Change from during to after (t-value)
	ASR	(95%CI)	ASR	(95%CI)			
Indigenous							
alcohol	9.27	(4.89, 13.6)	6.74	(5.41, 8.07)	-27.3 (2.50)*	7.73 (6.06, 9.40)	14.7 (-1.25)
non-alcohol	61.1	(46.8, 75.4)	47.5	(39.6, 55.4)	-22.3 (2.71)*	48.3 (43.5, 53.1)	1.68 (-0.21)
Non-Indigenous							
alcohol	3.34	(1.72, 4.95)	2.18	(1.52, 2.84)	-34.7 (2.58)*	1.94 (1.34, 2.53)	-11.0 (0.72)
non-alcohol	18.1	(16.9, 19.3)	18.2	(16.8, 19.7)	0.55 (-0.21)	15.1 (13.5, 16.6)	-17.0 (3.96)**

Statistical significance (t-test) * p<.05 ** p<.01 *** p<.001

Table 11
Indigenous and non-Indigenous NT mean annual age standardised death rates for *chronic* conditions, before, during and after the combined Living Alcoholic Beverage Levy and Living With Alcohol program period (April 1992 – August 1997)

	Before Levy/LWA (N=3)		During Levy/LWA (N=6)		% Change from before to during (t-value)	After Levy (N=5)	% Change from during to after (t-value)
	ASR	(95%CI)	ASR	(95%CI)			
Indigenous							
alcohol	5.18	(3.83, 6.55)	6.55	(4.97, 8.14)	26.4 (-1.48)	5.21 (4.24, 6.17)	-20.5 (1.80)
non-alcohol	61.1	(46.8, 75.4)	47.5	(39.6, 55.4)	-22.3 (2.71)*	48.3 (43.5, 53.1)	1.68 (-0.21)
Non-Indigenous							
alcohol	1.74	(0.06, 3.42)	1.82	(1.53, 2.10)	4.60 (-0.25)	1.03 (0.40, 1.66)	-43.4 (3.31)**
non-alcohol	18.1	(16.9, 19.3)	18.2	(16.8, 19.7)	0.55 (-0.21)	15.1 (13.5, 16.6)	-17.0 (3.96)**

Statistical significance (t-test) * p<.05 ** p<.01 *** p<.001

As shown in Table 12, during the operation of the LWA program which continued for several years after the Levy had ceased, there was in fact a significant decline in the mean annual death rate from acute alcohol-attributable conditions for both Indigenous (22.4%) and non-Indigenous (38.0%) residents of the NT. Thus, regardless of Indigenous status, acute alcohol-attributable death rates declined for all residents during the LWA program period. In contrast, there was no evidence of any significant change in chronic alcohol-attributable death rates for Indigenous or non-Indigenous residents (Table 13). (It should be noted that these comparisons do not include adjustment for internal and external control series or other possible confounders – which have been shown to mediate the relationship between LWA-on and NT death rates).

Table 12
Indigenous and non-Indigenous NT mean annual age standardised death rates (per 10,000 adults) for *acute* conditions, before and during the Living With Alcohol program (August 1992 – December 2002)

	Before LWA program (N=3)		**During LWA program (N=11)**		**% Change (t-value)**
	ASR	**(95%CI)**	**ASR**	**(95%CI)**	
Indigenous					
alcohol	9.27	(4.89, 13.6)	7.19	(6.29, 8.09)	-22.4 (2.25)*
non-alcohol	61.1	(46.8, 75.4)	47.9	(43.9, 51.8)	-21.6 (3.46)**
Non-Indigenous					
alcohol	3.34	(1.72, 4.95)	2.07	(1.70, 2.44)	-38.0 (3.41)**
non-alcohol	18.1	(16.9, 19.3)	16.8	(15.4, 18.2)	-7.00 (1.01)

Statistical significance (t-test) * p<.05 ** p<.01 *** p<.001

Table 13
Indigenous and non-Indigenous NT mean annual age standardised death rates (per 10,000 adults) for *chronic* conditions, before and during the Living With Alcohol program (August 1992 – December 2002)

	Before LWA program (N=3)		During LWA program (N=11)		% Change (t-value)
	ASR	**(95%CI)**	**ASR**	**(95%CI)**	
Indigenous					
alcohol	5.18	(3.83, 6.55)	5.94	(5.02, 6.86)	14.7 (-0.92)
non-alcohol	61.1	(46.8, 75.4)	47.9	(43.9, 51.8)	-21.6 (3.46)**
Non-Indigenous					
alcohol	1.74	(0.06, 3.42)	1.45	(1.08, 1.83)	-16.7 (0.75)
non-alcohol	18.1	(16.9, 19.3)	16.8	(15.4, 18.2)	-7.00 (1.01)

Statistical significance (t-test) * p<.05 ** p<.01 *** p<.001

SUMMARY AND DISCUSSION

This evaluation investigated how rates of alcohol-attributable death in the NT were affected by the implementation of the comprehensive LWA program introduced in August 1992 and the Alcoholic Beverage Levy (initially used to fund the LWA program) introduced in April 1992. Although the life of the Levy was limited (ending in August 1997) the LWA program continued on, this also allowed the impact of the LWA program to be examined in the absence of the Levy. Time series trends in both acute and chronic alcohol-attributable death rates, as well as Indigenous versus non-Indigenous death rates were examined. The main analyses compared periods during which the programmes were operational to non-operational periods. These comparisons were made while controlling for simultaneous trends in other deaths specific to the NT and simultaneous trends in alcohol-attributable deaths from a comparable control region external to the territory. The analyses also took into account the possible impact of other contributing factors that occurred during the study period but which were not directly related to the LWA program, including the implementation of the Cask wine levy and the introduction of legislation that reduced the maximum legal alcohol level for driving from 0.08 mg/ml to 0.05mg/ml.

Taking into account the effects produced by the implementation of the Cask wine levy, the implementation of 0.05 legislation, and the simultaneous trends in the two control series, the main results of this evaluation can be summarised as follows:

- between April 1992 and August 1997, the combined effect of the LWA Alcoholic Beverage Levy and the LWA program had a significant impact on *acute* alcohol-attributable deaths in the NT;
- between August 1992 and December 2002, there was no evidence of an effect of the LWA program on *acute* alcohol-attributable deaths in the NT;
- in the absence of the Alcoholic Beverage Levy, the LWA program failed to have a significant impact on *acute* alcohol-attributable deaths;

- between April 1992 and August 1997, there was no evidence of a combined effect of the Alcoholic Beverage Levy and the LWA program on *chronic* alcohol-attributable deaths in the NT;

- between August 1992 and December 2002, there was no evidence of an overall effect of the LWA program on *chronic* alcohol-attributable death rates in the NT;

- between August 1997 and December 2002, when the Alcoholic Beverage Levy had ceased but the LWA program continued, *chronic* alcohol-attributable deaths in the NT declined significantly;

In relation to the impacts of the LWA program and the Alcoholic Beverage Levy on Indigenous and non-Indigenous alcohol-attributable deaths in the NT:

- between 1992 and 1997, when both the LWA program and the Alcoholic Beverage Levy were in operation, both Indigenous and non-Indigenous *acute* alcohol-attributable deaths declined significantly;

- between 1992 and 1997, when both the LWA program and the Alcoholic Beverage Levy were in operation, there was no evidence of a decline in *chronic* alcohol-attributable deaths for either Indigenous or non-Indigenous NT residents;

- between 1997 and 2002, when the Alcoholic Beverage Levy had ceased but the LWA program continued, *chronic* alcohol-attributable deaths in the NT declined significantly for non-Indigenous but not for Indigenous residents;

- between 1992 and 1997, when both the LWA program and the Alcoholic Beverage Levy were in operation, deaths identified as unrelated to alcohol consumption declined significantly for Indigenous but not for non-Indigenous residents.

It should be noted that due to data limitations, it was not possible to apply the same rigorous control methods used in the main analyses when examining the impact of the intervention on Indigenous and non-Indigenous deaths.

The LWA program was originally funded by the implementation of an Alcoholic Beverage Levy on all drinks with greater than 3% alcohol content. This resulted in a small increase in the retail cost of all but low strength alcoholic beverages (e.g. light beer). Apart from generating funding for the programmes and services offered by the LWA program, itself, the Levy had a strong impact on the numbers of deaths caused by alcohol-attributable injury in the NT. Indeed, in the absence of the Levy, the LWA program failed to show any evidence that it reduced the numbers of acute deaths caused by risky and high risk drinking. However, it is not possible to firmly conclude from this study whether the reduction observed in acute alcohol-attributable deaths in the NT was a result of the Levy alone or some combined, interactive impact of the Levy and the programmes and services offered by the comprehensive LWA program.

Moreover, these findings are complicated by the fact that declines in acute alcohol-attributable deaths in the NT occurred about one year prior to the official implementation of the LWA program and Levy. Declines were also evident however for the control region in the early 1990's. Economic indicators reveal that there was in fact an economic downturn across all Australian states and territories at this time. Thus, the declines seen in the NT prior to the implementation of the Levy/LWA program were likely due to macro economic influences. It may also be the case that had the Levy/LWA program been introduced in a more positive economic climate, the observed changes may not have been as substantial. Nevertheless, the estimated size of the impact takes into account corresponding trends in alcohol-attributable deaths in the control region and therefore is appropriately adjusted for macro economic factors.

It was also apparent that toward the end of the study period, chronic alcohol-attributable deaths declined significantly. Indeed the decline occurred after the Levy had been removed. This belated reduction in chronic deaths may be due to the fact that chronic alcohol-related disease takes many years to develop, likewise, it may take several years before the impact of the programmes and services aimed at assisting individuals with chronic alcohol related disease would become evident among aggregate counts of deaths. It is also possible that the decline in chronic deaths represents a late effect of reduced consumption bought about by the Levy. The earlier evaluation by Stockwell *et al*. (2001) estimated that per capita alcohol consumption in the NT declined by 22% after the introduction of the Levy/LWA program. In any case,

despite the late onset of the decline, given that there was no corresponding decline in non-alcohol related NT deaths during this time and that there was no corresponding decline in alcohol-attributable deaths in the control region; the decline in chronic deaths can be attributed to the LWA program and/or Levy with reasonable confidence.

Further analyses also examined the impact of the Levy/LWA program and longer LWA program period separately for Indigenous and non-Indigenous NT residents. The results indicated that, like their non-Indigenous counterparts, Indigenous residents also experienced declines in acute alcohol-attributable deaths during the Levy/LWA program period. However, in relation to chronic alcohol-attributable disease, Indigenous residents did not appear to experience the same degree of benefit as their non-Indigenous counterparts during the last few years of the study period.

Interestingly however, there was a marked and significant decline in non-alcohol related deaths occurring among Indigenous people during the period when both the Levy and LWA program were in place. At the same time, non-alcohol related deaths among non-Indigenous residents did not decline.

One possible explanation for this unexpected finding may be due to declines in deaths from respiratory diseases (e.g. influenza, viral pneumonia, bronchitis, emphysema, asthma, unspecified chronic airway obstruction) among Indigenous communities in the NT over the last 20 years (personal communication, NT Health Services). Respiratory disease was one of the main causes of death among the Indigenous NT population between 1997 and 1995 (Zhao *et al*., 2004) and morbidity due to respiratory disease is over five times higher for Indigenous compared to non-Indigenous Australians (Cunningham *et al*., 1997). Notably, although alcohol may be a contributing lifestyle factor in the development of many respiratory diseases, most do not attract an alcohol aetiologic fraction and are therefore not included among estimates of alcohol-attributable deaths. In-roads made by NT health services into the incidence of infectious diseases that may result in a variety of respiratory conditions, particularly among those with impaired immunity due to chronic alcohol and tobacco misuse (McKenzie *et al*., 2003), may lie behind these changes (personal communication, NT Health Services).

Another possibility is that since the prevalence of both risky/high risk drinking and respiratory disease among Indigenous populations is far greater than that for non-Indigenous populations, a reduction in alcohol consumption, an improvement in health services for individuals with alcohol problems and general improvement in health may be more likely to reduce Indigenous deaths due to respiratory disease than non-Indigenous deaths. Thus, any change in the numbers of deaths from unspecified chronic airway obstruction would be evident among non-alcohol related trends. In any case, the underlying reasons for this finding need to be further investigated and will be the subject of an ensuing investigation.

CONCLUSION

The results of this study present a strong argument for alcohol taxes combined with comprehensive programmes and services designed to reduce the harms from alcohol. As evidenced here, an increase in the real price of alcohol brought about by such economic strategies, even when seemingly minor, bear significant health and economic benefits. Without the support of a price increase, programmes and services for reducing alcohol related harms may have limited benefits for reducing harms that tend to arise from episodes of drinking to intoxication, such as road injury and violent assault. Nevertheless, alcohol specific programmes and services such as those provided by the LWA program may also have positive, yet longer term impacts on chronic alcohol-attributable disease. This evaluation also supports the conclusion that the combined impact of programmes and services with an increase in the real price of alcohol is effective at reducing acute harms among both Indigenous and non-Indigenous communities.

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APPENDIX A

Table 14
Acute and chronic alcohol-attributable conditions, and excluded tobacco-related conditions (English et al., 1995)

Alcohol-attributable conditions		
Acute	**Chronic**	**Tobacco-related conditions**
Acute pancreatitis	Alcoholic cardiomyopathy[1]	Lung cancer
Alcohol abuse[1]	Alcoholic dependence[1]	Peptic ulcer
Alcoholic beverage poisoning[1]	Alcoholic liver cirrhosis[1]	Chronic bronchitis
Alcoholic gastritis[1]	Alcoholic poly neuropathy[1]	Peripheral vascular disease
Alcoholic psychosis[1]	Chronic pancreatitis	Renal pelvic cancer
Aspiration[1]	Epilepsy	Bladder cancer
Assault	Female breast cancer	
Child abuse	Heart failure	
Drowning	Hypertension	
Ethanol toxicity[1]	Ischaemic heart disease	
Fall injuries	Ischaemic stroke	
Fire injuries	Laryngeal cancer	
Gastro-oesophageal haemorrhage	Liver cancer	
Low birth weight	Oesophageal cancer	
Haemorrhagic stroke	Oesophageal varices	
Methanol toxicity[1]	Oropharyngeal cancer	
Occupational and machine injuries	Psoriasis	
Other ethanol and ethanol poisoning[1]	Unspecified liver cirrhosis	
Road injuries – pedestrian		
Road injuries – non-pedestrian		
Spontaneous abortion		
Supraventricular cardiac dysrhythmias		
Suicide		

[1]conditions wholly attributable to alcohol consumption

Table 15
Most common underlying causes of death for adults in the NT classified as non-alcohol related, Indigenous and non-Indigenous, 1985-2002

Indigenous	Non-Indigenous
Diabetes	Cancer of bronchus/lung
Chronic obstructive pulmonary disease	Chronic non-specific lung disease
Cancer of bronchus/lung	Colon cancer
Asthma	Prostate cancer
Pneumonia	Bronchitis
Cancer no specific site	Diabetes
Chronic renal failure	Cancer no specific site
Cervical cancer	Senile dementia
Undefined cause	Chronic obstructive pulmonary disease
	Stomach cancer