

HEALTH EFFECTS OF HEAVY KAVA USE IN INDIGENOUS AUSTRALIANS

Submitted by

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STATEMENT OF AUTHORSHIP

Except where reference is made in the text of the thesis, this thesis contains no material published elsewhere or extracted in whole or in part from a thesis by which I have qualified for or been awarded another degree or diploma.

The papers presented in this thesis consist of work of both joint authorship and sole authorship. In all instances, I am the primary author and have made the most substantial contribution to the work presented. Other authors that have also contributed to the work presented, and their contributions, are detailed here:

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This thesis includes material compiled during two periods of candidature for the PhD at the Northern Territory University. The first was from 1988-1991 and the second from 1999-2003. During the majority of these years, and the intervening years between the periods of candidature, the author lived and worked in the Arnhem Land region in Aboriginal communities where kava was used. The thesis includes observations, information and understandings gleaned during both periods of candidature spanning around 15 years of the short (21 years) history of kava use in Arnhem Land.

Thanks to the community Councils and the Aboriginal community residents in Arnhem Land where the author lived and worked since 1988.

Finally, thanks to A/Prof Ross Bailie who supervised the thesis.

THESIS ABSTRACT

Background

The psychoactive constituents of kava (*Piper methysticum* Forst. f.) have been used by humans as mood-altering agents for centuries. Kava's long history of use in Pacific island societies precedes its recent inclusion in western pharmacopoeia from the latter part of the nineteenth century. During the 1990s, the use of kava became more widespread around the world, especially as enthusiasm for herbal remedies in alternative therapies expanded rapidly in western countries.

Aboriginal people living in remote parts of Arnhem Land in the Northern Territory are now involved in the unfolding world history of kava and the international debate that surrounds it. From 1982, with little or no prior knowledge of kava use, some Arnhem Land Aboriginal people began to use dried powdered kava mixed with water. The population of kava using communities in Arnhem Land is small (just 7700 people) with the number of Aboriginal people who ever used it probably numbering no more than around 3100 by the end of the 1990s. Despite these very small numbers, information about kava drinking derived from research in these isolated Arnhem Land groups continues to have a considerable impact on the international debate about the health effects of its use.

Kava's health effects on individuals and communities in Arnhem Land became controversial soon after its introduction. However, there was a dearth of reliable epidemiological data until 1987 when a pilot survey was conducted in one community in eastern Arnhem Land. Kava users in that study were found to have a scaly skin, to be underweight and to have lower body fat. In this first community-based study of kava's effects, users showed biochemical changes in their blood suggesting increased risks of serious infectious disease and liver and kidney dysfunction. Some neurological functions appeared to be different in kava users. The pilot study influenced policy approaches to restrict kava supply in Arnhem Land in the early 1990s.

Kava use by Aboriginal people continued to be a cause for concern during the early 1990s, however, when clinical observations suggested that kava users were over-represented in admissions to hospital for pneumonia. There were also ongoing concerns for the nutritional status of kava users and their families, that some heavy users suffered unusual kinds of ‘fits’ or ‘seizures’ and that the incidence of sudden cardiac events was higher in young Aboriginal sportsmen who used kava.

These concerns paralleled rising concerns about social and economic effects of kava use. A rise in kava use appeared to coincide with emerging community dysfunction from 1982. Community and family life were reported to be disrupted by ‘excessive’ kava use apparently without social controls, and the economic hardships brought about by a vigorous informal, and later illegal, trade in the substance. The debate and commentary that flowed from these concerns influenced policy and helped to engender both legislative attempts by the NT Government (with bi-partisan support) to control kava supply and consumption; the first attempt in 1990, and the second in 1998.

Aim

The main aim of this thesis is to provide information to more thoroughly describe kava’s health effects and to assist policy makers to gauge the health, social and economic effects of regulatory efforts. No epidemiological study has investigated kava’s health effects in Arnhem Land since 1987. In response to continued kava use, regulators have implemented controls on kava supply and distribution in the Northern Territory. However, few data are available to provide precise regulatory guidelines.

Methods

Current patterns of kava consumption are described using data from sample surveys of communities in eastern Arnhem Land (the Miwatj Region). Using participant observation data gathered during 30 months residence in one western Arnhem Land community during 1989-1991, patterns of kava consumption in different social settings are described.

A cross-sectional study was conducted in one community in eastern Arnhem Land in 2000, in order to compare the results of the 1987 pilot study with kava's possible effects in the contemporary situation and to investigate a wide range of physical, biochemical, haematological, neurological, cardiovascular and nutritional indicators.

To assess the independent effect of kava use as a risk factor for pneumonia and ischaemic heart disease, two case-control studies were conducted concurrently. All admissions to hospital of individuals aged over 15 years for pneumonia and ischaemic heart disease from the Miwatj Region's communities for periods during the mid-1990s were studied. These admissions were compared with randomly-selected controls who were not admitted to hospital and matched for age, sex and home community.

Using historical records and information gathered during community consultation processes, a history of the development of the regulatory system now being implemented to control kava supply and distribution is provided. Some parameters useful to monitor the health and social effects of kava's continued use are outlined.

Results

The study found no convincing evidence that kava use is associated with serious irreversible health effects.

In the cross-sectional study, kava users showed a characteristic 'dermopathy' (scaly skin) and increased levels of some liver enzymes and lower lymphocyte counts. These changes were found to be reversible with decreased exposure to kava or with stopping kava use. Markers of cardiovascular risk were increased across the population but were not elevated in kava users. Kava users displayed lipid profiles similar to those observed in *anorexia nervosa*. No evidence for long-term neurological damage was found in those who had, in some cases, used kava continuously for up to 18 years, since 1982 when it was first introduced to Arnhem Land. Average kava consumption reported in this study in 2000 was 118g/week which is considerably less than the average reported in the early 1990s (368g/week) and perhaps one-third the levels

reported in the 1987 pilot survey. The data indicate that in 1999, 53% of males and 27% of females were using kava.

Liver function changes observed in kava drinkers appear to be reversible with no evidence for long-term liver damage. Clinical surveillance in Arnhem Land over 20 years has not documented any fulminant hepatic failure attributable to kava use. This is despite Aboriginal kava drinkers consuming kava's psychoactive constituents in doses from 10-50 times the recommended therapeutic doses for herbal products. Why kava in the form of manufactured extracts could cause liver injury in some populations while there is no evidence for such concerns in Aboriginal kava users who use an aqueous extract of the dried crushed plant materials is not known and requires further research.

No convincing evidence was found that kava users were at an increased risk of ischaemic heart disease. This lack of an association, however, does not rule out the possibility of an increased likelihood of death from a sudden cardiac event in heavy kava users, especially in those who may already have established heart disease.

Similarly, no convincing evidence was found that kava users were at an increased risk of pneumonia. However, for both pneumonia and ischaemic heart disease the direction of non-significant associations suggested possible increased risks among kava users.

Public health concerns about kava use were intense in 1990-91 when in one western Arnhem Land community, around 70% of men and 62% of women were using kava at an average level of 368g/week of kava powder. This represented a dramatic increase from 1989-90 when 46% of men and just 13% of women were using it at an average rate of around 145g/week. This level of use was achieved when the NT Government first tried to regulate kava supply in 1990. The first regulatory system lacked suitable controls negotiated with Aboriginal communities specific to local geographical and social circumstances. Average amount of time/person spent drinking kava doubled between 1989-1990 and 1990-1991 and the proportion of the community's available cash spent on purchasing kava increased markedly. Heaviest consumption levels approached the highest known in Arnhem Land at the time (>610g/week).

Discussion

On its face, this suggests that kava should be banned in Arnhem Land Aboriginal communities. However, people in some Arnhem Land communities decided in 1998 to continue to use kava under a system of controlled supply while others continued to use kava supplied by an illegal trade in kava with no regulation of supply. While this thesis shows no evidence that kava used in the way Aboriginal people have used it has irreversible effects, this is not to say that irreversible effects may emerge if it continues to be used in Arnhem Land. Ongoing monitoring of kava's effects is therefore essential. As a guide, it appears that kava's reversible health effects become more prominent when a community begins to consume it in the range of from 240-425g/week per person on average. The emergence of health effects is likely to accompany social effects and effects on community life with more than two-thirds of the men and more than half the women drinking kava and with around one-fifth of the kava drinkers spending more than 14 hours/week in kava drinking activities and with one-fifth of the available cash in a community used to purchase kava.

Summary of recommendations

While no clear evidence for long-term irreversible effects of kava use by Indigenous Australians emerged in this study, some results are, nonetheless, suggestive of an increased risk in kava users of cardiovascular disease, serious infections and weight loss on a background of poor nutrition. Close monitoring for these potential adverse effects of kava use in Aboriginal communities is recommended in addition to initiatives encouraging moderation in consumption. Policy approaches should ensure that local community control is reinforced over the availability of kava and the profits acquired from any trade in kava.

THESIS PRESENTATION

The thesis is presented as a series of papers including one review of literature pertinent to the topic, one description of the principal methods used, six empirical papers, and two papers and one commentary based on a recently-published editorial discussing policy issues addressing the central theme (see Appendix A for paper details). This is in accordance with the Northern Territory University's "Rules for the presentation of theses submitted for a professional doctorate or higher degree by research" (Appendix B) which provides for published papers to be included as an integral part of a thesis, with appropriate formatting (section 4.4). The Director of Postgraduate studies has recognised that this thesis is predominantly composed of published papers and has approved the thesis presentation (Appendix C).

All of the papers presented in this thesis have been submitted to, published in, or accepted for publication in journals that are listed by MEDLINE or are internationally refereed. To meet the requirement to have the same formatting throughout the thesis with consistent referencing style (Appendix C), it was decided to follow the uniform requirements for manuscripts submitted to biomedical journals, a format which uses the Vancouver referencing system.¹ Several of the papers were published in journals that do not use the Vancouver referencing style or use modifications of it. Therefore, the format of the final version of each paper may differ from the format of the paper presented in each chapter of the thesis.

Where the paper has been published, a copy or reprint is included in Appendix A. Where the paper is 'in press', a copy of the acceptance letter from the journal is contained in Appendix A. Where the paper has been submitted, a copy of the acknowledgment of receipt is contained in Appendix A. Figures and tables are placed in the text closest to the point at which they are first cited, to facilitate reading of the thesis. Also, where reference is made to the author's own published or 'in press' work, and where that work is contained within the thesis, the relevant chapter is cited. Again this is intended to facilitate reading of the thesis. A separate reference section for the discussions and conclusions chapter and for the introduction chapter is presented.

Figures, tables and pages are numbered from the beginning of each chapter. For example, chapter one, figure 1 is 'Figure 1-1' while chapter two, table one is 'Table 2-1' and so forth.

The papers presented here have been written as part of and in accordance with a publication strategy approved by the Postgraduate Studies Committee of the Menzies School of Health Research and which was accepted by the Committee along with the proposal for the thesis. Each paper has been written to form separate parts of a cohesive research study as well as individual or stand alone investigations. Four of the papers in the thesis provided essential information about the epidemiology of kava or about other substance use (Chapter 4, paper #3), exposure to kava use (Chapter 2, paper#1 and Chapter 3, paper#2) and the definition of kava using groups (Chapter 5, paper#4). This information provided the basis for further work of co-investigators concerning the neuropsychological effects of kava. Copies of these additional papers are included at Appendix A. The data available in Chapter 5, paper#4 and paper#5, have been used in an editorial introducing a discussion of kava's possible liver toxicity. This editorial, published with this author as second author, forms the basis for the discussion of the global controversy about kava's hepatic toxicity presented in Chapter 8 (page 8-62), and a copy is included at Appendix A.

The papers that constitute the thesis are presented in an order that will allow the reader to understand the context in which each investigation was undertaken and the inter-relationship between each paper. To assist with this, introductory notes are provided at the beginning of each chapter describing the relationships between the paper(s) in the chapter and those in the preceding chapters. The status of each paper (either published, 'in press' or submitted for publication, for example) is also stated at the beginning of each chapter.

The introduction includes a description of the overall aims and the background to the thesis as well as a general review of some of the pertinent literature. It includes a description of the principal study setting, the eastern Arnhem Land (Miwatj) region, and a general description of its population. While parts of the study contain information collected in, or referring to, other parts of Arnhem Land, the Miwatj region

was where most of the information was collected. The study design is summarised in the introduction and an outline of the thesis is provided. Along with the general discussion chapter, the introduction aims to integrate the material presented in the research papers. In the discussion and conclusions chapter, a summary of findings is presented in regard to the central themes of the thesis, and the implications of the work for future research and policy development.

All of the papers in this thesis resulted from a study of kava's health effects carried out by the Menzies School of Health Research which was funded by the National Health and Medical Research Council of Australia. While each paper provides a description of the protocol and methods used and the inclusion and exclusion criteria employed, the specificity of these descriptions may have been limited by the requirements of the journals to which they were submitted, and in the case of published or 'in press' papers, by revisions made in response to comments received from paper reviewers. Some of the specific studies may have focused on subsets of the whole study population depending upon the aims of the paper. As a result the numbers of subjects and definitions of groups from which data is reported and the reference dates for measuring exposures may vary between papers.

Ethical approvals for the research were provided by both the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research and the Ethics Committee of the Northern Territory University. A special memorandum of understanding was negotiated with the Aboriginal community in which the cross-sectional study was conducted. A copy of this along with ethics approvals is provided at Appendix D.

References:

1. International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Ann Intern Med* 1997;126:36-47.

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CHAPTER 1

INTRODUCTION

INTRODUCTION

In this thesis I present the findings of several investigations into the health effects of kava use, particularly heavy kava use. These community-based investigations were carried out in Arnhem Land Aboriginal communities (Northern Territory, NT Australia) since the beginning of 1989. Kava has been used in some parts of the Arnhem Land region by Aboriginal people since 1982, although people knew about kava drinking before 1982 because of interactions with people from Pacific island countries from the early 1960s. I also describe some of kava's social and economic effects and the policy responses that have now been implemented to try to minimise the harms from kava abuse in the NT. The results of this unique set of investigations of kava in Indigenous Australians are interpreted in order to try to improve understandings of the health and social effects of kava use generally.

What is kava?

The name 'kava' describes both the plant and the beverage derived from the rootstock of '*Piper methysticum*' (Forst. f.) ('intoxicating pepper'), a perennial, dioecious shrub which grows to heights of around four metres and which is cultivated in many Pacific Ocean island countries.¹ It is a member of the family Piperaceae, a group of tropical herbs, shrubs, small trees and woody climbers. This family includes many well known Old World spice plants such as *Piper nigrum* L. (the 'black pepper' of commerce), for example.

Studies of kava's botanical origins suggest that '*Piper methysticum*' is not just a single species, but instead one that is better described as a sterile group of cultivars of the wild plant '*Piper wichmanii*' which grows in Vanuatu, the Solomon Islands and on the island of New Guinea.¹ Vanuatu has been proposed as the centre of domestication of *Piper methysticum*.² People have cultivated kava for at least 1000 years and perhaps up to 3500 years³ in what appears to have been a very close association. The plant relies on people for its propagation rather than seed dispersal.¹

A wide variety of cultivars has been described and some of the extraordinary richness of folk taxonomies for *Piper methysticum* has been documented.^{1, 4} The generic term 'kava', or sometimes 'kava kava', has obscure linguistic origins,¹ but it has come to describe the substance in the contemporary scientific literature as well as in commerce around the world. In their knowledge systems, Aboriginal people in Arnhem Land have no folk taxonomy for kava and mostly refer to the powdered material they use as 'kava' without further gradations or distinctions.

Some older Aboriginal people in Arnhem Land also know the terms 'yaqona', 'waka' and 'lewena' but these are not regularly used. Yaqona (pronounced 'yangona') is the Fijian term for kava. 'Waka' is a term for one of the grades of yaqona in the kava trade in Fiji. It is regarded as the strongest and most desirable grade and is derived from the lateral roots of the plant. 'Lewena' is derived from the plant's rootstock. 'Kasa' which is another grade of yaqona is regarded as the weakest and least desirable, and therefore the cheapest, and is derived from the basal portions of the plant's stems. Few Arnhem Land Aboriginal people know the term 'kasa'.¹ The term used throughout this thesis is 'kava'.

There is an extensive ethnographic literature describing kava's social and ceremonial role in Pacific island societies. More than 50 years of anthropological studies in these areas has documented the significance of kava in facilitating ritual practices, invocation of the supernatural, normative behaviours and social processes.^{1, 5-8} Kava continues to have major symbolic significance in these regions. Illustrating its importance, historical studies have documented the observation of kava rituals as an adjunct to colonial contacts with Pacific island societies since the 17th century,^{4, 9} and kava continues to hold an important place in the affirmation of Pacific island culture and independence.¹ Today kava plays a pivotal role in the economy of the region,¹⁰ in maintaining and strengthening Pacific identity¹¹ as well as in ongoing Pacific traditions.¹ In addition to this association with Pacific island culture, kava has also held a prominent position in pharmacopoeia in societies outside the Pacific from the time that its efficacy for a range of ailments began to be indicated in the 19th century.¹²

No studies have been published, nor is there any evidence from my own observations, to suggest that kava has any formal social or ritual role for Arnhem Land groups. It has no place in Aboriginal folk medicine although it has become an accompaniment to many aspects of day-to-day life. Anthropological research has documented songs, rituals and knowledge systems that comprise the rich cultural heritage of Arnhem Land peoples.¹³⁻¹⁶ During the course of research and while compiling the data reported in this thesis, no evidence emerged that kava has become part of a body of knowledge that is passed from one generation to the next in the kinds of formal ways that Aboriginal cultural heritage is transmitted. Kava has no role in the important song cycles or rituals that have ceremonial significance for Arnhem Land Aboriginal people. However, during the 1990s, songs pertaining to kava were created by Arnhem Land musicians and began to find their way into contemporary constructions of Aboriginal music¹⁷ indicating that cultural processes that integrate innovations from the wider society are probably continuing in the contemporary context. By contrast, tobacco plays a role in ritual. Songs for both tobacco and alcohol are part of cultural heritage in many parts of Arnhem Land. Tobacco and alcohol have a much longer history in the region and were probably items of trade with Macassan trepangers since at least the 18th century in northern Australia.^{18, 19} Kava drinking in Arnhem Land is recreational or is an adjunct to other activities in peoples' lives. The ethnographic literature contains almost no mention of kava or its social role in Arnhem Land societies in contrast to the extensive literature that describes the significance of kava in Pacific island societies.

Kava's pharmacological and toxicological properties – why do humans use it?

Systematic research to discover the nature and modes of action of kava's psychoactive constituents began in the latter part of the 19th century¹ with the first major scientific examination of kava published in 1886 by L. Lewin.^{1, 20, 21} As a result of this and subsequent research, we have an extensive body of literature on the subject. This literature has helped us to understand something of the pharmacological basis for kava's attraction to humans.

'Kava lactones' or 'kava pyrones',²² are the components generally believed to produce kava's desirable mood-altering effects, mainly muscle relaxing and anxiolytic actions.²⁰ Kava lactones are extracted from the raw plant material using a variety of techniques. The extracts consumed are usually aqueous emulsions or ethanoic or acetonic extracts. While the lactones have been known since the work of Lewin and others,²⁰ the mechanisms through which they act in humans to alter mood and behaviour are not yet completely understood.

While kava clearly has mood altering qualities, in ethnopharmacology, the substance has not generally been regarded as a psychotomimetic, i.e. a substance which profoundly alters perception of space and time in the same way that hallucinogens are known to do, but instead it has been regarded as a hypnotic.²³ Nevertheless, it is generally recognised that kava's psychotropic qualities can lead to altered psychic function, behaviour and experience.^{1, 24-27} Kava's recognised pharmacological effects include sedation, sleep inducing effects, analgesia, muscle relaxant and anticonvulsant effects.^{1, 24-27}

Kava lactones are found in the resinous plant components and are a group of mostly lipid soluble pyrones of the sesquiterpine group of lactones with a chemical structure characterised by a 5,6-dihydro- α -pyrone ring.²⁸ In extracts of kava, the lipid-soluble fraction is high in lactone content in comparison with the water soluble component.²⁶ The water soluble fraction appears to be largely inactive but may cause sedation without ataxia or loss of muscle control.²⁶ The potency of kava lactones has been attributed to the six main lactones found in the lipid soluble fraction namely; kawain, dihydrokawain, methysticin, dihydromethysticin, yangonin and desmethoxy-yangonin.^{22, 28-30}

Experiments with animals show that the lactones kawain and dihydrokawain are rapidly absorbed in the gastrointestinal tract and delivered to the brain with peak concentrations reached ten minutes after administration and then followed by a rapid decline.^{22, 25, 29, 31} The other four main lactones, methysticin, dihydromethysticin, yangonin and desmethoxy-yangonin, reach maximum levels after a longer period of from 30-50 minutes, and the decline in their concentrations is slower.^{22, 25, 29, 31} There is good

evidence to suggest that kava lactones have a synergistic effect since lactones administered alone in laboratory experiments appear to have decreased effects compared to when they are administered in combination.^{25, 31, 32}

A recent review of the literature³³ concluded that there is clear evidence that kava disrupts the neurotransmitter systems that underlie behaviour with non-specific interactions demonstrated between kava lactones and most neurotransmitter systems. The review summarised evidence for modulation of serotonergic and glutamatergic systems and the antagonism of dopamine systems and pointed out that while kava probably indirectly enhances the binding capacity of GABA-receptors, it does so in ways that have not been clearly determined.³³ Since the available evidence suggests that it is likely that kava lactones are most active in the limbic system,³³ i.e. in the more primitive brain structures, the effects of kava lactones on behaviour overall are probably best described as weak and diffuse in comparison to the effects of more conventional pharmaceuticals. Animal experiments have also shown that kava constituents may exert their sedative and hypnotic effects in a similar way to the benzodiazepine group of drugs.³⁴ Recently it has been reported that conventional mood-altering drugs, which have well known pathways and foci of neurological activity established in clinical trials, also show a large overlap with what we know about the mechanisms of action of kava pyrones.³⁵

When humans drink kava, its muscle relaxing effects become evident in their ataxia, slowed reactions and a tendency for photophobia with a weakness in the lower limbs accompanying the ataxia.³⁶ These relaxant effects and kava's reputation as a calming substance in its social use have led to controlled clinical studies to determine the efficacy of kava for reducing anxiety. These studies were reviewed in a recent meta-analysis of 14 double-blind randomised controlled studies that used a Hamilton Rating Scale for Anxiety.³⁷ The meta-analysis showed that kava demonstrated superiority over placebo in treating anxiety and tension of a non-psychotic origin. Release of muscle tension is consistent with reports that kava lactones act to directly alter neuronal excitability through voltage dependent ion channels.^{35, 38-40} The local anaesthetic effects of kava are most directly recognised as a rapid numbing of the mouth and tongue when an aqueous kava mixture is ingested. It has been suggested that these

effects are as potent and long lasting as those of some local anaesthetics used in clinical practice.²⁵ It is possible to also attribute these effects, along with kava's reported analgesic and anti-convulsant effects, to the inhibition of voltage gated ion channels.³³

This reputation of kava as an effective natural anxiolytic was recognized early in the 1990s when a special expert committee (Commission E) of the German Federal Institute for Drugs and Medical Devices (BfArM) recommended that kava could be prescribed for treating symptoms of anxiety.⁴¹ The dosage recommended by BfArM was 60-120mg/day kava lactones to achieve therapeutic effects.^{41, 42} The use of manufactured extracts of kava, including for the treatment of anxiety, expanded rapidly around the world during the 1990s but mainly in Europe and north America.^{10,43} The expansion of interest in herbal medicines in western societies as an alternative form of therapy⁴⁴ was probably facilitated by the power of internet marketing. Kava's success in particular is likely to have been facilitated by enhanced access to agro-exports from peripheral economies with this access becoming increasingly reinforced by new forms of economic dependency relationships with central economies in a dynamic world trade environment in the 1990s.¹⁰

The success of kava as an alternative therapy, however, appears to have been short-lived. Kava is now internationally controversial and this is of direct relevance for this thesis since Aboriginal people continue to use kava while concerns about the possible hepatic toxicity of kava have emerged in western countries. Serious irreversible liver damage has been reported from several countries since 1998 in people using herbal products that contain ethanoic or acetonc extracts of kava lactones.¹⁻³ Since 1999, 11 cases in Europe and two in America were reported of severe hepatic toxicity with liver failure that required transplant and four of these patients died.⁴⁵ One case, also fatal, has been reported in Australia.⁷ Consequently, manufactured kava-based products have become subject to medical alerts or bans on their sale in Europe,⁴ north America^{5,6} and Australasia.⁷ The reasons for the extraordinary success of kava followed by its dramatic decline and the effects this has had in Pacific island kava exporting economies are beyond the scope of this thesis and probably warrant a separate study entirely. However, the results of the investigations reported in this thesis have informed this current debate about kava.

Kava in Australia

There is evidence that the kava plant was transported to Australia with indentured labourers who came to work in the cane fields of north Queensland around 1869,⁴⁶ but no evidence has been found that the plant was grown in the area. Government authorities reported kava use in the Torres Strait around 1910.⁴⁷ Pacific islanders living in Australia have used kava and continue to do so in accordance with cultural practices.⁴⁸ A small group of Pacific islanders from Tonga and Fiji, the majority from Tonga, live in the Miwatj region where this study was carried out, primarily in the regional centre. The kava that Pacific islanders have used is imported to Australia from home countries in the Pacific. Australia also has a natural therapies industry that includes kava as one of its components. The Commonwealth's Therapeutic Goods Administration had 87 products that included kava on its register at 14 August, 2002 when concerns about kava's liver toxicity were formally recognized.⁴⁹ No other indigenous group in Australia appears to have taken up the practice of drinking kava to anything like the same extent as Aboriginal people in Arnhem Land.

Kava and Aboriginal People in Arnhem Land

Kava is not grown in Arnhem Land or anywhere else in the NT. But since 1982, Aboriginal people in some Arnhem Land communities have used it.⁵⁰⁻⁵² Dried and powdered parts of the plant from Fiji, Tonga and Vanuatu have been imported to Arnhem Land since 1982 for local consumption.⁵¹ The use and abuse of kava in some segments of the Aboriginal population in Arnhem Land since that time have created widespread concern about its health effects.^{52, 53}

The events and circumstances surrounding the introduction of kava to Arnhem Land have been documented in a number of published and unpublished reports.^{51, 52, 54-57} Many of the people involved in the introduction of kava to Arnhem Land are still living and working in the region. Through the Uniting Church Aboriginal Advisory and Development Services (AADS), some of the communities in Arnhem Land came to be supported in their community development by workers who were themselves from

Pacific island countries. In late 1981, the first of four visits to Fiji was made by local Aboriginal people in the company of their community workers. The purpose of the visit was to study village development. During this visit, while residing with their Fijian hosts, the people participated in kava drinking. The visitors became attracted to its potential use in their home community as a recreational drug that had mood-altering properties that did not lead to the violence associated with excessive alcohol use, a problem that had emerged in their particular community for the first time during the 1970s. Following their return to their community in eastern Arnhem Land, they arranged for continuing supplies of kava through a merchant in Sydney. Over the next few years, the idea of kava drinking spread to other Arnhem Land communities so that by 1985 it was being used in most of the Arnhem Land communities that now have a history of ever having used kava.⁵⁸

Evidence for increased kava consumption in Arnhem Land

Alarm about the rapid uptake of kava use by Aboriginal people arose soon after its introduction. At first, the main concern reported was the tendency for kava drinking to take place at the expense of normal work patterns.^{55, 59} However, rapid increases in kava use were reported in Arnhem Land populations during the 1980s and concerns began to shift to the health consequences of its use as weight loss and the appearance of a scaly skin began to be seen in kava users.^{51, 60} These trends have been summarised in two reviews.^{52, 57} By 1993, 11 years after kava's introduction, the total amount of kava being used in Arnhem Land, when it was last possible to gather reliable information from importers, was reported to be 28 tonnes a year.⁵² To put this into some perspective, this represented 10% of the total amount of kava exported to all countries from Fiji, the major kava exporter in the Pacific, at the time.¹⁰ Around 60% of men and 40% of women in Arnhem Land were reported to be using kava.⁵²

Evidence for adverse health and social effects in Arnhem Land

Epidemiological work in central Arnhem Land carried out in 1987 and published in 1988 reinforced concerns about a relationship between kava consumption and ill health. Mathews et al described ‘heavy’ kava use associated with health effects such as a scaly skin, a “puffy” face and a patient’s own concern for their health.⁶¹ Amongst people classified as ‘very heavy’ users of kava there was found a greater likelihood of being underweight and having greatly increased levels of γ -glutamyl transferase (GGT), indicating abnormal liver function.⁶¹ In this pilot study, kava users showed ECG evidence and dyspnoea suggestive of pulmonary hypertension, decreased platelet volumes and decreased numbers of blood lymphocytes, effects on blood biochemistry with increased high-density lipoprotein levels and decreased levels of plasma protein, albumin, urea and bilirubin. Kava users were also found to be more likely to show haematuria and to excrete urine of low specific gravity with a high pH. Neurological examination indicated increased patellar reflexes but without deterioration of performance on simple measures of cognition.⁶¹ The study recommended urgent action to reduce the consumption of kava in Aboriginal communities and to improve the nutrition of Aboriginal people who were using it.⁶¹

During the late 1980s, compelling *prima facie* evidence emerged for considering kava as a risk factor for heart disease. This came about as a result of anecdotal reports combined with clinical evidence that heavy kava use, in particular, was a risk factor for sudden cardiac deaths in the Aboriginal population in Arnhem Land.⁶² Heavy kava use coupled with heavy and recent alcohol use, were proposed as risk factors for sudden deaths among young Aboriginal sportsmen.⁶³

Concerns that kava use was a risk factor for serious infectious disease also emerged during the 1990s. In particular, it was proposed that kava use was associated with melioidosis, for instance.⁶⁴ Clinical observation also suggested that many people suffering from pneumonia were kava users (Professor Bart Currie, personal communication).

A hospital presentation for an acute neurological event associated with heavy kava use in an individual who did not use alcohol and with no other identified cause⁶⁵ was an example of other unusual presentations to some of the health clinics in the region's communities during the mid-late 1990s. This widened the implications for heavy kava use even further to include concerns about its neurocognitive effects.

The controversy about kava's possible health effects,⁶¹ its pharmacological effects, along with public commentary^{48, 66} informed Northern Territory policy makers' responses to concerns about Aboriginal people using kava.^{67, 68} To address these concerns, the NT Parliament enacted legislation (May 1998) for the second time in eight years to control kava marketing and use.^{52, 69}

Critics of the public health importance of research findings in the pilot study⁶¹ and of the level of public health risk that kava posed suggested that ill health effects purported to be caused by kava use were instead due to a special situation that reflected Aboriginal peoples' substandard socioeconomic position within their own country.^{1, 24} This suggestion in a very brief debate is compelling given indigenous Australians' high levels of morbidity and mortality from other causes. These matters are of considerable political importance as well⁷⁰ and underscore the need to better understand the broader context of kava's social effects. A perfunctory debate about an enduring issue in Aboriginal health remained unresolved.

Summary questions

Are there increased health risks for kava users, and if so, what are these risks? Is kava use associated with health effects and social and economic disruption in Aboriginal populations? If so, what are the options for minimising these effects where they exist?

Mathews et al⁶¹ suggested that further research could be fruitfully directed to examine whether the health effects of kava use are reversed when usage ceases. These authors also suggested that research should be conducted to examine interactions among the effects of kava and alcohol and poor nutrition or 'anorexia' seen in kava users.⁶¹ In

view of their preliminary findings of health effects in Aboriginal kava users they recommended ‘..urgent social action..’⁶¹(p.545) to reduce the consumption of kava and to search for other means by which its harmful effects might be prevented.⁶¹

Some specific questions were proposed by these authors⁶¹ including the following. Does long term kava use cause hepatic damage that progresses to cirrhosis of the liver? Is kava a contributory cause of sudden death in small numbers of young men from kava using communities? Does the use of kava cause acute or chronic pulmonary hypertension and related cardiac problems? How does kava use cause changes in red blood cells, platelet volumes and blood lymphocytes and what are the consequences of these changes?

The group of studies presented in this thesis describing the epidemiology of kava use in Arnhem Land and some of its health consequences represents a unique body of information. No comparable surveys have been published to describe kava’s health effects in other societies although there appears to be increasing concerns about kava’s effects in the Pacific island region as lifestyles and kava consumption patterns are changing in that region.⁷¹⁻⁷³ Investigations reported in this thesis provide the only comprehensive set of community-based observational studies of kava use that exists in the literature.

Alcohol as a Possible Confounder of Kava’s Effects.

The effects of heavy and episodic alcohol use in Aboriginal populations are widely known.^{74, 75} Kava was introduced to Arnhem Land, in part, to address the issues of alcohol abuse that had emerged during the 1970s.⁷⁶⁻⁷⁸ Some health effects of kava identified in the pilot study were similar to those found in heavy alcohol users.⁶¹ However, an independent effect of kava itself was supported by data from a small follow-up study where, among six kava drinkers who had moderated or abstained from kava, three had abnormal liver function tests that returned to within normal ranges of variation when reassessed eight months later.⁷⁹ Critics of the earlier pilot study in Arnhem Land suggested that the effects of alcohol abuse and the abuse of other

substances such as tobacco, may also account for the observed health effects or confound any independent effects of kava.^{1, 24} The series of studies presented in this thesis also addresses the possibility of confounding effects of other substance use, especially alcohol use.

Aims of the Study

The thesis has four aims. Since ischaemic heart disease and pneumonia are major causes of morbidity and mortality in the Aboriginal population of the NT,⁸⁰⁻⁸³ it was considered important to assess whether kava has contributed to this serious health burden. The study investigates the role of kava consumption as an independent risk factor for ischaemic heart disease (IHD) and serious infectious disease c.f. pneumonia, given the *prima facie* evidence for their association with kava use. The study investigates whether kava consumption is associated with an increased risk of these diseases and whether kava may interact with other risk factors for IHD and pneumonia.

To provide background for these studies, and mindful that no epidemiological study has investigated kava's health effects since 1987, a second aim of this thesis was to conduct a cross-sectional study similar to that carried out by Mathews et al.⁶¹ The objectives were to compile data that could be used to compare the health status of kava drinkers with those who do not use it in terms of a range of haematological, biochemical, physical and neurological parameters and to compare and contrast these results with the previous cross-sectional study where possible. These data permitted an assessment of immune abnormalities in kava drinkers, and an examination of some of kava's neurological effects and differences in markers of cardiovascular disease risk between kava users and non-users. Data from the cross-sectional study also permitted closer investigation of possible liver toxicity in kava drinkers along with a consideration of the causes of poor nutrition and changed lipid profiles among Aboriginal people who use it.

While it is known that kava continues to be used in Arnhem Land despite regulatory efforts, little is known of the extent of its use and what social and economic impacts

that may occur in association with its use? A subsidiary aim of the thesis was to document some of these aspects.

A further aim of the thesis was to examine the policy responses aimed at minimising kava's harmful effects. The use and abuse of kava has been a major concern for regulatory and health authorities in the Northern Territory for more than twenty years. There have been two attempts by the NT Government to regulate the supply and consumption of kava since it first began to be used in Arnhem Land in 1982. One of these attempts occurred in 1990^{52, 53} and the other occurred more recently in 1998.^{57, 68, 84} This thesis examines these regulatory efforts and considers their impacts on kava use and its ill health effects together with some of kava's social and economic impacts. Since kava is also now internationally contentious, because of liver toxicity possibly associated with the use of herbal remedies that contain kava, this thesis also addresses some of the contentious unresolved issues in the debate about the banning or restriction of kava-based products in western countries. Implications for Indigenous Australians and Pacific islanders who continue to use kava are also discussed.

Main questions the study seeks to answer

The study seeks to answer these specific questions.

Question 1. Pneumonia and ischaemic heart disease

Is kava use associated with an increased risk of pneumonia? Is kava use associated with an increased risk of ischaemic heart disease? And if so, are these risks increased with increased kava use? Furthermore, do these risks occur independently of alcohol or other substance use?

Question 2. Cross-sectional study

In a sample of Aboriginal people living in one community in the Miwatj region where kava has been used, do we find that increased exposure to kava use is associated with abnormal biochemical, haematological, immunological, cardiovascular and

neurocognitive findings? Furthermore, do these risks occur independently of alcohol or other substance use?

Question 3. Specific effects of kava

What evidence is there for effects of kava use on liver function, cardiac function and nutritional status? Are these effects associated with increased exposure to kava use? Furthermore, do these risks occur independently of alcohol or other substance use?

Question 4. Patterns of kava use and social and economic impact

To what extent has kava been used in Arnhem Land communities and what social and economic impacts have occurred in association with its use?

Question 5. Policy consequences

What kinds of policies and interventions have been implemented to minimise the harmful health and social effects of kava use? How have these policies and interventions developed, what has informed their development and, what can be learned from this experience for development of future policies and interventions?

Question 6. International implications

What are the implications of this research for the use of kava as a mood-altering substance in other parts of the world?

Location of the research - Arnhem Land and the Miwatj Region

Figure 1-1 shows the localities where kava has been used in Arnhem Land. The Miwatj Region of eastern Arnhem Land was found to be a convenient setting within which to conduct the major part of this study (Figure 1-2). The indigenous population of the Miwatj Region was estimated to be 7130 in 1996.⁸⁵ The population reached 7940 by 2001.⁸⁶ The majority of these people (78%) live in 10 communities with 200-1500 people. The balance live in a further 10 homeland localities and also occupy around 100 smaller family out-stations distributed throughout the Miwatj Region's 37,997km² (Figure 1-2). Aboriginal people in the region lead a lifestyle based on traditional social

Figure 1-1. Kava using communities in Arnhem Land (Northern Territory, Australia)

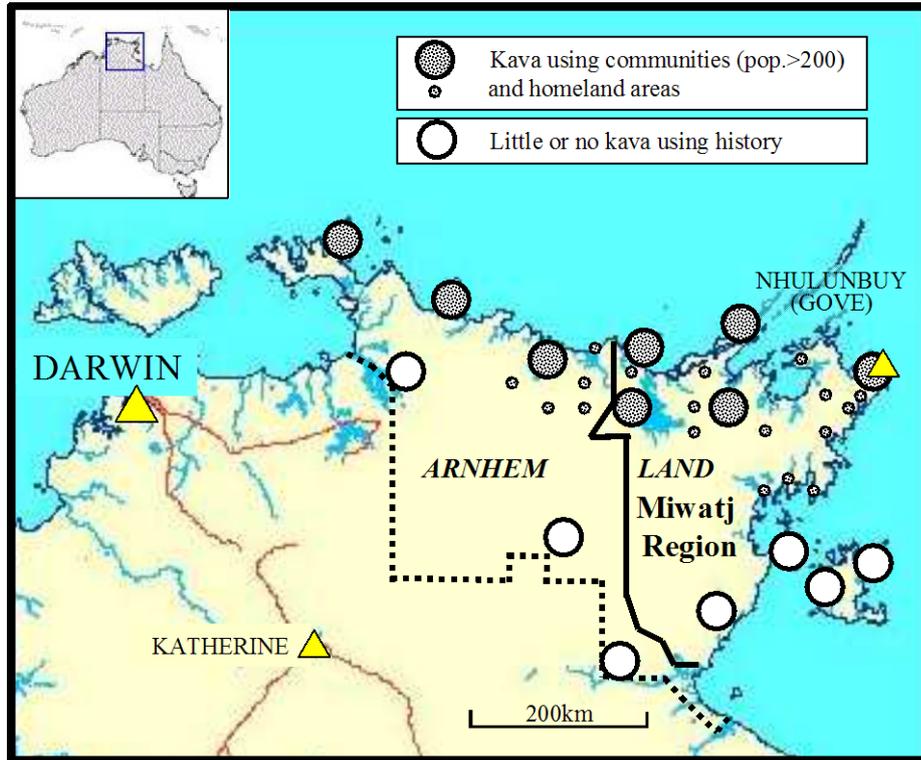
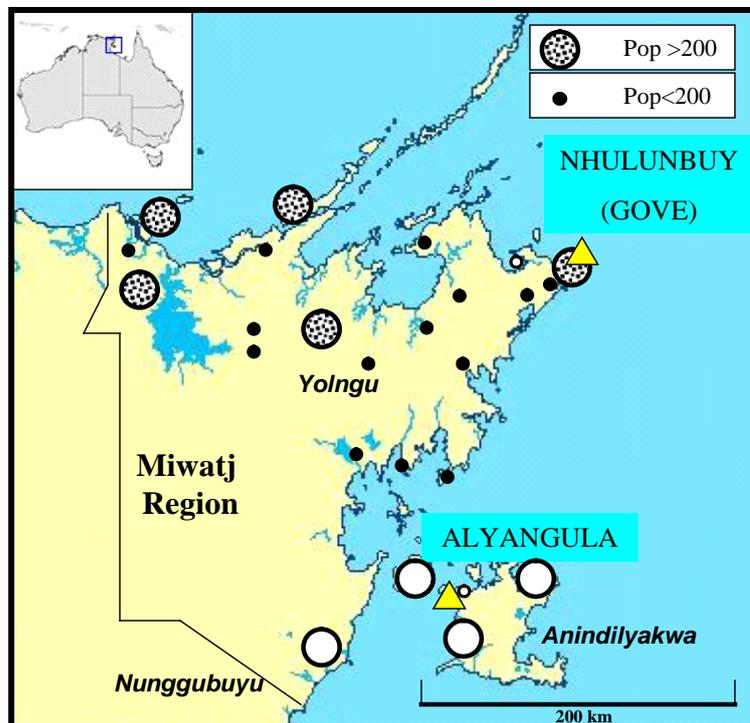


Figure 1-2. The Miwatj Region (eastern Arnhem Land, NT)



practices with mortuary rites and secret-sacred ceremonies continuing to be an important part of life and with language and social concepts more or less intact. There are three distinctive social groupings within the Aboriginal population (Figure 1-2). The largest, approximately 4700 people, identify as *Yol \u* and speak one of the closely interrelated dialects of *Yol \u matha*.⁸⁷ This group occupies homeland areas and communities in the northern and western parts of the region. *Anindilyakwa* people number around 1500 and live in three communities located on Groote Eylandt and one smaller island closer to the mainland.⁸⁸ *Nunggubuyu* people number around 900 and live primarily in one community in the far southern corner of the region.⁸⁹ The *Yol \u* people in the northern parts of the region have used kava regularly since 1982 whereas the *Nunggubuyu* and *Anindilyakwa* people have had little or no access to kava.

There are two mining towns (Nhulunbuy, 3443 people and Alyangula, 994 people in 1996⁸⁵) where the majority of the non-indigenous population resides (Figure 1-2). The development of both towns and the mines which they service has exerted a major influence on the lives of Aboriginal people living nearby and has also influenced the lives of those living in other parts of the region. These towns are service centres for their respective subregions. These services are utilised to varying extents by the region's Aboriginal population with Nhulunbuy the major regional focus. Alcohol is available in both of these towns but access to alcohol is restricted in all of the region's Aboriginal communities. Aboriginal people have had regular access to alcohol in the towns of Nhulunbuy and Alyangula for at least 30 years⁹⁰ although it was restricted in the latter town in 1988. People also drink alcohol when visiting Darwin, the NT's major urban area.

A regional hospital is located in Nhulunbuy to which most people are admitted from the region's communities when the need arises (Figure 1-2). Occasionally, people from these communities are admitted directly to Royal Darwin Hospital (RDH) or the Katherine District Hospital (KDH) (Figure 1-1).

Overall Study Design.

To attempt to answer the study questions three methodological approaches were used. The major part of this study was a case-control analysis to determine any association with heavy kava consumption (particularly in combination with alcohol, smoking and other risk factors) and admission to the regional hospital with IHD or pneumonia as a primary diagnosis. Secondly, using a cross-sectional approach a community-based study was conducted in which neurological, biochemical, haematological, and immunological methods as well as some physical diagnostic techniques utilised in cardiovascular assessments were used to determine possible adverse effects of heavy kava consumption. To assess patterns of kava use and their social and economic impact, cross-sectional data from interviews conducted in the study in one community along with the random sample of controls in the case-control study were integrated with data collected during a community-based participant observation study. Finally, policy approaches and interventions are described and reviewed using observations and data from consultations and discussions carried out since 1989.

A focus on the regional hospital at Nhulunbuy (Figure 1-1) as the primary referral centre for the eastern Arnhem Land region made for a discrete study area with a central service point. For the case-control studies which formed a major part of the research for this thesis, it was possible to identify those admitted to hospital for specific health conditions and compare them with a sample of otherwise healthy people who had not been admitted. Kava use patterns and data for other relevant risk factors could be compared between these groups. For the cross-sectional study carried out in one of the region's communities, clinical data could be cross-checked with hospital admission information. Detail of the methods used is included in the methods section of each paper.

OUTLINE OF THE THESIS

Chapter 1: Introduction

The thesis is comprised of this introduction and the following chapters and included papers. The present chapter has provided some background and a statement of the aims and objectives of the thesis and the rationale for the study. It also provides a brief description of the region and the population in which the study was carried out. The next chapter comprises a review of some of the literature pertinent to the topic.

Chapter 2: Review of literature on kava dosage and consumption

Paper #1: Kava in Arnhem Land: a review of consumption and its social correlates
Drug and Alcohol Review 2000; 19 (3): 319-328.

A review of relevant literature was undertaken prior to developing ways to measure exposure to kava use to evaluate the information available about patterns of kava consumption in Aboriginal people. While Mathews et al had identified 'very heavy' kava users as using in excess of 440g/week of kava powder,⁶¹ the review revealed that it was difficult to directly relate this level of consumption with the dosages administered in controlled trials and laboratory studies which have relied mostly on standardized kava extracts administered under conditions controlled within the studies with no uniform approach to dosage across the studies. Furthermore, before the review was carried out, it was not clear how the dosages normally consumed by Aboriginal kava drinkers compared with what was known about dosages consumed in Pacific island populations. In the literature and commentary about kava and Aboriginal people that was published during the 1980s and 1990s writers often declared that Aboriginal Australian kava users consumed 'vast dosages'⁷⁶ at rates '100 times'⁹¹ greater than in Pacific island societies. If this was the case, then these usage levels would have clearly distinguished Aboriginal kava drinkers as a very unusual group making any health effects found in this thesis unique and beyond comparison with any other population or with the results from controlled studies of kava's pharmacological effects in humans and animals. The aim of this review, then, was to describe the evidence regarding high

levels of kava consumption by Aboriginal people in Arnhem Land and, if possible, to more precisely estimate the amounts that people normally consumed.

The paper published from this review reported that the evidence for much greater kava consumption among Aboriginal people compared with Pacific island groups was anecdotal and that contrary to this anecdote, weekly consumption of kava in Arnhem Land appears to have been similar to consumption levels known in Pacific island societies. This result provides a more accurate perspective on Aboriginal kava drinking and also implies that the results of studies of kava use in Aboriginal Australians can be used to inform similar studies in other populations of kava users.

Chapter 3: Principal methods and procedures used

Paper #2: Validity and utility of community health workers' estimation of kava use *Australian and New Zealand Journal of Public Health* 2002; 26 (1): 52-57.

Finding ways to estimate exposure to kava use and to other substance use retrospectively was the principal challenge in this research, and especially for the case-control studies. The numbers involved in the case-control studies, the cross-sectional study and the study of deaths meant that kava use information was required for around 1200 people or one quarter of the region's Aboriginal population aged over 15 years. Some of these people I knew were dead; I had attended the funeral ceremonies for some of them. And it was possible to interview just a small proportion of those living. From my knowledge of the movements of Arnhem Land people, I also knew that many were no longer resident in the region or moved regularly between the NT's major urban centres and their home communities. Some lived in the larger communities that are more easily accessible but others lived in the smaller, remote homeland localities already described that are distributed across an area of almost 40,000km².

In this diverse area with a highly mobile population I had to find a systematic way to retrospectively measure exposure to kava use in people who may have been exposed to its use off and on for up to 15 years. Logistics were an issue, but the greater difficulty

was that because of the controversies about health effects and the ‘black market’ trade in kava in Arnhem Land, kava drinking had become a highly stigmatised activity. Community tensions were further exacerbated when kava became illegal four months after the study commenced. These were major challenges to be overcome for the successful conduct of the work.

One of the approaches taken was to collect data by proxy by interviewing knowledgeable Aboriginal health workers and supporting these proxy estimates with documentary evidence and interviews where available. The paper features a discussion of the validity and utility of this approach and compares the method used with a more conventional interview approach. To do this, the level of agreement was assessed between a consensus of Aboriginal health workers in two different communities using interviews conducted with community members and health workers and using individuals’ self-reported kava consumption. Exposure measures included history of kava use, current kava use and history of heavy use. Agreement between health workers’ consensus classification and individuals’ self-report was analysed and agreement amongst several health workers in a consensus classification without self-report was assessed.

Methods used to collect data for other parts of the study include the following.

For the cross-sectional study (see paper #4 Chapter 5), the methods used were based on interviews and these data were combined with data collected using the consensus classification approach validated in the published paper (paper #2 Chapter 3). In the participant-observation study (paper #1 Chapter 2), conducted during 1989-91 it was possible to estimate the amount of kava people used by direct observation and to corroborate this with community supply information. Community Council documents and records were used to provide community supply information and data on the community’s cash resources and the amounts spent on kava. Information about the developing regulatory system was gleaned from involvement in consultation processes to implement controls over the availability of kava from 1998 onwards.

Chapter 4 Patterns of kava consumption and the use of other substances in Arnhem Land

Paper #3: Diversity of substance use in eastern Arnhem Land (Australia): patterns and recent changes. *Drug and Alcohol Review* 2002; 21(4): 349-356

This chapter describes patterns of kava use in eastern Arnhem Land. Further to the review of kava consumption levels it was also necessary to describe patterns of other substance use and possible changes in those patterns over time and to estimate the time that people may have been exposed to their use, since the use of other substances was potentially a major confounder of kava's effects. Interview data from the cross-sectional study and from the randomly selected group of controls in the case-control study were used. Data supported the suggestion that kava use declined after it became illegal in 1998 and also supported anecdote of a rise in the use of cannabis in the preceding 3-5 years. Petrol sniffing had receded in the population and alcohol use was unexpectedly low on average. Males were predominantly the users of these substances. Tobacco use was widespread. One quarter of the kava users had used it continuously for 16 years or more at the time of the study. This data was also of importance for the cross-sectional study, which was carried out in March 2000, 20 months after kava had become illegal in the NT, since it was likely that kava use among those studied was lower than in the past. The data provided a picture of a dynamic substance use complex in Arnhem Land that may have confounded the independent effects of kava use, an important consideration for the case-control studies (Chapters 5 and 6).

Chapter 5 Cross-sectional study in one community

Paper #4: Health effects of kava use in an eastern Arnhem Land Aboriginal community *Internal Medicine Journal* [in press]

In one eastern Arnhem Land community, associations between kava use and a wide range of haematological and biochemical characteristics of a sample of 101 individuals were described by comparing a group of kava users with non-users. In this exploratory

study, it was found that kava use was associated with dermopathy characteristic of heavy use, liver function abnormalities, and decreased lymphocytes. While lipid profiles appeared to be disrupted, markers of blood thickening and other cardiovascular risk measures were not different between kava use categories. There were no differences between the kava use categories in terms of any of the neuropsychological tests performed. Possible interactions between kava, alcohol and other substances were difficult to ascertain and required further examination.

Paper #5: Reversible liver function changes in users of aqueous kava extracts *Journal of Toxicology. Clinical Toxicology* [in press]

The participants in the cross-sectional study included a group that had discontinued kava use for at least one year. Others continued to use kava but had their last drink of kava 1-2 months or 1-2 weeks before the study was carried out. Another group of continuing users had used it within the previous 24 hours. This provided the opportunity to investigate the reversibility of changed liver function in former kava users which was already known from clinical observation but which had not been quantified. Liver toxicity associated with the use of manufactured products that contained kava became a controversial issue in many countries at about the same time that this cross-sectional study was being established and carried out. Herbal remedies that contain kava were banned from sale or became the subject of product withdrawals as the international controversy grew. Regulations for the controlled supply of kava to Arnhem Land communities were being developed simultaneously. Policy approaches to support the controlled availability of kava to Aboriginal kava drinkers were therefore challenged by the need to more closely examine possible hepatic toxicity of kava drinking. It was found that liver function changes in moderate users of kava reverse more quickly than was previously thought with no evidence for any long term liver injury in Aboriginal kava drinkers. This analysis more precisely quantified alcohol, tobacco and cannabis use than in previous studies and examined their possible confounding effects.

Paper #6: Kava use, dyslipidaemia and biomarkers of dietary quality in Aboriginal people in Arnhem Land in the Northern Territory (NT), Australia [submitted] *European Journal of Clinical Nutrition*

Evidence suggesting weight loss and loss of body fat possibly associated with poor nutrition in kava users was known from physical assessments carried out in the cross-sectional study. These results were supported by clinical observations and community reports of weight loss in kava drinkers since the 1980s. Poor nutrition has been associated with a possible increased risk of cardiovascular disease in Aboriginal people. The reasons why kava drinkers should show signs of malnutrition or ‘anorexia’ as it was described by Mathews et al⁶¹(p.545) in 1987 were not clear. This paper summarises the cross-sectional data on physical assessments and the results of blood tests for anti-oxidants and lipids. The paper presents hypotheses for further analysis and comparison. Weight loss and loss of appetite may be a direct effect of kava itself or an outcome of an obsession with the practice or an effect of economic deprivation in kava drinkers. Continuing kava users suffered weight loss and loss of body fat and were extremely malnourished. They also had elevated cholesterols but there were no differences in anti-oxidant nutrient levels when kava users were compared with non-users.

Chapter 6 Kava use and hospital admissions for pneumonia

Paper #7: Case-control study of the association between kava use and pneumonia in eastern Arnhem Land Aboriginal communities *Epidemiology and Infection*. [in press]

This chapter investigates a possible association between kava use and admissions to hospital for pneumonia during 1994-97 from the region’s communities. The results of a case-control study are reported. Cases included 115 people aged over 15 years, and for whom kava use information was available, who were admitted to hospital between 1994-97 with symptoms of pneumonia plus an x-ray consistent with the medical officers’ principal diagnosis. A total of 415 controls were randomly selected from community lists with up to four controls matched with each case by age, sex and home

community. In an analysis that controlled for confounders, including the effects of alcohol and tobacco use, no evidence was found for an association between kava use and pneumonia requiring hospital admission.

Chapter 7 Kava use and hospital admissions for ischaemic heart disease

Paper #8: Case-control study of the association between kava use and ischaemic heart disease in Aboriginal communities in eastern Arnhem Land (Northern Territory) Australia. *Journal of Epidemiology and Community Health* [in press].

This chapter examines kava consumption as a risk factor for IHD. The results of a case-control study of admissions to hospital for IHD during 1992-97 are reported. A study design (case-control) similar to that used to investigate the association between kava use and admission to hospital for pneumonia was used. This study used data for 83 cases and 302 matched controls. No clear evidence for an association between kava use and IHD emerged. Twenty people died with IHD as the principal cause without admission to hospital during 1992-1997. Even with these cases and their matched controls included in the analysis, no association between IHD and kava use was found.

Chapter 8 Policy aspects

Paper #9: Enough! or too much. What is 'excessive' kava use in Arnhem Land *Drug and Alcohol Review* 2003; 22(1): 45-53.

'Excessive' kava use has not been defined. This study integrated information from three kava using communities in order to describe 'excessive' kava use more precisely in terms of the onset of health, social and economic effects. It was found that health effects and effects on community and social life emerged when kava was used in the range of from 240g-425g/week of kava powder, when around two-thirds of the males and more than half the females begin to drink it, with almost 20% of available cash in the community being used to purchase it. Health effects with weight loss, elevated

liver enzymes and low blood lymphocytes outside a normal reference range also begin to occur in those who use kava at this level. These parameters may be useful for monitoring the ongoing effects of kava use in Arnhem Land.

Paper #10: Policy approaches to support local community control over kava supply and distribution in the Northern Territory (Australia). *Drug and Alcohol Review* [in press]

An outline of the salient aspects of mechanisms to regulate the sale and distribution of kava in the NT is provided. Beginning in 1998 policy efforts were implemented to support local community control over the sale and distribution of kava. The new regulatory regime features plans to manage kava developed by local Aboriginal communities in consultation with the NT's licensing authority. The key objective of the plans is to control kava supply and the profits from its trade and to eliminate a persistent illegal trade which continues to be a principal threat to the new regulatory system. The new regulatory system has adopted harm reduction principles to reduce kava's health and social effects and has been informed by the results of the research reported in this thesis (Appendix F).

The global controversy about the hepatic toxicity of kava (*Piper methysticum*): a discussion. Based on the following published paper:

Currie BJ, Clough AR. Kava hepatotoxicity with Western herbal products: does it occur with traditional kava use? *Medical Journal of Australia* 2003; 178:421-422.

The discussion in this section is based on this recently-published editorial for which I was second author.⁹² The editorial commented on two papers, one reported the first case of fatal liver failure in an Australian patient who had used a herbal remedy that included kava as one of its constituents⁹³ and the other reported a viewpoint from the Pacific highlighting the lack of any evidence of liver toxicity despite Pacific Islanders having used kava extensively for centuries.⁹⁴ The latter paper raises several issues of contention that I attempt to address in this unpublished discussion.

Chapter 9 Discussion and conclusions

The principal results of the study are summarized and the strengths and weaknesses of the study discussed. Recommendations are made for further research, policy and practice.

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CHAPTER 2

REVIEW OF LITERATURE ON KAVA DOSAGE AND CONSUMPTION

INTRODUCTORY NOTES

Paper #1: published

Clough AR, Burns CB, Mununggurr N. Kava in Arnhem Land: a review of consumption and its social correlates. *Drug and Alcohol Review* 2000;19:319-28.

A principal concern for the study was to quantify the amount of kava people consumed and to compare these estimates with other published and unpublished data since it had been declared in the literature and in public commentary that Aboriginal people used kava at much higher levels than in other populations. To do this, the amounts of kava observed to be used by people in 26 different kava drinking sessions where I was present were estimated. These estimates were compared with an estimate based on community supply information which was available from community Council and supplier records. Because of the long period of interaction with the community (30 months), I came to know kava drinkers very well. A second aim of the paper was to describe associations between kava drinking and the social settings where it was consumed so that these could be used as markers for assessing exposure to kava use in other studies reported in this thesis. Aboriginal health workers became pivotal in this task as is described in chapter 2 and the associated paper.

The data reported in the present paper remain unique in the literature and since opportunities for this kind of participant-observation for extended periods are now rare, it is likely that these data will remain so. While the study clarifies kava dosage, it also conveys the difficulties of determining precise consumption estimates in community settings for a substance like kava with its varied chemical composition and where social factors influence the concentrations of the brews consumed.

Kava in Arnhem Land: a review of consumption and its social correlates

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ABSTRACT

The inconclusive debate about the effects and public health importance of the way Aboriginal people drink kava was early confounded by anecdote imputing health effects. Anecdote and comment have promoted the perception that dosage levels amongst Aboriginal people were much greater than in Pacific island societies. We review published data about kava consumption evaluating it with respect to information collected from participant observation in one Aboriginal community in Arnhem Land (Northern Territory) where people tend to consume kava at a steady tempo; 37g of kava powder containing around 3800mg of kava lactones in 670mls of water in an hour. Contrary to anecdote, weekly consumption of kava in Arnhem Land appears similar to consumption levels in Pacific island populations. The highest levels of consumption in Arnhem Land have been reported to be up to 900g/week of kava powder with heavy consumers drinking at least 610g/week, levels comparable to estimates for Pacific-island societies.

The significance of a steady tempo of drinking means that an individual's weekly kava consumption relates to the amount of time spent drinking which, in turn, is correlated with categories of social setting of drinking ($p < 0.0002$). Lone drinkers appear to be the heaviest users. Lowest consumption takes place in private domestic situations where people enjoy kava as part of family group activities. Surrogates of consumption levels may be found in local socio-economic circumstances. This approach may be useful when more direct measurement of consumption is difficult or impossible.

KAVA

Pharmacological research on kava (*Piper methysticum* Forst. f.) has documented physiological and neurocognitive effects.¹⁻³ Epidemiological work in Arnhem Land suggests an association between heavy kava consumption and indicators of ill health.⁴ Changing use patterns in Arnhem Land populations,⁵⁻⁸ possible health effects,⁴ pharmacological understandings along with public commentary^{9, 10} have informed Northern Territory policy makers' responses to concerns about Aboriginal people using kava.¹¹⁻¹³ The NT Parliament enacted legislation (June 1998) for the second time in eight years^{7, 14} to control kava marketing and use.^{11, 15} Would-be wholesale and retail licensees under the proposed management regime want to predict likely turnover and kava consumption levels amongst their customers. A licensing authority presumably needs some idea of health effects at different levels of consumption.¹⁶

Estimates of levels of kava consumption in the literature have usually been population based using data on the amount of kava delivered to a community or group of communities and estimates of numbers of consumers;⁷ seldom by other means, for instance, from self-reports in face to face interviews,^{5, 6} Estimates of average consumption per capita, and not the extremes of consumption, predominate in published reports. Just one study attempted to describe the range in consumption levels.⁴ None have used ethnographic methods of participant observation to describe styles and tempo of drinking. This paper attempts to address these gaps.

One of our concerns is that in the scant literature and published commentary on kava in Australia it is often declared that Aboriginal people in Arnhem Land have consumed it at extremely high levels, far greater than in Pacific island societies and that consumption takes place outside of a context of social controls.^{3,10,17-20} Anthropological literature on Arnhem Land societies, unfortunately, says little generally about kava's social context and is silent on estimates of consumption.²¹⁻²⁴ Results presented here indicate that the social context of drinking is likely to be closely linked to consumption levels, a matter of some importance to the inconclusive debate about kava's ill health effects. In what remains the principle systematic study, despite

acknowledged difficulties of estimating individual variations in daily and long term use at different levels of kava consumption, it was found that some observed health effects increased with levels of use⁴ and that signs of some of these effects apparently reverse upon ceasing use.²⁵ Critics of this research, and of the public health importance of its findings, pointed to the small numbers of ‘heavy’ and ‘very heavy’ kava users in the study and to the lack of published findings of similar morbidity in Pacific region kava-using populations and suggested that the cause of ill health effects may instead be due to a special situation that reflects Aboriginal people’s substandard socioeconomic position within their own country.²⁶ Their suggestion is compelling given indigenous Australians’ high levels of morbidity and mortality; matters of considerable political importance.²⁷ How *do* Aboriginal people in Arnhem Land drink kava, how much, under what social circumstances and can we make comparisons with other studies?

A Drink of Kava in Arnhem Land.

Kava can be consumed by drinking an infusion of either the dried powdered parts of the plant or of the fresh ground root, or by swallowing the product of a commercial extraction process.²⁸ The preferred method in Arnhem Land is an infusion of dried powdered kava in cold water, preferably chilled. Kava’s psychotomimetic² components, kava lactones, are mostly suspended in the infusion with smaller amounts in solution.²⁶ Cups of, usually, about 100ml are taken, a size comparable to a “standard bowl” in Fiji, for example.²⁹

What quantity of these lactones do Aboriginal people consume? Total lactone content of kava powder may be 10-15% (average 12.5%) of dry weight depending on the ecological conditions of plant growth and the plant parts used to manufacture the powder.^{26, 30} The effectiveness of lactone extraction in the brew is, of course, not standardised, subject to social-contextual as well as physical variations in the substance.³⁰ An earlier view suggested that just a small fraction, perhaps 5-10% (average 7.5%) of available lactones are extracted in a cold water infusion of dried powder.^{2,31,32} Later work indicated that the efficiency of extraction of the active constituents in infusions of 33g/l of kava powder in water is from 81%³³ to 83%.²⁹

Powdered forms available in commercial markets in Fiji appear to be adulterated to the extent that they are 22% to 29% deficient in active constituents.³⁴ The major active constituents all seem to deteriorate (at varying rates) in storage.³⁵ By contrast, infusions of *fresh* kava are consumed in village settings and public drinking places in Vanuatu. Before drying, plant parts are 80% water while lactones make up only 2-3% by weight.^{2, 31} The efficiency of lactone extraction from fresh kava is reported to be considerably greater than from infusions of dried powder.³⁰ Commercial processing, storage and handling of the dried kava powder are some of the many steps between the active constituents in the original substance available and the components that Aboriginal people in the end consume.

We do not infer that all of the health effects of kava in Arnhem Land result from ingesting lactones. The increased inactivity and signs of poor nutrition observed in those who drink kava on a background of poor health require careful interpretation. Mindful of these complexities, our aim here is to develop a method to estimate the amount of kava lactones consumed by individuals using hitherto unpublished data from participant observation including the mass of kava powder used in infusions, a likely dose of kava lactones, an estimated volume of liquid ingested and the amount of time over which consumption takes place. Results are reduced to a weekly basis, for local social and economic relevance. Our second aim is to reclassify published kava consumption estimates using this method introducing a categorisation of kava consumption according to social groupings. We correlate these with observed consumption levels from participant observation.

METHODS

(i) *Information from one community - group consumption of kava*

Information from community Council documents and field diaries of 30-months continuous residence from January 1989 to July of 1991 in one community in Arnhem Land (AC) describes many aspects of social life there including 26 social gatherings where kava was consumed. The slow tempo of kava drinking, close relationships with members of the small community and the long period of residence enabled the compilation of information. Notes of participants and their duration of stay at gatherings where kava was consumed while the observer was present were compiled. For each infusion seen prepared the amount of powder used was recorded, and it was possible to estimate the volume of liquid used and the approximate proportion of each mix subsequently discarded. While participating, the observer noted the number and estimated volume of the cups he consumed himself. The number of person-hours, estimated weight of powder mixed and estimated volume of liquid in infusions were totalled and averaged over occasions. Hourly rates of consumption of kava powder and the liquid infusion as well as the concentration of the infusions were calculated. The time spent drinking kava in a week was estimated from knowledge of individuals' attendance at other gatherings where kava was consumed in the community sometimes by direct observation but usually by reports from other community members and checked where possible by informal interview and discussion. This made it possible to assign kava users to one of five categories based on the time spent drinking. The mid-points of these categories are 2, 5, 9, 14 and 24 hours per week involved in activities where kava was also consumed. This number of hours spent drinking in a week respectively describes the behaviour of individuals who: drink occasionally on one night a week, those who drink regularly on one night a week, those who drink two nights a week, those who drink at least two nights a week but also during the day, and those who are known to regularly drink kava for 24 hour periods.

ii) Information from one community - community supply

For the total period, the amount of kava arriving in the community was recorded and checked with both the kava supplier and the airline that delivered it. Very little kava arrived in the community from sources other than the principle supplier and usually in small amounts in personal luggage.

iii) Two methods of estimating kava consumption levels

A per capita weekly kava consumption was estimated firstly, by dividing the total amount of kava that arrived by the number of people drinking it and the number of weeks in the period (Method 1). Secondly, the estimated hourly rate of consumption was multiplied by the estimated person-hours spent in a week drinking kava and divided by the known number of people drinking (Method 2). This unique information is used in confidence and with thanks for permission and encouragement from the local Council and community elders, who while remaining anonymous, wish to contribute to current research and debate about kava, the levels at which it may be used, its health effects and the recently enacted control measures. Current control measures and the sensitive contemporary political context of kava in Arnhem Land do not permit the kind of participant observation research that is required to produce this kind of data as it is difficult or even impossible to collect now.

(iv) A classification of social settings of drinking

Working with Aboriginal informants, a classification of kava users according to social settings of drinking was developed using recent data and knowledge of drinking styles in the eastern Arnhem Land region. Seven categories were developed: ceremonial and celebratory gatherings, drinking in tribal leaders' circles, household groups, card games, close companions and friends, people drinking alone and people who earn a regular income and who also regularly spend money to buy kava. In developing this classification we recognised that while there may have been changes in the kinds of

social settings in which kava is consumed, the steadiness of the tempo of drinking within the social settings appears to have remained remarkably similar over the 17 years since kava was introduced to the region.³⁶ It is this steady tempo that enables us to link the participant observation data from a prior period with contemporary perceptions of the social settings of drinking. Despite the steady tempo, the levels at which individuals have consumed kava appear to have changed considerably. Our instrument will assist to estimate levels of kava consumption under the present circumstances where kava is illegal. To do this we retrospectively apply this recently-developed classification to the participant observation data from one community. Accordingly the 83 people who were observed to consume kava in the western Arnhem Land community were grouped retrospectively according to these categories. While not mutually exclusive, the categories give an indication of the primary social setting within which people have consumed kava and continue to do so. The correlation between these categories and the estimated time devoted to drinking, and therefore consumption levels, was calculated using Fishers' exact test.

RESULTS AND DISCUSSIONS

i) Estimated consumption levels

Our participant-observation model shows that in 1989-1991 people prepared kava for drinking at a rate of 39g (36g-41g) of powder infused with some 700ml (667ml-752ml) of water in an hour. But they did not quite drink this much because usually about 5% of the liquid in each mix was discarded. Spillage and other waste were negligible. In an hour each person drank approximately 670ml (633ml-715ml) of liquid containing the products of a brew of 37g (34g-39g) of kava powder. Concentrations remained steady over the period at 54g/l-55g/l. The median volume of a single drink also remained steady at a median of 100ml for 274 cups consumed by the observer. Just under seven 100ml cups were normally consumed in an hour. The lactones available in 37g of kava powder may be 4600mg (12.5% of 37g). But, as already discussed, since extraction efficiency is likely to be around 82% of this, the lactones actually consumed may be around 3800mg an hour.

While the tempo of drinking remained steady, community level changes occurred. From January 1989 to June 1990 (78 weeks), some 379kg of kava arrived, an average of 4.9kg/week. From July 1990 to July 1991 (57 weeks) 1611kg arrived, an average of 28.5kg/week, a five-fold increase over the previous period (Table 2-1). These rates need to be adjusted upwards for the nine weeks in 1989-90 and nine weeks in 1990-91 when no kava arrived. In 1989-90, 34 people drank kava and 78 were non-drinkers. In 1990-91, there were 83 drinkers and 42 non-drinkers. The proportion of drinkers increased from just over 30% to 66%. Men drinking kava nearly doubled over the period while women drinking increased five-fold. In addition, the time spent drinking kava increased dramatically. The structural reasons for this and its consequences are complex and will be addressed in another paper.

Table 2-1 Estimated kava consumption in one community

| | 1989-90 | 1990-91 |
|---|------------|------------|
| Calculation of weekly consumption | | |
| Kava arriving per week (g of powder) | 4900 | 28500 |
| Number of drinkers (n) | 34 | 83 |
| Person-hours spent drinking kava per week | 128 | 803 |
| Method 1 - based on community supply and numbers of drinkers | | |
| 1 Weekly per capita consumption (g of powder) | 144 | 343 |
| 2 Adjusted for 5% kava liquid discarded | 137 | 326 |
| 3 Adjusted for 9 in 78 weeks (1989-90) and 9 in 57 weeks (1990-91) when no kava arrived | 163 | 408 |
| 4 Adjusted for both | 155 | 387 |
| Method 2 - based on participant observation | | |
| 5 Weekly per capita consumption (g of powder) | 128 | 377 |
| Weekly per capita consumption (hrs spent drinking) | 4 | 10 |
| Weekly per capita consumption (ml of liquid) | 2383 | 6917 |
| Average of five values from Method 1 and Method 2 | 145 | 368 |

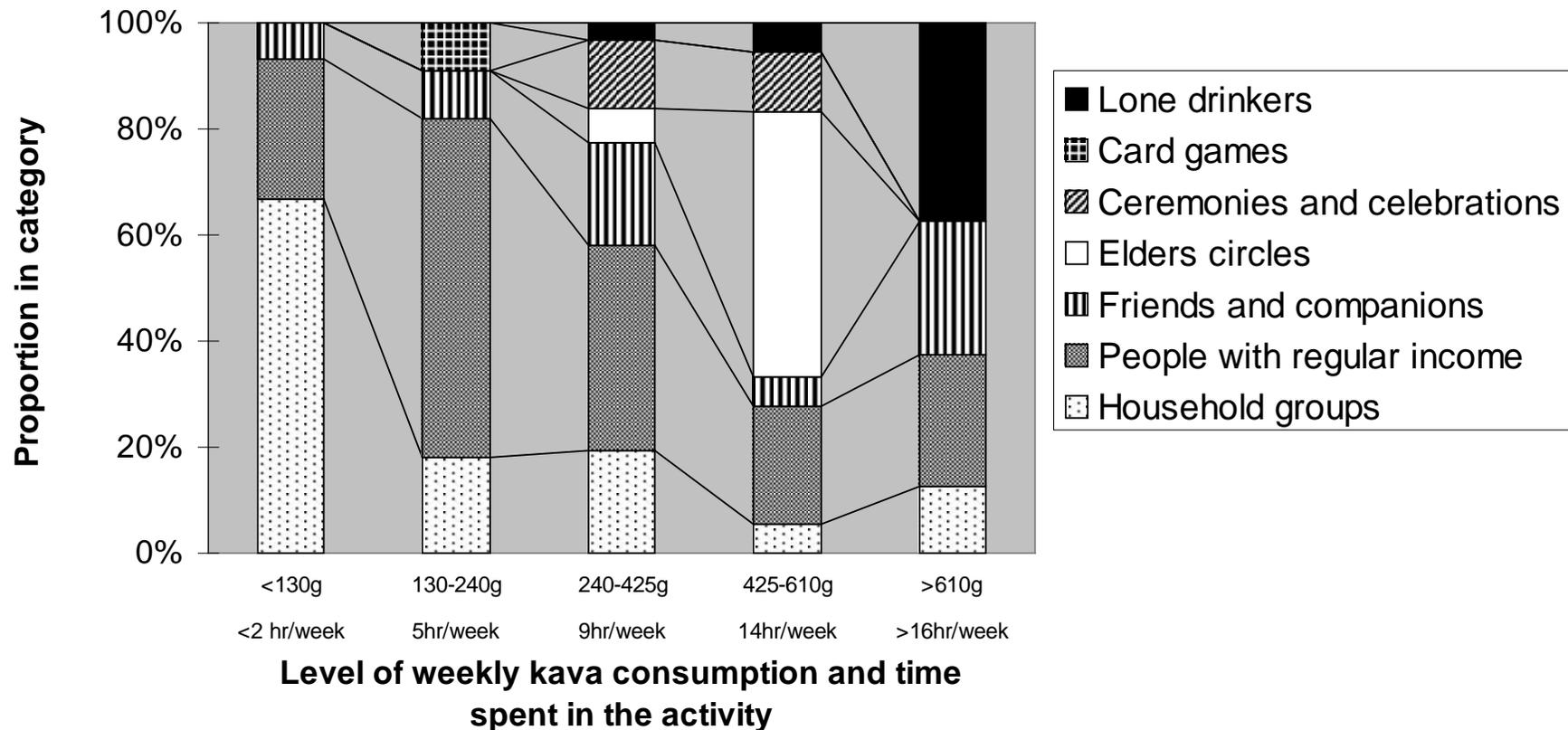
Table 2-1 shows calculations of weekly per capita consumption using two methods. Their convergence suggests that participant observation methods have accounted reasonably well for known quantities of kava delivered to the community. We arrive at our final estimate of weekly per capita consumption by taking the average of five calculations from the two methods (Table 2-1). So, in 1989-90 in this community, 34 people each consumed around 145g/week of kava powder equivalent to around 15000mg of lactones (i.e. 82% of 12.5% of 145g), imbibing about 2.4 litres of infusion and taking 4 hours to drink it. In 1990-91, 83 drinkers each consumed about 368g/week in 10 hours of drinking, ingesting 6.9 litres of infusion or some 38000mg of lactones .

In a nearby community in 1987, Mathews et al defined ‘very heavy’ (using 440g kava powder/week), ‘heavy’ (310g/week) and ‘occasional’ drinkers (100g/week).⁴ In 1989-90 in the community from which we drew our observations, kava users at that time could, on average, be described as ‘occasional’. But by 1990-91, kava users had become ‘heavy users’ overall. These differences across time and between localities in the weekly levels of use occurred against a backdrop of a steady tempo of drinking and probably reflect geographical and temporal circumstances in the uptake of the practice when compared to the nearby community studied by Mathews et al.

ii) *Social settings of drinking and levels of consumption*

Figure 2-1 shows 83 kava users in 1990-91 categorised according to the observed amount of time each spent drinking, the amount consumed and the social setting within which the individual was normally seen to participate. There is a correlation between these categories ($p < 0.0002$, Fisher’s exact test). People drinking alone were the heaviest users (upwards of 610g/week). Drinking in leaders’ circles was heavy (425g-610g/week). Most people drinking on ceremonial occasions used 240g-425g/week as did close companions and friends. People earning income drank at a range of levels but mostly at 240g-425g/week. Low levels of drinking occurred amongst private family groups, (<130g/week).

Figure 2-1. Categories of kava consumers and weekly consumption in one community (1990-91)



Comparison and Evaluation

In Table 2-2 we extrapolate from published data (bold type) to other elements of consumption. There is surprisingly little information about consumption levels available from studies of Pacific island societies.³⁷ But the levels reported are revealing. In Vanuatu, lactones consumed in infusions of *fresh* ground root are reported to be some 6000mg/person per week in just 600ml of liquid.^{26, 37} If Aboriginal kava users are extracting all available lactones, an unlikely situation, Arnhem Land consumption levels could be no more than 6-10 times greater than in Vanuatu at average levels of consumption.

Surprisingly high consumption levels are reported from Tonga. Tongan men, it is reported, may spend around 50 hours a week and drink up to 13 litres of kava a night, equivalent to 1800g-5000g of powder a week, levels twice and up to six times known extreme levels in Arnhem Land. Tongan informants tell us that we should regard these levels as extreme (Mr Hala Tupou, personal communication) In Ruze's Tongan study of kava dermatopathy characteristic of heavy use, only heavy kava users were recruited.³⁸ By comparison, however, we note that it is reported that heavy users in Fiji, rivalling Tongan men, may consume over 70 litres and 1800g/week, again far more than in Arnhem Land.³⁹

Table 2-2 Summary of estimated consumption levels

| Source and date | Locality | Nature of the primary information | Consumption per week | | | | |
|--|---|--|----------------------|--------------------------------|--------------------------|---------------------------|-----------------------------|
| | | | Time (hrs) | Volume of liquid consumed (ml) | Kava powder consumed (g) | Kava lactone content (mg) | Kava lactones consumed (mg) |
| | | | a=b/670 | b=c/37*670 | c | d=0.125*c | e=0.82*d |
| Alexander et al 1985 | Communities in Arnhem Land | Kava delivered to communities January to May 1985 with all adults 15+ (1970?) presumed to be drinkers | 1 | 700 | 39 | 5000 | 4100 |
| | | Seven communities with from 2 to 44 g/day usage calculated (median = 25g/day per person) | 5 | 3200 | 175 | 21900 | 18000 |
| Alexander et al 1987 | Communities in Arnhem Land 1985-1987 | Seven communities with from 3 to 22 g/day usage calculated (median = 7.5 g/day per person) | 1 | 1000 | 53 | 6600 | 5400 |
| | | 14g/day per person to | 3 | 1800 | 98 | 12200 | 10000 |
| | | 53 g/day per person | 10 | 6700 | 371 | 46400 | 38000 |
| Mathews et al 1988 | Milingimbi 1987 | Occasional users 100g/week | 3 | 1800 | 100 | 12500 | 10200 |
| | | heavy users 310g/week | 8 | 5600 | 310 | 38800 | 31800 |
| | | and very heavy users 440g/week | 12 | 8000 | 440 | 55000 | 45100 |
| | | extreme users | 24 | 16300 | 900 | 113000 | 92700 |
| Miwatj Health Aboriginal Corporation 1992-94 | Two areas in east Arnhem 1992 and 1994 | Individuals screened for a community controlled health service asked about the frequency of kava use and the number of cups taken on average. In 1992 the mean consumption of 143 kava drinkers was 2.5l/week with a maximum of 9.6l/week. | 4 | 2500 | 138 | 17200 | 14100 |
| | | In 1994 107 users drank 2.8l/week with a maximum of 7.2l/week | 4 | 2800 | 154 | 19200 | 15700 |
| Estimates in this paper 1999 | One Arnhem Land community 1989-91 | Average of estimates 1989-90 (150g/week) | 4 | 2400 | 145 | 18000 | 14800 |
| | | and 1990-91 (366g/week) | 10 | 6900 | 368 | 46000 | 37700 |
| | | heaviest users | 16 | 11000 | 610 | 76000 | 62300 |
| Alexander et al 1988 | The same Arnhem Land Community 1985 | Based on kava delivered and her estimates of the total population aged 15+ | 6 | 3700 | 206 | 25800 | 21200 |
| D'Abbs 1995 | The same Arnhem Land Community 1992 | January to October 1992 with 1008kg delivered to the community with a mean of 100.8kg per month and 50 drinkers identified | 13 | 8420 | 465 | 58100 | 47600 |
| D'Abbs 1995 | One community in east Arnhem in 1991-92 | Some 50-66% of the adult males and 33-50% of the adult females drank kava at from 66g to 88g/day | 13 | 8400 | 462 | 57800 | 47400 |
| | | | 17 | 11200 | 616 | 77000 | 63100 |
| Lebot et al 1997 | Vanuatu | Port Vila Nakamals, Vanuatu 1987-88 | 1 | 600 | n/a | 6000 | 6000 |
| Frater 1952 | Fiji | Estimates from general knowledge of consumption levels in Fiji | 104 | 70000 | 1800 | 225000 | 184500 |
| Ruze 1990 | Tongan men | Focused on the male heavy drinkers with prominent skin rash. The 200 drinkers surveyed spend 12-100 hours a week drinking kava, an average of 50 hours | 50 | 33500 | 1840 | 230000 | 188600 |
| | | and in an evening they would drink up to a maximum of 13 litres of kava. | 136 | 91000 | 5000 | 625000 | 512500 |

Can we Relate these Consumption Levels to Pharmacological Effects?

Variable approaches to quantifying dosages make comparisons between pharmacological studies and our data difficult. For example, word recognition decreases with lactone doses of 4200mg/week.⁴⁰ Side effects (e.g. oral and lingual dyskinesiae, tonic rotation of the head, painful twisting movements of the trunk) with doses of 2100mg/week have been described.⁴¹ Doses of 700g/week of kava powder, at the heavy end of consumption in Arnhem Land, were related with observed health effects; and these dosages were thought 'comparable' to those reported in Fiji.²⁵ No effect on alerting and speed of access of information from long-term memory was found with single doses of 120g/l, a dosage (again) thought usual in Fiji, or with doses of 1g/kg body weight, thought to be much greater than usual.⁴² Few obvious acute effects were seen using single doses of kava brewed at a rate of 200g/l, a concentration considerably greater than we have observed (Table 2-1).⁴³ Single doses of 1g powder/kg body weight (75g, or about two hours of drinking for a 75kg adult in Arnhem Land) have been reported to have no effect on word recognition alone but did so in conjunction with alcohol.⁴⁴ Commercially available therapeutic products recommend weekly dosages of 840mg of lactones, somewhat less than likely consumption in our data.⁴⁵ Regulations to control kava consumption in 1990 prevented a licensee from supplying more than 50g/day or 350g/week per person, a comparatively heavy level of use.¹⁶

Notwithstanding variable approaches to dosage, it seems possible that dosage levels routinely used in pharmacological work on humans may be at the higher ends of the range of consumption in secular kava drinking in Arnhem Land. While our model requires more precise calibration, it illustrates the need for caution in generalising from results of pharmacological studies alone.

Conclusions

Anecdotal evidence that kava consumption levels in Arnhem Land were greater than in Pacific island populations appeared in the early 1980s.^{5, 6} These anecdotes have now

become generally accepted.^{46, 47} Early enthusiasm that Aboriginal peoples' behavioural responses to kava were desirable alternatives to their responses to alcohol⁴⁸ has persisted in some quarters.⁴⁹ By the late 1980s, the optimism that kava would alleviate problems of alcohol abuse had waned in the literature even amongst those who had initially lauded kava's use.²¹ Very quickly kava came to be described as a 'craze',⁵⁰ 'cult'⁵⁰ and 'habit',⁵¹ and an example of the 'principle of alien poisons'⁵¹ with 'macabre'⁵⁰ effects. It was declared that Aboriginal people consumed 'vast dosages',⁵¹ up to 'a hundred times'²¹ more than in the Pacific. The influence of such opinion, lends a convincing persuasion to imputed health effects. It has also pervasively influenced policy formulation and research. Such conclusions need to be revisited.

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CHAPTER 3

PRINCIPAL METHODS USED

INTRODUCTORY NOTES

Paper #2: published

Clough AR, Bailie R, Burns CB, Guyula T, Wunungmurra R, Wanybarrnga SR.
Validity and utility of community health workers' estimation of kava use.
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The preceding chapter demonstrated that Aboriginal kava drinkers drink kava at rates that are comparable with rates in other populations, but that we lack a sound basis for comparing results reported in pharmacological studies with the effects of kava in populations that use it. The chapter also demonstrated that to collect similar data on kava exposure for a larger number of individuals in more communities would require a very large investment of resources together with reliable data on community supply.

Preliminary estimates of the sample sizes for the case-control studies and the cross-sectional study indicated that retrospective data for around 1200 people would be required with data about other substance use and other aspects of health status also needed. It was decided for this reason and, because of logistical and ethical reasons and to meet community expectations, data collected from proxy estimates would be an appropriate approach. The data reported in Chapter 2, paper#1 demonstrated that the knowledge of Aboriginal Health Workers could be useful for such proxy estimates. In order to test the validity of this approach, trial interviews were conducted and the results were compared with the results of a consensus classification using assessments provided by knowledgeable Aboriginal health workers.

The reader should be aware that these data were collected in eastern Arnhem Land communities in 1999 and 2000 and do not relate directly to the data collected in western Arnhem Land and reported in the previous chapter.

Validity and utility of community health workers' estimation of kava use.

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ABSTRACT

Objective: Estimating illicit substance use in epidemiological studies is challenging, particularly across ethical, cultural and language barriers. While developing the methods for a case-control study of the health effects of heavy kava consumption among Aboriginal people living in a remote region of the Northern Territory (NT), we examined the validity and utility of alternative methods for estimating exposure.

Methods: We assessed the level of agreement between a consensus of Aboriginal health workers in two different communities using interviews conducted with community members and health workers and individuals' self-reported kava consumption. Exposure measures included history of kava use, current kava use and history of heavy use. Agreement between a health worker consensus classification and individuals' self-report was analysed and agreement amongst several health workers in a consensus classification without self-report was assessed.

Results: Health workers concurred about an individual's history of kava use ($\kappa=0.83$), current use ($\kappa=0.43$) and also level of use ($\kappa=0.33$). There was very good agreement between health workers' consensus and self-reported history of use ($\kappa=0.77$). Agreement amongst health workers about current kava use was poor ($\kappa=0.08$) while there was fair agreement between health workers and self-reported heavy kava users ($\kappa=0.36$). Data from review of clinic patient notes supported agreement between consensus classification and self-reported history and level of use ($\kappa=0.39$).

Conclusions: Self-reported kava use may be a poor estimate of current use especially when obtained from interviews with community members away from a confidential clinic setting. Consensus classification by knowledgeable Aboriginal health workers provided comprehensive coverage, efficiently and with greater reliability and assisted to identify 'excessive' kava use. This approach may have application in exposure assessment in similar settings and for future monitoring of kava's health effects.

INTRODUCTION

The eastern Arnhem Land region of the NT includes 10 Aboriginal townships and associated homeland areas each with from 200-1500 people. These are dispersed across 37 997km² with a total Aboriginal population of 7130 as well as two mining centres where the majority of the non-Aboriginal population (5983 people) reside.¹

Kava is consumed here as an infusion with cold water of dried, powdered parts of the plant *Piper methysticum* Forst. f. imported from south Pacific countries. Its widespread use by Aboriginal people in this 'Top End' region of the NT began in 1982.^{2,3} We seek to develop reliable methods to estimate an individual's kava consumption, particularly 'excessive' consumption for use in epidemiological studies of kava's health effects and to assist to monitor and minimise harm from kava abuse. This research is part of a case-control study of the possible increased risk of admission to the regional hospital for pneumonia or ischaemic heart disease amongst kava users.

'Heavy' and 'very heavy' kava use has been associated with indicators of ill health; scaly skin, weight loss, abnormal liver function, pulmonary hypertension, decreased platelets and blood lymphocytes along with other effects on blood biochemistry, haematuria and a urine of low specific gravity and a high pH.⁴ Recent research and clinical reports suggest heavy kava use is a risk factor for sudden cardiac deaths⁵ and, coupled with heavy alcohol use, is a risk factor for sudden deaths amongst young Aboriginal sportsmen.⁶ Kava use as a risk factor for melioidosis has also been proposed.⁷ In the context of these research findings, and of perceived excessive kava consumption within some Aboriginal communities, policies and strategies have been developed to minimise excessive use and resultant harm. But 'excessive' use has not been adequately described.

Measuring exposure to kava consumption faces particular challenges. The way in which kava is consumed is one. Drinking kava is usually a group activity where each brew is

shared.^{8,9} Kava powder used in infusions is neither of a standard quality nor does it contain uniform amounts of active constituents.¹⁰

A greater challenge for our regional study is that some communities required that we work within the local health centre and not approach individuals in the community.¹¹ With this ethical constraint on research procedures we could not rely on systematic collection in our regional case-control study of self-reported kava consumption from interviews. Because of recent legislated controls, direct or participant observation is problematic legally and kava users may, understandably, be unwilling to disclose their current consumption patterns. While the possession of up to two kilograms of kava is a misdemeanour, amounts above this are ‘trafficable’ or ‘commercial’ quantities under the Kava Management Act (1998).¹²

Previous methods of estimating kava consumption relied on the availability of accurate figures from licensed suppliers to corroborate self-report and consensus classification.¹³ This kind of information was not available after 1994 when the NT Government relaxed controls set in place in 1990 on the supply and sale of kava.¹⁴ The expansion of the informal trade after 1994 and the banning of kava in 1998, made it even more difficult to acquire reliable information, further limiting the capacity to verify estimates.^{15,16}

An important result of an earlier study reporting on participant observations from 1989-91 was that an individual’s weekly kava consumption is related closely to the time spent drinking and, in turn, to the range of social situations experienced where kava is consumed.³ In addition, it was found that 20% of the population in that community drank kava at a level of 425g/week or more, a level which corresponded to spending over 14 hours/week drinking kava.³ This result was corroborated by community supply information over a period of 30 months. To try to overcome some of the aforementioned constraints, we use this result combined with consensus classifications provided by Aboriginal health workers and self-reported data.

METHODS

Exposure measures

Participants interviewed (Group 1 and Group 5, below) were asked whether they had ever used kava ('Yes' or 'No') and if they were current (continuing) users of kava ('Yes' or 'No'). Health workers were asked to rate participants similarly. We also asked health workers and participants to describe the estimated time in a week a subject usually spent drinking kava and the time spent in activities where kava was consumed.

Flip charts were prepared to assist in the interviews conducted by one of us (AC) with the assistance of junior health workers. English and Djambarrpuy\u, a local language spoken by a majority of community residents, were used.

Data Collected

Data can be classified into five groups.

Group 1:

- **self-report** data, n=101 people interviewed in a cross-sectional study (March 2000) in a clinic environment.
- **consensus classification** for each of these 101 individuals by two health workers working in conference. Two health workers who were not in the community at the time (and who therefore could not have known of the 101 responses) were asked to classify individuals with respect to exposure measures. Agreement between self-report and consensus classification was analysed.

Group 2: (n=91) includes consensus classification with respect to exposure measures for each individual in a group selected from the same community for a case-control study of relative risks of hospital admissions amongst kava users

and non-users. Between June and October 1999 up to four Aboriginal health workers were interviewed independently from each other in order to assess exposure to kava use for participants in this study.

Group 3: Chart review data was available for n=24 out of 101 individuals in Group 1 and for n=25 out of 91 individuals in Group 2. Discharge summaries for cases in the case-control study (Group 2) were reviewed and data on exposure to kava use were recorded where available. Comparisons were made with health worker consensus classifications in each of Group 1 and Group 2.

Group 4: Sixteen individuals appeared in both Group 1 and Group 2, i.e. they appeared as participants in both the cross-sectional study and the case-control study. Agreement was assessed between the consensus classifications by health workers and self-report data and also amongst the group of health workers.

Group 5 comprises data for individuals interviewed away from a confidential clinic situation. A random sample (n=20) of 120 individuals in a case-control group in another nearby community was selected. Consensus classification data was collected for these individuals. Additionally, the 20 individuals in the sample were approached in the community in the company of a health worker and interviews were attempted. The purpose of this was to compare data collected by interviewing individuals outside a clinic with both health worker consensus classification and interview data collected while working inside a clinic. But, as is discussed in the 'results' section, many of these individuals were, for a range of reasons, unavailable for interview.

Approvals, Data Recording, Storage and Analysis.

A memorandum of understanding between the local Aboriginal community Council and the Menzies School of Health Research guided the research. Ethical approval was

given by a local ethics committee that works to National Health and Medical Research Council guidelines and which includes an Aboriginal subcommittee with power of veto.

Data were coded for electronic storage and analysed using the Stata statistical analysis package (Version 6, Stata Corporation, College Station Texas). The kappa (κ) statistic was used to measure the differences between observed frequencies of agreement with those expected by chance.¹⁷⁻¹⁹

RESULTS

Group 1: Cross-sectional Data

Table 3-1a shows the frequency of agreement between the in-conference consensus of two female health workers and the self-reported information derived from interviews with 101 people in the cross-sectional study. Data for 98 individuals was ultimately available for comparison. Self-report agreed with health worker consensus on 89% of occasions. The agreement is good according to suggested guidelines ($\kappa=0.77$, $P<0.001$).^{20,21} The two health workers (females) did not agree with self-report on 11 individuals. Ten of these were men. Amongst the women who were assessed (11), agreement between consensus and self-report occurred for all but two of them.

Data was available to compare consensus classification and self-report for 50 kava users with respect to their level of use. Table 3-1c shows that agreement identifying the heaviest kava users (estimated at 425g/week or 14 hours of drinking) was fair (approaching moderate) at a rate of 72% ($\kappa=0.36$, $P=0.006$). But Table 3-1b shows that health workers did not agree with self-reported current kava use ($\kappa=0.08$, $P=0.144$).

Group 2: Consensus Classification data

Here, up to four health workers (male and female) commented on kava use history and use levels for each individual. They didn't always agree, as Table 3-2 shows. Entries in Table 3-2 take into account that there are more than two health workers assessing each individual with variable numbers of health workers making assessments but with just two possible ratings, positive or negative. Variables containing the number of positive ratings and the number of negative ratings are compared for each individual.²² Agreement is very good with only 3 split decisions in a total of 90 about a history of kava use (Table 3-2a) ($\kappa=0.83$, $P<0.001$). For current kava use (Table 3-2b), agreement

is moderate ($\kappa=0.43$, $P<0.001$) with a fair degree of agreement (Table 3-2c) about heavy kava use ($\kappa=0.33$, $P=0.006$).

Table 3-1 Health workers' consensus and self-reported kava use

| (a) History of kava use | | Self Report | | Totals |
|---|--------------|--------------------|-----------|---------------|
| | | YES | NO | |
| Kappa=0.77, z=7.57, p<0.001 | | | | |
| Consensus of two female health workers | | | | |
| | YES | 54 | 5 | 59 |
| | NO | 6 | 33 | 39 |
| | Total | 60 | 38 | 98 |

| (b) Currently using kava | | Self Report | | Totals |
|---|--------------|--------------------|-----------|---------------|
| | | YES | NO | |
| Kappa=0.08, z=1.06, p=0.144 | | | | |
| Consensus of two female health workers | | | | |
| | YES | 32 | 18 | 50 |
| | NO | 1 | 2 | 3 |
| | Total | 33 | 20 | 53 |

| (c) Heavy kava use | | Self Report | | Totals |
|---|----------------------------------|----------------------------------|----------------------------------|---------------|
| | | >14hrs (425g)/week | <14hrs (425g)/week | |
| Kappa=0.36, z=2.52, p=0.006 | | | | |
| Consensus of two female health workers | | | | |
| | >14hrs (425g)/week | 27 | 7 | 34 |
| | <14hrs (425g)/week | 7 | 9 | 16 |
| | Total | 34 | 16 | 50 |

Table 3-2 Health worker consensus on kava use

| a) History of kava use | | Number assessing 'No' | | | | | Totals |
|-------------------------------|--------------|------------------------------|-----------|----------|----------|-----------|---------------|
| Kappa=0.83, z=13.63, p<0.001 | | 0 | 1 | 2 | 3 | 4 | |
| | 0 | 1 | 7 | 1 | 6 | 19 | 34 |
| | 1 | 17 | 1 | 1 | 0 | 0 | 19 |
| Number assessing 'Yes' | 2 | 5 | 3 | 2 | 0 | 0 | 10 |
| | 3 | 17 | 2 | 0 | 0 | 0 | 19 |
| | 4 | 9 | 0 | 0 | 0 | 0 | 9 |
| | Total | 49 | 13 | 4 | 6 | 19 | 91 |

| b) Currently using kava | | Number assessing 'No' | | | | | Totals |
|--------------------------------|--------------|------------------------------|-----------|----------|----------|----------|---------------|
| Kappa=0.43, z=3.72, p<0.001 | | 0 | 1 | 2 | 3 | 4 | |
| | 0 | 0 | 3 | 0 | 2 | 0 | 5 |
| | 1 | 15 | 4 | 2 | 1 | 0 | 22 |
| Number assessing 'Yes' | 2 | 8 | 3 | 0 | 0 | 0 | 11 |
| | 3 | 13 | 0 | 0 | 0 | 0 | 13 |
| | 4 | 6 | 0 | 0 | 0 | 0 | 6 |
| | Total | 42 | 10 | 2 | 3 | 0 | 57 |

| c) Heavy kava use | | Number assessing <425g(14 hours)/week | | | | | Totals |
|--|--------------|---|-----------|----------|----------|----------|---------------|
| Kappa=0.33, z=2.52, p=0.006 | | 0 | 1 | 2 | 3 | 4 | |
| | 0 | 0 | 12 | 2 | 1 | 0 | 15 |
| | 1 | 8 | 6 | 0 | 0 | 0 | 14 |
| Number assessing >425g (14 hours)/week | 2 | 6 | 4 | 0 | 0 | 0 | 10 |
| | 3 | 10 | 2 | 0 | 0 | 0 | 12 |
| | 4 | 0 | 0 | 0 | 0 | 0 | 0 |
| | Total | 24 | 24 | 2 | 1 | 0 | 51 |

Group 3: Chart Reviews and Discharge Summaries

Data from chart reviews for 25 individuals from the case-control study (Group 2) were compared with the consensus of health workers. This was also done for the 24 individuals in the cross-sectional study (Group 1) (Tables 3a and b). The individuals portrayed in each table include five of the 16 who were surveyed as part of both the cross-sectional and the case-control study. Two of these five were also hospital admissions, i.e. cases in the case-control study. The remainder were different individuals in each table. These tables show that the chart reviews supported the consensus classifications in both studies, with a fair level of agreement (60%) between charts and health worker consensus in Group 2 ($\kappa=0.28$, $P=0.022$). There is 67% agreement between health worker consensus and self report in Group 1, a fair level approaching moderate agreement ($\kappa=0.39$, $P=0.004$).

It is informative to try to account for some of the discrepancy in Table 3-3a. In one case the male health worker described a man who, as he himself told us, had recently taken up drinking kava after a two-year break, but who had a record in charts of being a non-user. The health worker's information proved more current than the documentary information. In Table 3-3b one of the two non-users identified by the female health workers but whose charts contained a note that they were kava users was a woman who had recently reduced her kava consumption to occasional use. The other was a man whose chart recorded no kava use within the last two years, recent changes which may not have been noticed by the female health workers. Subsequently the senior male Aboriginal health worker in the community confirmed that this man, in fact, had a history of kava use.

Finally just two individuals had a hospital discharge summary that could be inspected, notes which described both men as heavy users of kava, information that agreed with both the health worker consensus and their self-report.

Group 4: Overlaps Between Group 1 and Group 2

Sixteen individuals appeared in both the cross-sectional study and the case-control group. Therefore information is available from several sources for comparisons. Assessments by up to four health workers in the case-control group, assessments by

Table 3-3 Consensus classification and chart review

| a) Case-control study | Chart Review | | | Totals |
|---|-----------------|-----------|----------|-----------|
| | Heavy kava user | Kava user | Non user | |
| Kappa=0.28, z=2.02, p=0.02 | | | | |
| Consensus of up to four health workers | | | | |
| Heavy kava user | 13 | 4 | 0 | 17 |
| Kava user | 1 | 1 | 5 | 7 |
| Non user | 0 | 0 | 1 | 1 |
| Total | 14 | 5 | 6 | 25 |

| b) Cross-sectional study | Chart Review | | | Totals |
|--|-----------------|-----------|----------|-----------|
| | Heavy kava user | Kava user | Non user | |
| Kappa=0.39, z=2.63, p=0.004 | | | | |
| Consensus of two health workers | | | | |
| Heavy kava user | 12 | 4 | 0 | 16 |
| Kava user | 2 | 2 | 0 | 4 |
| Non user | 0 | 2 | 2 | 4 |
| Total | 14 | 8 | 2 | 24 |

two health workers in the cross-sectional study and self-report in interview provided up to six estimates. Table 3-4 shows the raw data. While numbers are small, agreement on these ratings was very good for a history of kava use ($\kappa=0.82$, $P<0.001$), moderate for level of use ($\kappa=0.42$, $P<0.001$), but was poor for current kava use ($\kappa=0.06$, $P=0.300$).

When self-report data were removed from this analysis agreement amongst up to six health workers improved for both history of use ($\kappa=0.92$, $P<0.001$) and level of use ($\kappa=0.67$, $P<0.001$). Agreement amongst independent health worker assessments of current kava use was unanimous ($\kappa=1.00$, $P<0.001$).

Group 5: Interviewing in a Clinic Compared with Interviewing away from a Clinic Environment

One of the 20 selected had passed away before interviewing commenced. For another eight of these it was impossible to conduct an interview. Two women were temporarily out of the community, another had moved semi-permanently to a nearby community, another was in Darwin having a baby. Two men were incapacitated and could not provide information in interview; an elderly man suffering from dementia, and another grieving from the recent death of a close family member. Two more refused or avoided an interview because of their stated discomfort with providing information about an illicit substance.

For the 11 out of the 20 interviewed, health workers confirmed for nine of them their self-reported history of use. They confirmed answers for all eight that described their current kava use, and for five of eight self-reported descriptions of their level of use. By contrast, health workers readily provided information on all of the 20 including the deceased person.

**Table 3-4 Sixteen participants: self report and health worker assessment
(1='Yes', 0='No')**

| | Self Report | Consensus of two health workers | Health worker 1 | Health worker 2 | Health worker 3 | Health worker 4 |
|-----------------------------|-------------|---------------------------------|-----------------|-----------------|-----------------|-----------------|
| History of kava use? | | | | | | |
| Participant 2 | 1 | 1 | 1 | . | 1 | 1 |
| 4 | 1 | 1 | . | . | 1 | . |
| 8 | 0 | 1 | 1 | . | 1 | 1 |
| 10 | 1 | 1 | 1 | . | 1 | 1 |
| 12 | 1 | . | . | . | 1 | . |
| 45 | 1 | 0 | . | . | 0 | . |
| 46 | 0 | 1 | 1 | . | 1 | 1 |
| 49 | 1 | 1 | 1 | . | 1 | 1 |
| 58 | 1 | 1 | 1 | . | 1 | 1 |
| 63 | 0 | 0 | 0 | 0 | 0 | 0 |
| 68 | 1 | 1 | 1 | 1 | 1 | 1 |
| 80 | 0 | 0 | 0 | 0 | 0 | 0 |
| 88 | 0 | 0 | 0 | 0 | 0 | 0 |
| 93 | 1 | 0 | 1 | 1 | 1 | 1 |
| 94 | 1 | 1 | 1 | 1 | 1 | 1 |
| 118 | 1 | 1 | 1 | . | 1 | 1 |
| Using kava now? | | | | | | |
| Participant 2 | 1 | 1 | 1 | . | 1 | 1 |
| 4 | 1 | 0 | . | . | 0 | . |
| 8 | . | 1 | 1 | . | 1 | 1 |
| 10 | 1 | 1 | 1 | . | 1 | 1 |
| 12 | 0 | . | . | . | 1 | . |
| 45 | 1 | . | . | . | . | . |
| 46 | . | 1 | 1 | . | 1 | 1 |
| 49 | 0 | 1 | 1 | . | 1 | 1 |
| 58 | 1 | 1 | 1 | . | 1 | 1 |
| 63 | . | . | . | . | . | . |
| 68 | 0 | 1 | 1 | 1 | 1 | 1 |
| 80 | . | . | . | . | . | . |
| 88 | . | . | . | . | . | . |
| 93 | 1 | . | 1 | 1 | 1 | 1 |
| 94 | 0 | 1 | 1 | 1 | 1 | 1 |
| 118 | 0 | 1 | 1 | . | . | 1 |
| Heavy kava user? | | | | | | |
| Participant 2 | 1 | 1 | 1 | . | 1 | 1 |
| 4 | 0 | 0 | . | . | 0 | . |
| 8 | . | 0 | 1 | . | 1 | 1 |
| 10 | 1 | 1 | 1 | . | 1 | 1 |
| 12 | . | . | . | . | 1 | . |
| 45 | 0 | . | . | . | . | . |
| 46 | . | 1 | 1 | . | 1 | 1 |
| 49 | 1 | 0 | 0 | . | 1 | 0 |
| 58 | 0 | 1 | 1 | . | 1 | 1 |
| 63 | . | . | . | . | . | . |
| 68 | 0 | . | . | 0 | 0 | 0 |
| 80 | . | . | . | . | . | . |
| 88 | . | . | . | . | . | . |
| 93 | 0 | . | . | 1 | 1 | 1 |
| 94 | 0 | . | . | 1 | 1 | 1 |
| 118 | 1 | . | . | . | . | 1 |

DISCUSSION

Health workers tended to agree amongst themselves about kava consumption for individual community members. Self-report was lower than health workers' estimates. Analysis of Group 5 data, showed that it was more efficient and effective to conduct interviews and assessments with health workers in a clinic situation than to seek to interview individuals who may not have been available in the local community. But a discrepancy in estimated kava use, especially current kava use is highlighted when the data from self-report is compared with health worker classification. Analysis of data from Group 1 shows the extent of this. A frequency of agreement between self-report and consensus classification of just 64% was achieved, a figure little different from expected ($\kappa=0.08$, $P=0.144$). Under reporting of substance use is well known,^{23,24} a problem that was perhaps exacerbated in our research given kava's illegal and, accordingly, stigmatised status at the time. Health workers may have over-estimated kava use. But, while we cannot be certain without more comparative studies, it seems more plausible that participants under-reported their current use in interviews. It is also understandable that, since they concur about the data amongst themselves, in a confidential clinic setting health workers more readily disclose kava use information than individuals interviewed in a more public community setting about their personal substance use habits. Female health workers provided less reliable information about male kava users, which supports the reasonable notion that health workers know more about community members of the same gender, a result which does not detract from the plausibility of the information provided by health workers as a group.

When health worker assessment is the only available data source, in the absence of self-report or other corroborating information, i.e. when it must be relied upon as proxy^{24,25} for more directly-obtained data, our results suggest that we can do so with reasonable assurance. Consensus classification is available to us because of the detailed knowledge that Aboriginal health workers have of their communities which are usually small and isolated with a population of interrelated clan and family groups and where domestic and public interactions are close and well-known. This knowledge has limits

in the kind of context we have described. Health workers are perhaps less accurate when describing current kava usage patterns but more reliable when they assess history of kava use and overall tendencies in levels of consumption. This may be because of limits on what it is possible to know about current events. On the other hand it may be related to the dynamic legal and supply circumstances of kava already mentioned. To illustrate this, out of 26 reformed kava drinkers who provided information in the cross-sectional study, 17 had given up since kava was banned 22 months prior to our study.

Health workers agreed amongst themselves ($\kappa=0.33$) and with self-report ($\kappa=0.36$) about levels of kava use. Chart review data, where available, corroborated this agreement ($\kappa=0.39$). In one community, Mathews et al reported ‘very heavy’ (using 440g kava powder/week), ‘heavy’ (310g/week) ‘occasional’ drinkers (100g/week) and ‘extreme’ use (perhaps 900g/week) in 1987.⁴ In another community nearby in 1989-91,³ average consumption levels of 370g/week were found with heaviest drinkers consuming more than 610g/week. Participant observation established that these amounts were equivalent to 10 hours per week and over 16 hours per week spent drinking kava respectively. Some 20% of kava drinkers in this same community drank at a level of >425g/week, a figure that is equivalent to around 14 hours of drinking in a week and one that approximates the ‘very heavy’ category of Mathews et al. As in previous studies, data on the amount of kava delivered were used to verify these estimates.

In the present study, of the 34 people who described themselves as heavy drinkers (Table 3-1c), a majority (26) said that they had also suffered kava rash in the past. Of the 16 lighter drinkers, just five had ever suffered kava rash. These data suggest that the level of consumption demarcating heavy use may be the level at which people begin to suffer kava dermopathy, a condition reported elsewhere as a characteristic of sustained heavy use.²⁶ Therefore, a level of 425g/week may be regarded as a level upon which attention should be focused to further assess harmful health outcomes amongst Aboriginal populations who consume kava.

Suggested Guidelines for Using Health Worker Consensus Classification Data

With our approach, reliability accrued by applying the same procedure to different individuals with health workers using a similar approach to each assessment under repeatable conditions. These results suggest the following procedures in recording values of exposure to kava use in studies of kava's health effects using health worker consensus where it is not possible to obtain reliable self-report information.

- The value reported by the majority of the health workers who comment should take priority.
- A value reported by a health worker of the same gender as the individual in the study should be given greater weight if there is no majority view.
- If a decision cannot be made with respect to either current usage of kava or a history of use, then the consensus in one can inform a split decision in the other. For example, if by consensus, the person was considered to be a current user of kava, then a split decision about any history of kava use is resolved.
- Discharge summary or chart review data should be used to confirm the consensus, if available. With respect to level of use, the same guidelines apply.
- If a lone health worker provides comment and/or others are not sure, the view of the one should prevail after first checking the chart review and discharge summary data.
- If no value can be decided upon, then the value should be recorded as 'no response/no information'.

Such an approach should ideally be taken to complement self-report information. Note that we do not reject evidence from a single health worker because of an inability to establish consensus. Reliability in this situation depends on health workers' profound ethnographic knowledge. While health workers are not necessarily objective and impartial observers, individually or in consensus, they can tell a great deal about their people, their health and their habits. At this level of detail, epidemiological research can be constructively informed by ethnographic knowledge, the depth and quality of

which amongst health workers in remote Aboriginal communities is unparalleled and hitherto seldom explicitly recognised in published reports. The methods described here may have application to exposure assessment in epidemiological studies of the health effects of other substances and in similar settings around the world, namely in small, rural and remote communities. The comparatively efficient methods developed here may be useful for community health workers to monitor the health effects of ongoing kava use in their communities.

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CHAPTER 4

PREVALENCE OF KAVA AND OTHER SUBSTANCE USE

INTRODUCTORY NOTES

Paper #3: published

Clough AR, Guyula T, Yunupingu M, Burns CB. Diversity of substance use in eastern Arnhem Land (Australia): patterns and recent changes. *Drug and Alcohol Review* 2002;21:349-56.

The preceding chapter established procedures for determining exposure to kava use when data collected by proxy is the principal source of information. Systematic surveys of patterns of drug and alcohol use were last conducted in the 'Top End' of the NT during the 1980s. These data were too old to be of use to inform this study. Recent data on drug use patterns from national reporting systems were not of sufficient level of detail to be of use in this study of one region.

Data were collected using the procedures established in Chapter 3, paper#2 for a randomly selected sample of controls in the case-control study (Chapter 6, paper#7 and Chapter 7, paper#8) which permitted an assessment to be made of the nature of substance use patterns across the Miwatj Region. This was of importance for the case-control studies in particular. Data collected during interviews in the cross-sectional study (Chapter 5) also permitted an assessment of the extent of substance use in one community and of its contemporary economic significance. These data also revealed the extent to which people had used kava and other substances during their lives.

Finally, my current research in Arnhem Land enabled me to incorporate some more recent data into the published paper which demonstrates that kava continues to be used in the region. This was of importance in order to evaluate the need for policy approaches to support controlled availability of kava in Arnhem Land. The data revealed a decline in kava use within a dynamic complex of substance use in Arnhem Land which had not been systematically documented previously.

Diversity of substance use in eastern Arnhem Land (Australia): patterns and recent changes

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ABSTRACT

Objective: Describe patterns of substance use among remote Aboriginal community populations.

Setting: Eastern Arnhem Land ('Miwatj') region of the Northern Territory's (NT) 'Top End', with a population of 4217 Aboriginal people over 15 years of age.

Design: Cross-sectional description and comparison.

Study Procedures: Sample 1: n=689 from the region using data from health worker consensus classification of kava, alcohol, tobacco, petrol and cannabis use. Sample 2: n=101 from one community using self-reported use, age at commencement, duration, amounts consumed and expenditure.

Results: In 1999 (sample 1), 46% of males and 18% of females were kava users, alcohol: 53% males, 12% females, tobacco: 68% males, 65% females, and cannabis: 31% males, 8% females. Less than 5% sniffed petrol. In one community in 2000, 39% males and 20% females reported using cannabis during the previous month. In this community between 1999 and 2000, the proportion of current kava users among men declined (77%-52%, $P=0.015$) with a tendency in women for a decrease in the proportion of tobacco users (87%-69%, $P=0.096$). The increase in the proportion of cannabis users in men (21%-39%, $P=0.068$) was not statistically significant. But in women the increase was significant (0%-20%, $P=0.013$). Gross expenditure on tobacco and kava were similar in 2000; both greater than cannabis and alcohol. Median years used ranged from four years for cannabis and 20 years for tobacco.

Discussion: Data supported anecdote of a recent rise in cannabis use especially in women. Kava use declined in men. Tobacco use patterns in women may have been changing. Average per capita consumption of alcohol was low compared with other 'Top End' areas. Such varied and dynamic substance use patterns pose challenges for research and policy.

INTRODUCTION

Just a few published studies describe contemporary patterns of substance use among local Aboriginal community populations,¹⁻³ or in regional⁴⁻⁷ or national assemblages in Australia.⁸ Data for the local community level is scant especially data describing short-term changes or fluctuations in substance use patterns.

While conducting studies of the health effects of kava use in eastern Arnhem Land (Miwatj Region), we became aware of possible short-term changes and that patterns of substance use were not static. For example, we noted widespread reports of a recent rise in cannabis use, a decline in kava supply to communities, fluctuations in petrol sniffing in young people along with anecdote that small numbers were experimenting with other illicit drugs.

There is ongoing concern about substance misuse in indigenous communities in the NT. Recent media reports describe substance misuse issues surrounding alcohol,⁹ petrol sniffing,^{10,11} kava¹² and cannabis.¹³ Epidemiological data also reflect their impacts, e.g. drug and alcohol psychosis hospital admission rates of Aboriginal males between 1993 and 1997 in the NT were nearly four times higher than non-Aboriginal males.¹⁴ The economic and social burden of substance abuse was recently highlighted by Government through the efforts of indigenous NT Parliamentarians.¹⁵ However, these economic and social costs have seldom been quantified. Drug and alcohol use are increasingly understood to be part of a complex of risk factors for extreme forms of harm and self-harm in indigenous populations around Australia.¹⁶⁻¹⁸ In the NT, commentators¹⁹ and community people alike now unequivocally ascribe such harmful behaviours to substance misuse in remote communities. But there is little current information available to inform discussion of these issues of critical local economic and social concern.

We therefore decided to summarise in this paper information we have available from our studies in Arnhem Land communities. We present evidence suggesting a recent rise in cannabis use and a decline in kava use and possible changes in tobacco use. We briefly describe the substance use economy in one community and report the extent to which alcohol, kava, tobacco, petrol and cannabis have been used in the Miwatj Region.

METHODS

In 1996, there were 4217 Aboriginal people (2005 males and 2212 females) over 15 years in the Miwatj Region²⁰ living in 10 major communities scattered over almost 38,000km², each serviced by a Community Health Centre. Data from two samples in the region's population are used: 1) a random sample of people living in each community in the region and, 2) data from a more detailed study in just one community. These data were collected for the purposes of determining exposure to substance use in order to compare cases with controls on a range of health measures in other related studies. Although the samples were not chosen to represent the age and sex profile of the region, the data provide useful descriptive information. The data were not collected in order to document trends. This was not a longitudinal study. However, the data are unique given that it is unlikely that large-scale regional surveys will be conducted here in the near future.

Sample 1 (n=689) for the Miwatj Region

Sample selection

A sample of 353 males and 336 females was chosen at random from rolls prepared from active community clinic files and patient lists and represents 16% of the study population.

Data Collection: Health Worker Consensus Classification of Kava, Alcohol, Tobacco, Petrol and Cannabis Use

The data collection procedures used were developed in other studies.^{3,21,22} Their validity and utility have been assessed in an earlier publication to which the reader is referred for detail.²³ For each substance, health workers in each community health centre were asked whether subjects had a history of use, and whether they were continuing to use it. These data were collected in 1999. Clinic charts were reviewed to compare with health worker consensus. This

information allowed us to determine those in the population who were current substance users in 1999.

Sample 2 (n=101), Data Collected in 2000 for One Community

A cross-sectional study was conducted in one community in the region early in 2000. In accordance with a memorandum of understanding negotiated with the local community Council, people were invited to the health centre to participate. The sample (65 males, 36 females) was not random. A quota approach was used to attempt to represent community age- and sex-structure, and in the end the sample comprised between 27%²⁰ and 30%²⁴ of the community population. The sampling frame constructed from a community list included 339 Aboriginal people (150 males and 189 females) present in the community at the time of the survey.

Overlap between sample 1 and 2

This community was one of the 10 surveyed as part of sample 1 for the whole region so there was some overlap between the samples. There were 65 people (35 males, 30 females) from this community included in sample 1 in 1999, and sixteen of these 65 also appeared in the more recent sample 2 in this community.

Data Collection: Self-reported Kava, Alcohol, Tobacco, Petrol and Cannabis use.

Participants were asked in interview by one of us (AC) if they had ever used these substances and if they were currently using them, about quantities of each substance consumed within the past month and about the extent to which they shared with others. Responses were checked with two health workers who were not in the community at the time of the study (and therefore blinded to participants' responses) as well as with chart review data.

Amounts Consumed

Quantities used were elicited using flip charts⁴. Volume of alcohol types reported consumed were converted to estimates of pure alcohol.⁸ Participants reported tobacco consumption as the usual number of 'sticks' consumed per day. The packet available in the local cannabis trade, contained less than 2g of leaf. Participants were asked how many of these packets they used and shared on recent occasions. For kava, alcohol and cannabis, the amount reported was multiplied by the number of users estimated in the population yielding a gross estimate of each substance consumed in the community in the month preceding the study.

Community Supply Information

Tobacco is sold through the local community store whose management kindly provided supply data for a 13-week period shortly after the study was conducted when stock holdings and turnover were examined. Supply information for illegal or stigmatised substances was not possible to collect. Kava was banned in the NT in 1998 under the Kava Management Act.^{25,26} Information about cannabis supply to the community was not available and alcohol is locally illegal because the community is 'dry' under the restricted area provisions of the NT Liquor Act as are most communities in Arnhem Land.²⁷ Petrol sniffing was curtailed in the community in 1996.

Estimated Costs of Substance Use

A bag of kava containing around 75g of powder cost \$20 (\$269/kg) and a packet (1.8g) of cannabis leaf \$50 (\$28/g) in 2000. Local prices of tobacco (\$0.31c/stick) and alcohol (\$0.15/g pure alcohol) were estimated in consultation with suppliers. A gross expenditure for a month in the community was estimated by multiplying the estimated number of users in the population by the amounts that were reported in the sample by the cost of each unit (Table 4-2). The average per user is also reported (Table 4-2).

Data Analysis

To estimate the population proportions (see Table 4-1), age-sex specific proportions in the sample were multiplied by the numbers in each age-sex group in the regional population. The results were summed and the total divided by the total regional population to calculate a prevalence weighted for differences between the region's age and sex structure and that of the sample. This method, and the method of estimating confidence intervals for these estimates, is described by Rothman and Greenland.²⁸

Crude prevalence of substance use in 1999 and 2000 in one community were compared using K^2 tests (Figure 4-3). Odds ratios were calculated by logistic regression using Stata 7.0.²⁹

Ethical Approvals

Ethical approval was provided by the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research which works to NHMRC guidelines and which has an Aboriginal subcommittee. Additionally, the MOU negotiated with the community council, governed the management of the research in sample 2.

RESULTS

Sample 1 (n=689) Data for the Region

Inspection of Table 4-1 and Figure 4-1 shows that, in the population of the Miwatj Region, the proportion of current substance users in 1999 was greater among males for each substance. After adjusting for age, males were more likely to be kava users (OR=3.70, 2.80-5.63, P<0.001), alcohol (OR=9.21, 6.06-13.98, P<0.001), cannabis (OR=6.03, 3.05-11.94, P<0.001) as well as tobacco (OR=1.57, 1.09-2.27, P=0.015). All petrol sniffers in the data in 1999 were male.

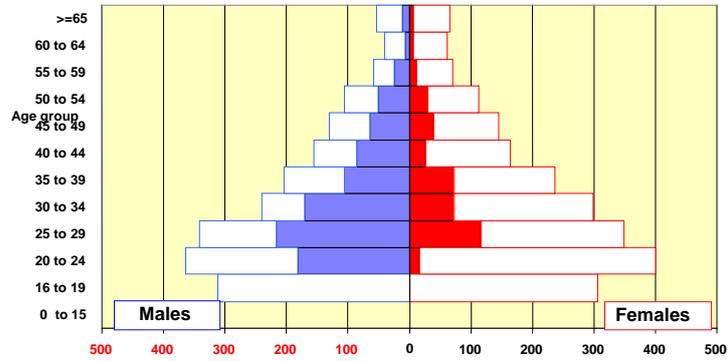
Kava is not used throughout the region unlike the other substances. One quarter of the region's population live in four communities that have not regularly used kava. The proportion of kava users in the six kava-using communities *only* was 53% males, 27% females.

Table 4-1 Prevalence (%) of current users of substances in 1999 in the Miwatj Region (estimated using samples of 353 males and 336 females in a population of 2005 males and 2212 females)

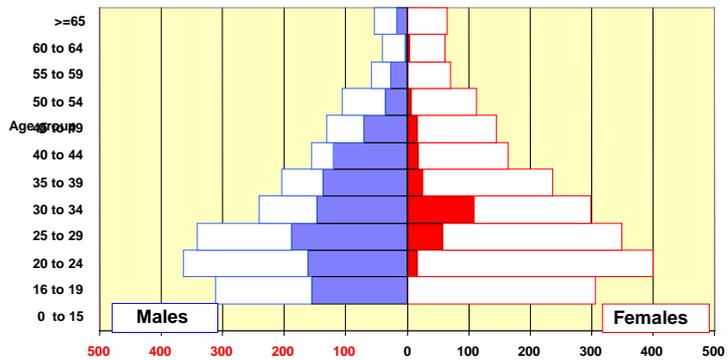
| | Kava % (95%CI) | Alcohol % (95%CI) | Tobacco % (95%CI) | Cannabis % (95%CI) | Petrol % (95%CI) |
|---------------------------|-----------------------------|--------------------------------|--------------------------------|---------------------------------|-------------------------------|
| Males (n=353) | 46 (40-51) | 53 (48-58) | 68 (63-73) | 31 (26-36) | 3 (1-5) |
| Females (n=336) | 18 (14-22) | 12 (8-15) | 65 (60-71) | 8 (5-11) | <1 (0-1) |

Figure 4-1 Substance use, eastern Arnhem Land (1999)
 (estimated using samples of 353 males and 336 females in a population of 2005 males and 2212 females, solid shading represents current users in each age and sex stratum)

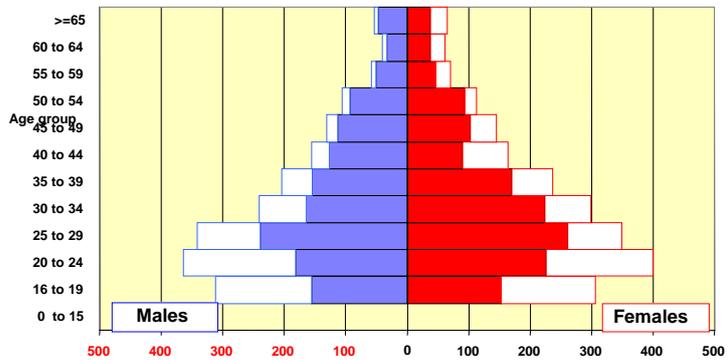
Kava



Alcohol



Tobacco



Cannabis

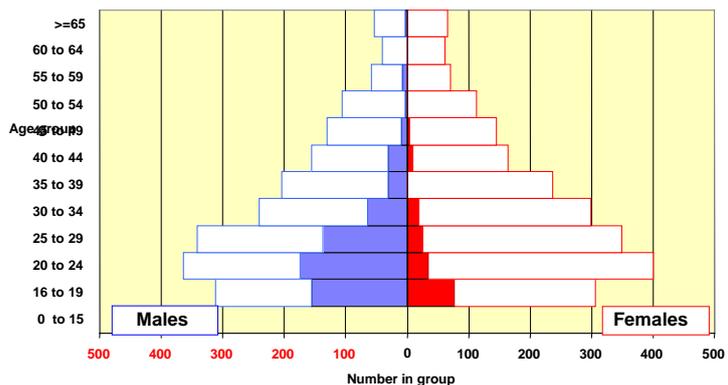
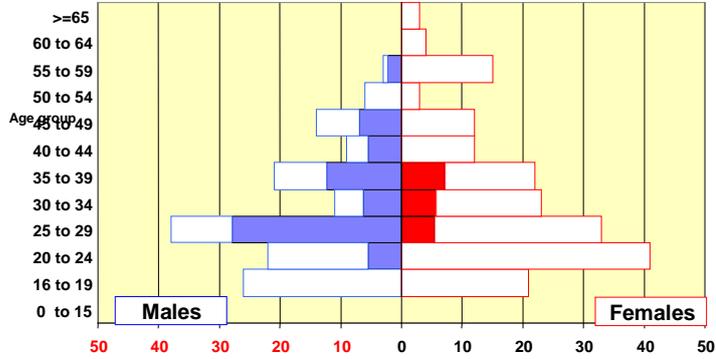


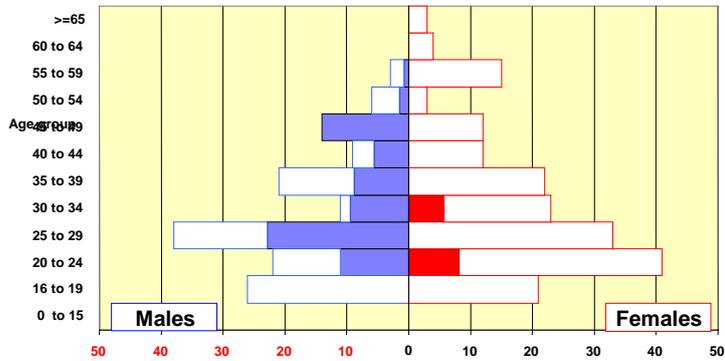
Figure 4-2 Substance use, one community (2000)

(estimated using samples of 65 males and 36 females in a population of 150 males and 189 females, solid shading represents current users in each age and sex stratum)

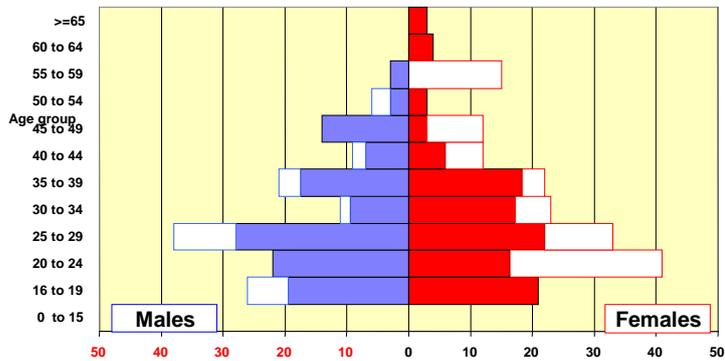
Kava



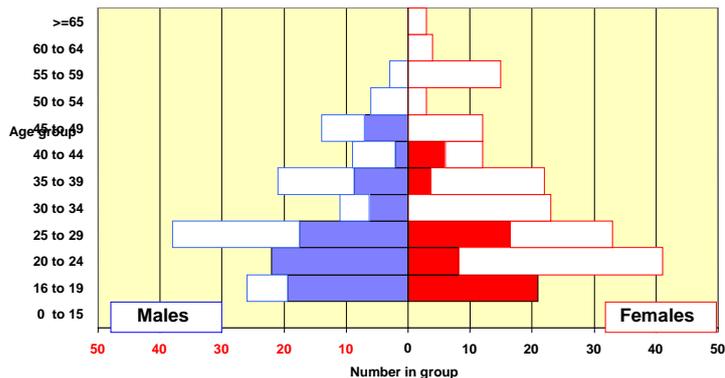
Alcohol



Tobacco



Cannabis



Sample 2 (n=101) Data for the Year 2000 in One Community

In this community in 2000, proportions of current kava users were 52% males and 11% females, alcohol 40% males and 6% females, tobacco 80% males and 69% females, cannabis 39% males and 20% females with few active petrol sniffers (see Figure 4-2). There was no reliable evidence of any history or current use of other illicit drugs in either sample 1 or sample 2.

There were differences between the regional patterns for 1999 (sample 1) and in one community for 2000 (sample 2). The proportion of women using kava in sample 2 (11%) was lower than in the kava using communities in sample 1 (27%). Proportions of alcohol users were lower in both men (40%) and women (6%) in sample 2, compared with men (53%) and women (12%) in sample 1. There were more tobacco users in the men in sample 2 (80%) than in sample 1 (68%). There were more male and female cannabis users in sample 2 (39% males, 20% females) than in sample 1 (31% males, 8% females). To what extent might these differences be due to recent changes in substance use patterns?

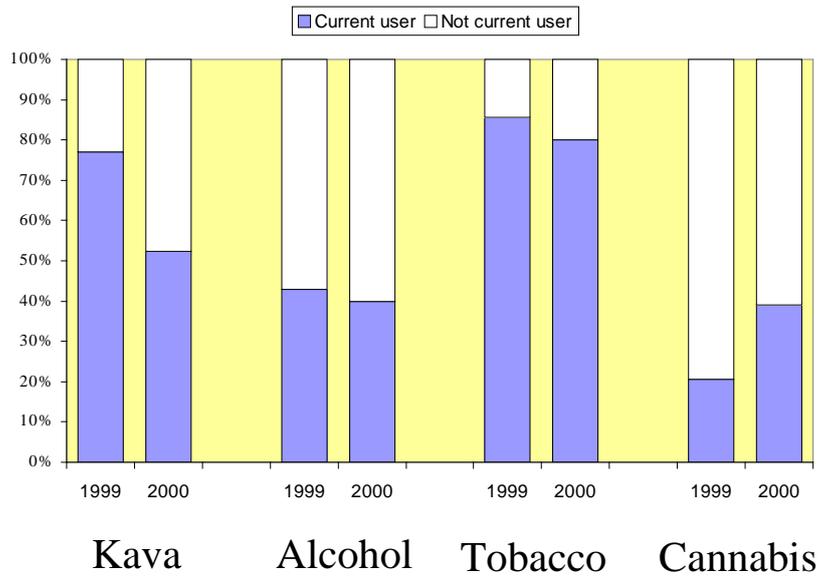
Possible Recent Changes

When data for current users from sample 2 in 2000 (n=101) are compared with the data from sample 1 for this same community (collected in 1999, n=65) we see the following results (Figure 4-3).

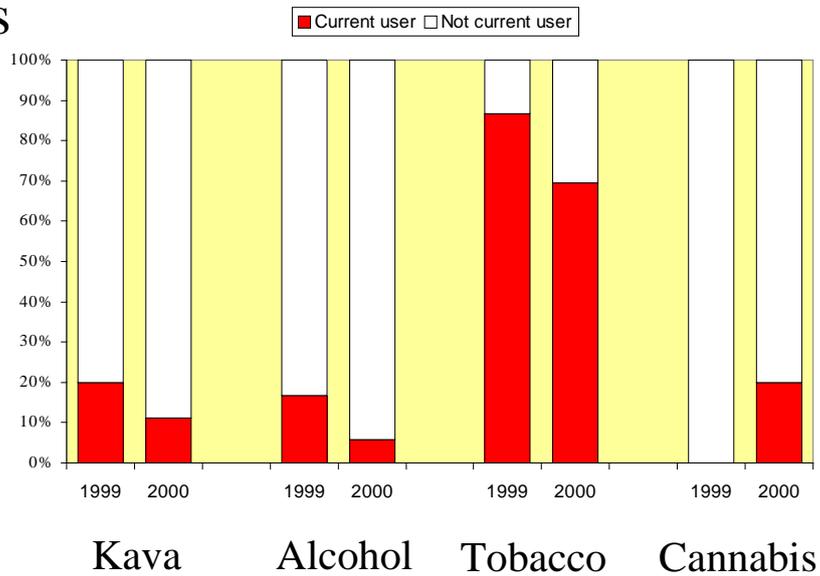
- a statistically significant decrease from 77% to 52% in kava use among males (P=0.015),
- an increased proportion of cannabis users in females from 0%-20% (P=0.013) along with a non-significant tendency for an increase in males from 21% to 39% (P=0.068) and,
- a non-significant tendency (P=0.096) for a decreased proportion of tobacco users among females from 87% to 69%.

Figure 4-3 Comparison of changes in proportions (%) of current substance users in 2 samples (1999 and 2000) in one community in the Miwatj Region

Males



Females



Among those who ever used kava, alcohol, tobacco and cannabis in this community, the median duration of cannabis use was four years and the median age at first use was 25 years (data not shown). The median age at first use for other substances was at least 4 years younger and the median duration of use 11 years longer than for cannabis (data not shown). While some had commenced using cannabis in recent years, around 38% (n=13) of current users in 2000 (sample 2) stated that they already had knowledge and experience of the drug outside of the community prior to 1996. Notwithstanding this, the older median age at first use and the median of four years duration of cannabis use suggests a comparatively new practice in this population and that it has been taken up by age groups other than the younger ones (see also Figure 4-2). The decline in kava use (Figure 4-3) was probably not directly related to the uptake of cannabis. Just two of the 21 individuals who ceased kava in 1998 took up cannabis in subsequent years (data not shown).

Past tobacco usage patterns and levels, may also have been quite different in this community. According to store management, six years previously stock holdings were five times greater than in 2000, tobacco sold in the community dropped dramatically in recent years and, this had also occurred in other community stores in the Miwatj Region. In interviews, smokers reported having recently quit (10%, n=7), reducing their consumption or changing to lower tar brands (33%, n=25).

Consumption Levels and Expenditure

Average number of sticks of tobacco reported smoked per day was 16. Supply data, however, showed that smokers probably consumed just 6 sticks per day (Table 4-2). Individuals may bring some tobacco to the community from the nearby town, supplementing the supply from the community store, the sole local supplier. However, clinic and store staff agreed this was unlikely to distort the estimates to such an extent. Tobacco use reported in Table 4-2, therefore, was based on community supply information.

Table 4-2 Estimated consumption and expenditure levels among current substance users in a sample in one community (sample 2, 2000)
(estimated using samples of 65 males and 36 females in a population of 150 males and 189 females)

| | Kava | Alcohol | Tobacco | Cannabis |
|--|----------|---------|----------------------|----------|
| Consumption levels | | | | |
| Amount estimated consumed in a month - sample | 20kg | 11,000g | 15,000 cigarettes | 50g |
| Estimated users in the community population | 114 | 111 | 254 | 146 |
| Estimated total amount consumed in a month - community population | 58kg | 35,000g | 50,000 cigarettes | 220g |
| Expenditure | | | | |
| Estimated total for community in a month | \$16,000 | \$5,000 | \$16,000 | \$6,000 |
| Estimated expenditure per user in a month | \$137 | \$48 | \$62 | \$42 |

Data Limitations

Estimating illicit or stigmatised substance use in epidemiological studies is challenging, particularly across ethical, cultural and language constraints as existed in this study. The methods used to assess exposure relied on consensus classification of knowledgeable Aboriginal health workers to overcome these challenges.²³ Limitations remain in the data that must be acknowledged.

Data from sample 1 are limited for purposes of comparison with other studies because they do not include information about frequency of exposure to substance use. With the retrospective techniques employed in this study it was not possible to collect this kind of data. Data collected in sample 2 were not based on a random sample and were perhaps biased because people with health problems may have been more likely to present to the clinic to participate in the opportunistic screening study. While the risks of this bias were minimised using a quota approach, the failure to achieve a random sample was an inevitable consequence of the ethical requirements of the study.

Similar qualifications about the data for duration of use and expenditure in sample 2 apply. Participants readily provided information about quantities used recently but found difficulty reporting former usage levels beyond the previous month. Quantities reported in Table 4-2, therefore, describe just one point in time and not overall average use or duration and frequency of exposure which are more conventional measures.³⁰ Self-reported consumption in Table 4-2 should be regarded as upper levels, since the impact of sharing on amounts used may have exaggerated the estimates.

Finally, the data in sample 2 are not strictly comparable to the data from sample 1 for the community since the definition of current use was different. In sample 2 current use meant 'used within the previous month' whereas in the data from sample 1 current use was not specifically defined with respect to the previous month. Accordingly the results of this comparison over time should be regarded as *prima facie* evidence only warranting further systematic investigation.

DISCUSSION

Notwithstanding difficulties in sample selection and differences in exposure measurement between samples, the data presented here provide a unique description of patterns and possible changes in substance use in a group of remote indigenous communities in the 'Top End' of the NT, Australia. Substance use patterns in the region have the following features.

Tobacco

Tobacco use among indigenous populations elsewhere is reported to be 56% to 58% (males) and 48% to 50% (females) with around half smoking more than 10 cigarettes a day.^{6,8} Our data suggest a greater but declining prevalence in the region and a lower average consumption level. The public health importance of recent possible changes in quantities of tobacco supplied and possible shifts in usage patterns along with a change to lower-tar brands in this population warrants further evaluation. Health workers in clinics throughout the region (including authors TG and MY) reported similar changes in their respective communities. Moreover, tobacco retailers in the major regional centre described a recent and sudden shift in demand among Aboriginal customers away from heavy brands to lower tar brands beginning in late 1999 to early 2000.

Alcohol

A prevalence of alcohol use (53% males, 12% females, Table1) is lower than reported in other indigenous populations.⁸ Consumption in a month in one community (sample 2) was around 318g (400ml) of pure alcohol per user (Table 4-2). Alcohol purchased annually in the Miwatj Region as a whole is around 9.6L per capita,²⁴ the lowest level for NT regions, equivalent to 800ml in a month. Annual per capita consumption of 18.5L³¹ by Aboriginal people (older than 14 years) in the 'Top End' is equivalent to 1500ml in a month, almost four times the amounts we found.

Kava

Earlier studies (1986-87) ³² reported a prevalence of kava use of 42% and more recently 56%²² and 66%³³ with a greater proportion of males (from 53% to 71%) than females (from 6% to 51%).^{32,34} The changes in kava use that appear to have occurred since 1999 parallel the drop in the estimated size of the informal kava trade in Arnhem Land from \$6-\$8 million in 1997-98, to \$5 million in 1999 to \$3.8 million in 2000.³⁵ The economic impact of kava use in remote communities, seldom quantified, has long been recognised as one of the important harms resulting from its abuse.^{25,36,37} Expenditure estimates (Table 4-2) require more precise determination, but it appears that in 2000, it cost an individual more to be a kava user than any other substance. And since kava, alcohol and tobacco use was a popular combination among current users (n=20 in the sample of 101 participants), the economic burden for a polydrug user was likely considerable.

Impacts on the wider community, however, need to be considered in light of the nature of the local kava trade. Local retailers purchase kava from agents in nearby regional centres at profit rates of perhaps 100% between agent and retailer.³⁵ With this pricing structure, the local retail price of kava to consumers (\$269/kg) was some eight times greater than Sydney wholesale prices. By contrast, the retail price of cannabis, also sold by local retailers, was two to three times known prices from middlemen in larger centres in the NT. Cash from kava or cannabis is different from alcohol and tobacco in that it is partially redistributed into the hands of local sellers. There is no such involvement in the trade in tobacco and alcohol. Both are an immediate drain on cash resources with no local on-selling or accumulation of cash. In other words, the c. \$21,000 a month expended on alcohol and tobacco (Table 4-2) leaves the local community economy immediately. But a proportion, perhaps as much as half, of the c. \$22,000 is likely redistributed or redirected for local community or family purposes from the trade in kava and cannabis.

Cannabis and Petrol Sniffing

Cannabis use was rarely reported in remote communities before 1991 although its use by Aboriginal people in urban areas and rural towns was well known.³⁸ In 1994, 22% (30% males and 15% females) of urban Aboriginal people were current users,⁸ a prevalence comparable to that in our data for 1999 (Table 4-1).

Health workers (including authors TG and MY) in all clinics visited in the Miwatj Region reported that in the last three to five years, cannabis became available for sale in their communities and that this had seldom been the case previously. This new feature is recognised by key informants in the Illicit Drug Reporting System in the NT¹³ and is a matter of some concern in communities. Cannabis has been used at some time by 44% of males and 35% of females in Australia,³⁹ and in the NT, 36.5% used cannabis within the previous 12 months.⁴⁰ And there exists a wider trend for lifetime use among increasingly younger cohorts.⁴¹ But our information from one community suggests that a comparatively broad prevalence of current use may have been achieved quickly, perhaps between 1999 and 2000, by the recent development of local trafficking in eastern Arnhem Land communities.

The practice of petrol-sniffing, while curtailed at the time of the survey in 2000, remained among a small resilient cohort in the community. A prevalence of a history of petrol sniffing of around 54% among young males has been found in other Arnhem Land communities.^{3,21} There occurred a resurgence of petrol sniffing in 2001-02 in a number of Miwatj Region communities while this paper was in preparation.

Conclusions

While we know from other studies that petrol sniffing declined over recent years in some localities,⁴² the effects of current and past petrol sniffing on emerging patterns of cannabis use, existing patterns of alcohol use and a broad prevalence of tobacco consumption require careful monitoring and interpretation to assist Aboriginal

communities to establish priorities for intervention. Controlled studies of health effects of any one substance in this population may be confounded by the effects of others.

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CHAPTER 5

CROSS-SECTIONAL STUDY

SUMMARY

INTRODUCTORY NOTES

Paper #4: in press

Clough AR, Jacups SP, Wang Z, Burns CB, Bailie RS, McDonald SP, et al. Health effects of kava use in an eastern Arnhem Land community [in press]. *Internal Medicine Journal*.

Paper #5: in press

Clough AR, Bailie RS, Currie BJ. Liver function test abnormalities in users of aqueous kava extracts [in press] *Journal of Toxicology; Clinical Toxicology*

Paper #6: submitted

Clough AR, Rowley K, O'Dea K Kava use, dyslipidaemia and biomarkers of dietary quality in Aboriginal people in Arnhem Land in the Northern Territory (NT), Australia [submitted] *European Journal of Clinical Nutrition*

Negotiating and establishing the community permissions and protocols to carry out the cross-sectional study reported in this chapter began at the end of 1998. Finally, in March 2000, the cross-sectional study was successfully conducted in accordance with appropriate procedures and protocols that were negotiated with the community concerned. The stigma attached to kava use and its illegal status were major discomforts for community people in this locality who remain divided even today about kava. In addition, contemporary perceptions of what is appropriate research in Aboriginal societies made for additional challenges to the successful conduct of this kind of health study involving clinical assessments. Studies of drug use tend to evoke equivocal responses from potential participants in most settings. Potentially

exacerbating this, kava was illegal when the study was carried out. A previous cross-sectional study of kava use was also conducted by researchers from the Menzies School of Health Research in a nearby Arnhem Land community and the results of that study directly informed policy approaches which led to kava restrictions in 1990 with communities strongly divided on the issue. Key Aboriginal people involved at that time remained angry that Government, informed by research, had intruded into what were regarded as local community matters.

To overcome these substantial difficulties, a memorandum of understanding was negotiated with the community (Appendix E). A main restriction was to work in the clinic and this meant that a random sample was not possible in this study. Participants were opportunistically recruited. Notwithstanding these difficulties a total of five papers have so far been prepared and submitted for publication from the data collected in this cross-sectional study. Three of these are reported in this chapter. Two papers describing the acute and chronic neurocognitive effects of kava have been prepared by other colleagues with input from this author (Appendix A).

The first paper presented here is a summary of outcome measures that were compared across groups of current and recent kava users and non-users in a sample of 101 participants. The second is a more detailed analysis of liver function changes that compared liver enzymes and associated characteristics in continuing kava users with those who had ceased kava and a group of non-users in 98 of the 101 participants for whom the last kava drinking occasion could be precisely determined. Finally, material summarizing some nutritional indicators compared across kava-using groups in the 98 participants is presented for discussion. Hypotheses for further analysis are presented.

The principal differences between kava users and non users is a tendency for weight loss and loss of body fat, lower lymphocyte levels, elevated liver enzymes indicative of an obstructive liver response and changed lipid profiles that resemble those found in anorexia nervosa. A preliminary analysis suggests that antioxidants are not reduced in kava users. All these changes appear to be reversible with abstinence from kava.

Health effects of kava use in an eastern Arnhem Land Aboriginal community

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ABSTRACT

Background: Heavy kava use in Aboriginal communities has been linked to various health effects, including anecdotes of sudden cardiac deaths.

Aims: To examine associations between kava use and potential health effects.

Methods: A cross-sectional study within a kava using East Arnhem Land Aboriginal community in tropical northern Australia. One hundred and one adults who were current, recent or non-users of kava were enrolled in March 2000. Main outcome measures were physical, anthropometric, biochemical, haematological, immunological and neurocognitive assessments.

Results: Kava users more frequently showed a characteristic dermatopathy ($p < 0.001$). They had increased levels of γ -glutamyl transferase and alkaline phosphatase ($p < 0.001$). Lymphocyte counts were significantly lower in kava users ($p < 0.001$). Fibrinogen, plasminogen activator inhibitor-1 and neurocognitive tests were not different between kava use categories. IgE and IgG antibodies were elevated across the whole group as were C-reactive protein and homocysteine.

Conclusions: Kava use was associated with dermatopathy, liver function abnormalities and decreased lymphocytes. If kava continues to be used by Aboriginal populations monitoring should focus on the health consequences of these findings, including a possible increase in serious infections. The interaction between kava, alcohol and other substances requires further study. While markers of cardiovascular risk are increased across the population, these were not higher in kava users, and this increase may be linked to the large infectious pathogen burden reflective of the socioeconomic disadvantage seen in many remote Aboriginal communities.

INTRODUCTION

Kava, the intoxicating drink prepared from crushed roots of the plant *Piper methysticum*, is widely used in south pacific countries on ceremonial occasions and in secular drinking.¹ Its effects on health became contentious when it was introduced to Arnhem Land Aboriginal communities in 1982.²⁻⁴ In late 1999 there were perhaps 1050 people using kava in Arnhem Land in eight community areas (A. Clough, unpublished data).

A survey on kava use in Arnhem Land conducted in 1987 suggested additional health burdens to Aboriginal populations. Kava use was associated with scaly skin and users were more likely to be underweight. Users also had decreased lymphocytes with increased γ -glutamyl transferase (GGT), high-density lipoprotein (HDL)-cholesterol levels and decreased total protein, albumin, urea and bilirubin.⁵

Recently it was suggested that kava use may be a risk factor for sudden cardiac deaths amongst young Aboriginal sportsmen in Arnhem Land populations, especially when coupled with heavy alcohol use.^{6,7} Possible mechanisms include abnormal coagulation with enhanced thrombosis, dehydration or arrhythmias. Other health risks for kava users include acute neurological effects⁸ and the infectious disease melioidosis, which may be greater amongst heavy users^{9,10} The aim of this study was to further investigate health effects of kava consumption in an Arnhem Land community in tropical northern Australia.

METHODS

A memorandum of understanding between the local Aboriginal community Council and Menzies School of Health Research guided the research. The Institutional Ethics Committee of the Menzies School of Health Research and Royal Darwin Hospital granted approval. All participants gave written informed consent. Study procedures were explained with the assistance of Aboriginal health workers. Clinicians in the study were blinded to kava-using status of participants.

Community surveillance estimated 75% of men and 25% of women were kava users. From a population of 150 men and 189 women, we were able to opportunistically recruit 101 consenting adults, a mix of males and females, approximately half were kava users. Exposure to kava use was assessed by self-report in interview compared with consensus classification by community health workers along with health clinic records using procedures already validated.¹¹ These were 38 current kava users (34 men, 4 women) who reported using kava at least once in the month prior to interview, 27 recent users (18 men, 9 women) who had had their last drink of kava prior to 'last month' and 36 people (13 men and 23 women) who reported they had never used kava. Information was also collected on other substance use including alcohol, tobacco, petrol sniffing and cannabis.

Subjects were examined for the presence of kava dermatopathy (a rash characteristic of heavy use), scabies, skin sores (pyoderma) and tinea. Skin sores and tinea were assessed and scored by counting the sites present,¹² with maximum scores of 18 for skin sores and 41 for tinea. Skin sores and tinea scores were categorised respectively into mild (0-2) and (0-10); moderate (3-7) and (11-20); and severe (7+) and (20+). Height and weight were measured and body mass index (BMI) calculated.¹³

Electrolytes, full blood count, urea, creatinine, liver function tests, lipids (non-fasting), serum folate, iron studies, homocysteine, fibrinogen, C-reactive protein (CRP) and plasminogen activator inhibitor-1 (PAI-1) were measured along with immunoglobulins and IgG subsets. Eosinophilia was defined as an eosinophil count of $>0.6 \times 10^9$ /L.

Serum osmolality was calculated.¹⁴ Urine was tested using Boehringer dipsticks. Samples 'positive' for blood were sent for microscopy after adding formalin. Numbers and percentages above and below the normal range were calculated for current kava users (Table 5-1).

Neuropsychological assessments utilised the touch-screen based Cambridge Neuropsychological Test Automated Battery (CANTAB) featuring minimum reliance on verbal skills.^{15,16} Data were recorded as the proportion of correct responses. Ocular-motor function was assessed with measures of latency and accuracy for prosaccades (towards target) and antisaccades (away from target) and an antisaccade error rate was calculated.

Statistical Methods

Data were analysed using *intercooled* Stata (Version 7, Stata Corporation, College Station, Texas). Variables were compared across the three groups using one way ANOVA or Chi-square (exact) tests. Positively skewed data were log-transformed before analysis.

RESULTS

A rash consistent with kava dermopathy was present in 45% of current kava users, 11% of recent users and 6% of non-users ($p < 0.001$) (Table 5-1). The two non-users with such a rash probably reflect incorrect clinical assessment rather than incorrect ascertainment of kava consumption. Scabies, skin sores and tinea were higher in current kava users but this was not statistically significant ($p = 0.52$), ($p = 0.11$) and ($p = 0.53$) respectively. Mean BMI was lower amongst kava users and a greater proportion of kava users (32%), compared with recent users (15%) and non-users (14%) had a BMI less than 18.5, however more of the non-users were female. When females were excluded there was still a trend for kava users to have lower BMI ($p = 0.062$).

There were no differences in neutrophils, monocytes and eosinophils across the categories of kava use. Of the group as a whole 25% had an eosinophilia. Lymphocyte numbers were significantly lower (Table 5-1) in current and recent kava users ($p < 0.001$), with 51% of users below the normal range.

Creatinine levels tended to be higher among kava users ($p = 0.06$) but there was no difference in calculated GFR (data not shown). The following parameters showed no association with kava use: urea, sodium, bicarbonate, chloride, inorganic phosphate, calcium, uric acid, serum folate, fibrinogen and PAI-1 (data not shown).

Cholesterol was significantly higher for current kava users with 28% over 5.5mmol/L compared with 11% for recent users and 11% for non-users. This remained significant when comparing only males. Interestingly both HDL and LDL were significantly higher in current kava users. Triglycerides were not significantly different between the groups.

Serum osmolality was not significantly different across the kava categories, although 17% of current kava users had values lower than normal. The presence of dipstick positive "blood" was confirmed as true haematuria by microscopy in only 1 of 24 cases.

Levels of GGT and alkaline phosphatase (ALP) were above the normal range in 61% and 50% of kava users respectively. When data were log transformed current users had significantly higher GGT and ALP than recent users and non-users (Table 5-1). However there were no differences between the groups in alanine transaminase (ALT), bilirubin, albumin or total protein.

IgE was elevated in every individual tested from the community and levels were significantly higher in current and recent kava users ($p=0.02$). Total IgG levels (median=18.1g/L) were also all above normal but were not different between the groups. Elevated IgG1 and IgG4 were responsible for the high levels (Table 5-1).

CRP and homocysteine were elevated in 36% and 16% overall, with no difference across the categories of kava use.

No significant differences were found for any of the ocular motor or neuropsychological assessments across the groups.

Kava use was associated with alcohol use ($p<0.001$), cannabis use ($p<0.001$) and a history of petrol sniffing ($p=0.035$). Both cannabis and alcohol use remained significant when comparing only males. There was a trend for kava users to be tobacco smokers ($p=0.076$).

Table 5-1: Kava use: comparison of mean, low and high values

| Outcome measure | Current User n=38 (f=4, m=34) | Recent-user n=27 (f=9, m=18) | Non-user n=36 (f=23, m=13) | P | Number(%) below lower level Current User | Number(%) above upper level Current User |
|--|--|---|---|----------|---|---|
| Physical assessments | | | | | | |
| Age mean(sd) | 35.2(9.3) | 37.6(11.8) | 36.5(13.5) | 0.72 | | |
| Kava Dermopathy(%) | 17(45%) | 3(11%) | 2(6%) | <0.001 | | |
| Scabies med(range) | 3(0-11) | 2(0-6) | 2.5(0-8) | | | |
| Tinea score [†] med(range) | 14.5(2-30) | 13(1-27) | 11(0-25) | 0.56 | | |
| Skin sores score [†] med(range) | 3(0-11) | 2(0-6) | 2.5(0-8) | 0.12 | | |
| Body mass index (kg/m ²)mean(sd) | 20.8(4.2) | 21.6(3.3) | 23.4(4.8) | 0.02 | 12(32%) | 3(8%) |
| Other substance use | | | | | | |
| Tobacco | 31(82%) | 21(78%) | 22(61%) | 0.07 | | |
| Alcohol | 17(45%) | 11(41%) | 0(0%) | <0.001 | | |
| Cannabis | 14(37%) | 13(48%) | 3(8%) | <0.001 | | |
| Petrol sniffing history | 14(37%) | 9(33%) | 4(11%) | 0.03 | | |

Table 5-1 (cont): Kava use: comparison of mean, low and high values

| Outcome measure | Current User n=38 (f=4, m=34) | Recent-user n=27 (f=9, m=18) | Non-user n=36 (f=23, m=13) | P | Number(%) below lower level Current User | Number(%) above upper level Current User |
|--|--|---|---|----------|---|---|
| Blood tests-parametric[†] | Mean(sd) | Mean(sd) | Mean(sd) | | | |
| Haemoglobin (g/L) M(132-170)F(115-155) | 148(13) | 143(15) | 138(15) | 0.02 | 1(3%) | 1(3%) |
| Haematocrit (PCV)(0.34-0.49) | 0.5(0.04) | 0.4(0.05) | 0.4(0.05) | 0.04 | 0 | 7(19%) |
| White cell count (x10 ⁹ /L)(4.0-11.0) | 7.0(2.8) | 7.3(2.4) | 8.4(2.1) | 0.05 | 3(8%) | 4(11%) |
| Lymphocytes (x10 ⁹ /L)(1.5-4.0) | 1.6(0.6) | 1.8(0.6) | 2.2(0.7) | <0.001 | 18(51%) | 0 |
| Neutrophils (x10 ⁹ /L)(1.8-7.5) | 4.6(2.3) | 4.5(1.7) | 4.9(1.8) | 0.60 | 3(8%) | 5(13%) |
| Platelets (x10 ⁹ /L)(150-400) | 246(70) | 242(55) | 285(79) | 0.02 | 1(3%) | 1(3%) |
| Creatinine (µmol/L) M(<120) F(<95) | 103(16) | 96.5(15) | 93(20) | 0.06 | | 4(11%) |
| Potassium (mmol/L)(3.4-5.5) | 4.0(0.3) | 3.8(0.2) | 3.8(0.3) | 0.04 | 1(3%) | 0 |
| Iron (µmol/L)M(12-31)F(11-27) | 13.6(6.4) | 13.8(5.5) | 10.6(4.9) | 0.04 | 17(47%) | 1(3%) |
| Osmolality (mmol/L)(280-300) | 282(6.4) | 285(5.3) | 285(5) | 0.08 | 6(17%) | 0 |
| Cholesterol total (mmol/L)(<5.5) | 5.1(0.9) | 4.6(0.8) | 4.2(0.7) | <0.001 | | 10(26%) |
| Low density lipoproteins (mmol/L)(<3.4) | 3.4(0.9) | 3.2(0.7) | 2.9(0.8) | 0.03 | | 17(57%) |
| Alanine transaminase (U/L)(<40) | 24(7) | 29(16) | 27(13) | 0.15 | | 0 |

Table 5-1 (cont): Kava use: comparison of mean, low and high values

| Outcome measure | Current User n=38 (f=4, m=34) | Recent-user n=27 (f=9, m=18) | Non-user n=36 (f=23, m=13) | P | Number(%) below lower level Current User | Number(%) above upper level Current User |
|---|--|---|---|----------|---|---|
| Blood tests –non-parametric[‡] | Median(range) | Median(range) | Median(range) | | | |
| C-reactive protein (mg/L)(<10) | 8(2-34) | 6(0-20) | 8(3-38) | 0.37 | | 14(37%) |
| γ-Glutamyl transferase (U/L)M(<60)F(<40) | 75(14-257) | 44(16-195) | 34(7-184) | <0.001 | | 23(61%) |
| Alkaline phosphatase (U/L)(35-135) | 133(86-212) | 113(77-166) | 109(61-240) | <0.001 | 0 | 18(47%) |
| Ferritin (ug/L)M(20-300)F(15-200) | 105(0-318) | 94(1-376) | 70(11-769) | 0.16 | 2(5%) | 2(5%) |
| Eosinophils ($\times 10^9/L$)(<0.6) | 0.29(0-1.4) | 0.27(1-1.8) | 0.27(0.1-2.3) | 0.24 | | 9(24%) |
| IgE total (kU/L)(<20) | 2439(1498-8464) | 2689(1595-3010) | 1786(364-9784) | 0.02 | | 28(100%) |
| IgG total (g/L)(6.1-13.0) | 18.1(14-27) | 19.5(14-27) | 20(16-30) | 0.54 | 0 | 33(100%) |
| IgG1 (g/L)(4.9-11.4) | 13(8-19) | 14(11-23) | 13(11-20) | 0.27 | 0 | 22(79%) |
| IgG2 (g/L)(1.5-6.4) | 2.1(0.9-3.8) | 1.7(0.9-3.3) | 2.3(0.3-4.1) | 0.65 | 6(22%) | 0 |
| IgG3 (g/L)(0.2-1.1) | 0.3(0.2-0.6) | 0.2(0.2-0.9) | 0.3(0.1-1.1) | 0.88 | 4(20%) | 0 |
| IgG4 (g/L)(0.08-1.4) | 1.3(0.1-6.5) | 1.2(0.1-1.6) | 1.1(0.4-4.7) | 0.44 | 1(3%) | 11(42%) |
| Homocysteine ($\mu\text{mol/L}$)(6.0-15.0) | 10(5-95) | 10(4-32) | 11(4-42) | 0.50 | 1(3%) | 8(22%) |
| High density lipoproteins (mmol/L)M(>0.9)F(>1.1) | 1(0.6-2.1) | 1(0.6-1.8) | 0.9(0.5-1.4) | 0.01 | 18(47%) | |
| Triglycerides (mmol/L)(<2.0) | 2.4(0.9-6) | 1.9(1-4.9) | 2.3(0.6-5.4) | 0.30 | | 25(66%) |

[†]See methods[‡]Not all parameters available for all subjects

DISCUSSION

The study community is nominally 'dry' (alcohol is prohibited), although retail outlets are located nearby so illicit drinking occurs. There is concern that poly-substance abuse is increasing in many Aboriginal communities. In our study, 82% of current kava users smoked tobacco, 45% drank alcohol, 37% used cannabis and 37% had a history of sniffing petrol. In the Mathews et al study⁵ alcohol was a confounding factor, which may have contributed to the abnormal liver function tests. However in our study there was a lack of association between alcohol use and GGT and ALP (data not shown). Furthermore, several people with abnormal liver function were heavy kava consumers but did not drink alcohol.

While there is currently no evidence for long term liver damage in regular kava users, this requires assessment over longer periods, especially in heavy users. Furthermore, the nature of the abnormal liver function tests we observed remains to be elucidated. Elevated GGT and ALP suggests the possibility of an obstructive rather than an inflammatory pattern of response. In Aboriginal people who cease kava drinking, abnormal liver function tests return to normal after one to two months of abstinence from kava use (Currie, B. unpublished data).¹⁷ Importantly, the absence of an elevated ALT suggests that in contrast with alcohol, acute inflammation may not be occurring with kava use. However, recent case reports of fulminant liver failure associated with use of commercially available kava-based anxiety treatments in Europe¹⁸ have resulted in kava-based herbal products being withdrawn from sale in several countries. This supports the need for further research into the effects of kava on the liver.

The more frequent occurrence of abnormally low lymphocyte levels coupled with higher IgE levels in current and recent kava users are suggestive of an immune system response. Elevated IgE and IgG levels in the whole population are likely to reflect recurrent and chronic bacterial and parasitic infections. The increases in IgG1 and IgG4 are consistent with combined bacterial and parasitic burdens, and elevated IgE levels and eosinophil counts in the whole group are likely to reflect the burden of intestinal parasites seen in remote Aboriginal communities.¹⁹ It is possible that the association of

heavy kava use with melioidosis^{9,10} may reflect increased exposure to the wet season environmental pathogen *Burkholderia pseudomallei* during prolonged sessions of sitting in a kava circle consuming kava. Increased parasite exposure in kava drinkers could also explain higher IgE levels if they reflect a larger burden of geohelminths such as *Ancylostoma duodenale* and *Strongyloides stercoralis*. However the consistently lower lymphocyte counts in kava drinkers in this and the previous study⁵ warrant concern about a kava related immunological predisposition to certain infections such as melioidosis, for which diabetes and alcohol excess are known major risk factors.^{8,10}

Functional equivalence of neurocognitive assessments across the groups suggests little if any deleterious effect on the central nervous system. This is in contrast to abnormalities found in petrol sniffers¹⁶ and heavy alcohol users.²⁰

Fibrinogen and PAI-1 are considered important markers of potential thrombotic predisposition. There was no association of these parameters with kava usage. In addition, lipid profile was not clearly suggestive of increased atheroma risk, with HDL elevated as well as cholesterol and LDL. The generally elevated levels of CRP across the population studied suggest a chronic systemic inflammatory response possibly reflecting increased exposure to various infectious pathogens. CRP is now recognised as an important marker of cardiovascular risk and reflects possible pathogenetic processes, which may relate to specific organisms or to general pathogen burdens.^{21,22} However, the CRP was not related to kava use and neither were homocysteine levels, another cardiovascular risk marker, which were also generally elevated.

In conclusion, some health effects of kava use such as abnormal liver function, kava dermatopathy and decreased lymphocytes were observed in this study. Monitoring these parameters will be useful as changes occur in licensing and distribution of kava. Further research is required to address concerns about possible associations of heavy kava use in Arnhem Land with various infectious diseases, especially melioidosis. Some of our results were confounded by concomitant alcohol use and the effects of using kava in combination with other substances requires further analysis. The general increase in IgE and IgG levels and CRP reflect the burden of infectious pathogens associated with the continuing socioeconomic disadvantage of those living in remote Aboriginal

communities. Elevated CRP is increasingly recognised as a marker of risk for cardiovascular disease and the generally high levels of CRP and homocysteine across all groups in this Aboriginal community are of great concern.

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Liver function test abnormalities in users of aqueous kava extracts.

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OUTLINE

Introduction: This paper presents evidence that liver function changes in users of aqueous kava extracts appear to be reversible. Data from one Arnhem Land community (Northern Territory, NT, Australia) with 340 indigenous people aged over 15 years in 2000 are used.

Methods: Cross-sectional study with 98 participants; 36 had never used kava. Among 62 kava users, 23 had discontinued kava at least one year before the study. Continuing users had not used kava for 1-2 months (n=10) or 1-2 weeks previously (n=15). Some (n=14) had used kava within the previous 24 hours. Liver function tests were compared across these groups taking into account differences due to age, sex, alcohol and other substance use.

Results: Average quantity of kava powder consumed was 118g/week and median duration of use was 12 years (range, 1-18 years). Kava usage levels were less than half those found in previous studies. More recent kava use was independently associated with higher levels of liver enzymes γ -glutamyl transferase (GGT) ($P<0.001$) and alkaline phosphatase (ALP) ($P<0.001$), but not with alanine transaminase (ALT) or bilirubin which were not elevated. In those who were not heavy alcohol users, only those who used kava within the previous 24 hours showed GGT levels higher than non-users ($P<0.001$) while higher ALP levels occurred only in those who last used kava 1-2 weeks ($P=0.015$) and 24 hours previously ($P=0.005$).

Discussion: Liver function changes in users of aqueous kava extracts at these moderate levels of consumption appear to be reversible and begin to return to baseline after 1-2 weeks abstinence from kava. No evidence for irreversible liver damage has been found.

INTRODUCTION

Serious irreversible liver damage has been reported from several countries since the late 1990s in people using herbal products that contain ethanol or acetone extracts of kava lactones.¹⁻³ Consequently, manufactured kava-based products have become subject to medical alerts or bans on their sale in Europe,⁴ north America^{5,6} and Australasia.⁷

In the Pacific islands region, where the kava plant (*Piper methysticum* Forst. f.) was domesticated, people have used it for centuries in a manner consistent with local cultural practices primarily in the form of aqueous emulsions of the crushed fresh or dried roots or lower stems.⁸⁻¹² Long-term observers of kava used in this customary manner in the Pacific stress that no reports of permanent liver injury have emerged.^{8,13} Indigenous Australians in Arnhem Land (Northern Territory, NT) acquired the practice of kava drinking from Fijians and Tongans and so have consumed kava powder mixed with water since 1982 when it was first imported for local use.¹⁴⁻¹⁶ Although there remains ongoing concern about kava's health effects,¹⁷⁻²¹ no clinical record or research has reported irreversible liver damage associated with kava use in this population either.

However, there is some evidence to suggest that liver function changes in users of aqueous kava extracts do occur. A recent (March 2000) survey in an Arnhem Land community found γ -glutamyl transferase (GGT) and alkaline phosphatase (ALP) levels above the reference range in 61% and 50% of kava users respectively. An earlier (1987) survey conducted in another Arnhem Land community reported that 'heavy' (310g/week of kava powder) and 'very heavy' (440g/week) kava users showed decreased levels of total protein, albumin and bilirubin and increased GGT when compared with 'occasional' users (100g/week) and non-users.²¹ The researchers concluded that the apparent hepatic toxicity of kava was possibly greater than alcohol, although the relative effects of alcohol and other substance use were not clarified.^{8,22} However, ongoing clinical observations from the 1990s in Arnhem Land indicate that abnormal liver function tests return to normal after 1-2 months abstinence from kava

with no evidence for long-term effects²³ while earlier limited evidence suggested that GGT became normal eight months after discontinuing kava.²⁴

This paper focuses on liver function changes in users of aqueous kava extracts, their temporal relationships with kava consumption and their possible improvement after kava is discontinued.

METHODS

Study Participants

The survey from which the data reported here are drawn investigated a wide range of possible health effects of kava.²³ The study compared a group of ‘current’ users who had used kava during the preceding month with a group of ‘recent’ users who had not used it for at least one month and these were compared with a group who had never used kava.²³ Among the ‘recent’ users, 23 people had, in fact, discontinued kava at least one year before they were assessed, and this provided the opportunity to compare liver function tests with continuing kava users who had last consumed kava 1-2 months (n=10) or 1-2 weeks (n=15) before the study. Finally, another group of continuing users had used kava during the previous 24 hours (n=14). It was possible to compare these kava using groups with a control group who had never used kava (n=36).

In the community studied, from 52%-77% of men and 11%-20% of women were using kava.²⁵ Procedures for the opportunistic selection of participants have already been described.²³ Sixty-five people (52 males, 13 females) had a history of kava use and 36 (13 males, 23 females) had never used it. For three of the 65 kava drinkers it was not possible to determine the last kava use occasion so these were excluded leaving 62 kava users in a sample of 98 participants.

Setting

The community is located in central-east Arnhem Land in the ‘Top End’ of the NT (Australia), 530km east of Darwin and 120km west of the nearest regional centre. Kava has been used continuously in some Arnhem Land localities,¹⁶ and in this community,^{23,25} from 1982. Alcohol has been available since the early 1970s from outlets in the regional centre.²⁵ Kava use declined from 1998 when it became illegal²⁵ and a recent rise in cannabis use in Arnhem Land has become evident.^{25,26} A

background of petrol sniffing is common since petrol sniffing was widespread throughout the region during the late 1980s and early 1990s.^{25,27} This pattern of substance misuse posed considerable challenges to identifying independent effects of kava.²⁵

Assessing Exposure to Kava Use

All participants were interviewed about their substance use behaviours and categorised according to the time since they last used kava (never used, more than one year ago, 1-2 months ago, 1-2 weeks ago, within the past 24 hours). Kava powder was available in the community in 75g bags. Quantities consumed per week were estimated from the number of 75g bags reported used. Participants were also asked when in their lives they commenced using kava and for how long they had used it. Pictorial representations were used in interviews conducted in a clinic setting with local health workers assisting. Knowledgeable senior Aboriginal health workers, who are also community members, confirmed self-reported kava use by consensus classification.^{21,28} Chart review using community health clinic files assisted to confirm health worker consensus.

Heavy, episodic alcohol drinking is well known in Indigenous Australian populations.^{19,29,30} In this study, health workers reported 31 out of 47 alcohol users in the sample known to drink in this way including 29 males and 2 females. However, average alcohol consumption reported was found to be equivalent to just 318g/month of pure alcohol,²⁵ with a maximum of 576g/month. This is from one-quarter to one-third of the average quantity consumed per capita in indigenous communities across the 'Top End' of the NT²⁵ and somewhat less than the average alcohol consumption considered to be harmful in Australia, which is 40g/day (males) or 20g/day (females).³¹ Heavy users within this group of apparently moderate alcohol users, were therefore described as those who reported using >318g of pure alcohol per month, who were known to health workers as heavy users and who had used alcohol within the previous year. The heavy users so defined had used alcohol for from 2-32 years in their lifetime

and all were continuing users. One man had used alcohol for 18 years, was known as a past heavy user, but had ceased drinking five years before the study. He was not included in the heavy using group. The others who were not classified as heavy users (n=15) had commenced alcohol use recently, within the preceding year.

Tobacco smokers who reported using 25 cigarettes/day (one pack) or more (Table 5-2) were classified by health workers as heavy users. Cannabis was periodically available²⁵ and consumed in hand-made 'bucket bongs' using 'cones' containing ca. 4mg of THC (unpublished data). Those who used >5 cones/week were described by health workers as heavy cannabis users (Table 5-2). There were no active petrol sniffers. A history of petrol sniffing was exclusive to males and 42% said they had sniffed petrol (Table 5-2). In Arnhem Land there are high rates of hepatitis-B infection.²¹ However, community screening data were not available for this study. Inspection of clinic charts did not reveal evidence of chronic liver disease in any of the participants.

Blood tests were performed measuring alanine transaminase (ALT), GGT, ALP, total protein, albumin and bilirubin. All blood tests were performed by a commercial laboratory that provides pathology services to Royal Darwin Hospital and community health clinics.

Data Analysis

Significance levels for trends across groups were calculated from logistic regression models for dichotomous outcomes or ranked qualitative traits. For quantitative traits significance levels for linear trends after one-way analyses of variance were calculated using the method described by Altman (1991).³² Backward step-wise regressions, using the 95%CI criterion for removing variables, were performed with each outcome measure as the dependent variable and with measures of exposure to kava use, age and other substance use as independent variables. Positively skewed data were log-transformed before analysis. Analyses were performed using Stata 7.0.³³

Ethics

A memorandum of understanding between the local Aboriginal community Council and Menzies School of Health Research guided the research.²³ Ethical approval was provided by the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research which has an Aboriginal subcommittee and which works to guidelines of the National Health and Medical Research Council of Australia. All participants gave written informed consent. Study procedures were explained with the assistance of Aboriginal health workers.

RESULTS

Time Since Last Kava Use, Other Substance Use and Associated Characteristics

Males were more likely to be kava users than females (age adjusted OR=6.8, 2.7-17.2, $P<0.001$) and to have used it within the previous 1-2 months (age adjusted OR=15.5, 4.5-53.7, $P<0.001$). Quantity of kava powder consumed averaged 118g/week and ranged from <40g/week to >195g/week. This is less than 368g/week reported earlier in other Arnhem Land communities.^{16,21} Median duration of kava use was 12 years (range 1-18 years). One-quarter of kava users had used kava continuously for 16 years or more (data not shown). Among the kava users, time since last kava use and quantity consumed were not associated (likelihood ratio $\chi^2=0.66$, $P=0.882$). However, duration of kava use was longer in more recent kava users ($|t|=2.4$, $df=58$, $P=0.009$, one-sided).

Table 5-2 summarises kava and other substance use within the community. Kava users, when compared with non-users and adjusted for age, were more likely to also use alcohol (OR=19.1, 5.7-63.9, $P<0.001$), cannabis (OR=14.1, 3.7-53.9, $P<0.001$), tobacco (OR=3.0, 1.0-8.0, $P=0.042$) and were more likely to have sniffed petrol (OR=5.5, 1.6-19.0, $P=0.003$). In males, alcohol and kava use remained strongly associated (OR=8.8, 2.2-34.5, $P=0.001$) but not in females (OR=0.9, 0.8-1.1, $P=0.162$). Heavy alcohol users were also more likely to use kava as well (OR=32.1, 4.1-250.2, $P=<0.001$) compared with those not classified as heavy users.

Table 5-2: Kava use and other substance use in an indigenous community in Arnhem Land (NT, Australia)

| Outcome measure | Never used kava | Kava user: time since last kava use | | | | P |
|--------------------------------|----------------------|-------------------------------------|----------------------------------|----------------------------------|---------------------------------|--------|
| | n=36 (m=13, f=23) | >1 year n=23 (m=14, f=9) | 1-2 months n=10 (m=9, f=1) | 1-2 weeks n=15 (m=14, f=1) | <=24 hrs n=14 (m=12, f=2) | |
| Tobacco use | 26(72%) | 20(87%) | 10(100%) | 12(80%) | 13(93%) | 0.282 |
| None | 10 | 3 | 0 | 3 | 1 | 0.647 |
| <25 cigarettes/day | 4 | 3 | 1 | 2 | 0 | |
| >=25 cigarettes/day | 22 | 17 | 9 | 10 | 13 | |
| Years used mean(sd) | 18(12) | 18(11) | 14(7) | 17(11) | 21(7) | 0.140 |
| Alcohol use | 4(11%) | 12(52%) | 9(90%) | 13(87%) | 9(64%) | <0.001 |
| None | 31 | 11 | 1 | 2 | 5 | <0.001 |
| <=318g pure alcohol/month | 2 | 8 | 3 | 5 | 5 | |
| >318g pure alcohol/month | 1 | 4 | 5 | 6 | 2 | |
| Years used median(range) | 0(0-3) | 1(0-32) | 8(0-20) | 13(0-30) | 1(0-32) | 0.001 |
| Heavy alcohol use | 1(3%) | 10(43%) | 5(50%) | 10(67%) | 5(36%) | <0.001 |
| Cannabis use | 5(15%) | 12(55%) | 8(89%) | 6(43%) | 6(43%) | <0.001 |
| None | 29 | 10 | 1 | 8 | 8 | 0.007 |
| <=20mg THC/week | 5 | 9 | 5 | 5 | 4 | |
| >20mg THC/week | 0 | 3 | 3 | 1 | 2 | |
| Years used median(range) | 0(0-4) | 1.5(0-20) | 1(0-14) | 0(0-16) | 0(0-20) | 0.373 |
| Petrol sniffing history | 4(11%) | 8(35%) | 4(40%) | 5(33%) | 5(36%) | 0.118 |

Abnormally High Values of GGT and ALP and Time Since Last Kava Use

Almost half (48%) of the kava users showed GGT above a normal reference range (OR=2.6, 1.0-6.5, P=0.034), with 37% having abnormally elevated ALP (OR=3.4, 1.2-10.1, P=0.017) (data not shown). There was no association between duration of kava use and abnormally elevated liver enzymes. However, quantity consumed per week *was* associated with an abnormally elevated ALP (likelihood ratio test, $\chi^2=9.33$, P=0.009). In those who were not heavy alcohol users, when adjusted for age, the quantity consumed per week and the duration of kava use, the tendencies for an association between time since last kava use and abnormally high GGT (likelihood ratio test, $\chi^2=5.0$, P=0.291) and ALP (likelihood ratio test, $\chi^2=3.61$, P=0.461) were not significant.

Liver Function Tests: Associations with Time Since Last Kava Use

In a univariate analysis there was a significant association between time since last kava use and observed levels of both GGT and ALP but not ALT, albumin, bilirubin or total protein (Table 5-3). Those with more recent exposure to kava use showed higher GGT and ALP. One-third of kava users had elevated total protein, with values generally elevated across groups but not associated with time since last kava use (Table 5-3).

The tendency for higher GGT with more recent kava use remained statistically significant (P<0.001) in multiple regression including independent variables; age, tobacco use, cannabis use and a history of petrol sniffing. In those who were not heavy alcohol users, the association remained significant (P<0.001). In heavy alcohol users the association was weaker (P=0.046) although still statistically significant. When analysing the separate groups, only those who were not heavy alcohol users and who had used kava within the previous 24 hours showed GGT higher than non-users ($|t|=4.0$, df=35,

Table 5-3: Kava use and liver function tests: comparison of values in an indigenous community in Arnhem Land (NT, Australia)

| Outcome measure | Never used kava | Kava user: time since last kava use | | | | P |
|---|----------------------|-------------------------------------|----------------------------------|----------------------------------|---------------------------------|--------|
| | n=36 (m=13, f=23) | >1 year n=23 (m=14, f=9) | 1-2 months n=10 (m=9, f=1) | 1-2 weeks n=15 (m=14, f=1) | <=24 hrs n=14 (m=12, f=2) | |
| Blood tests | | | | | | |
| Bilirubin (umol/L) (<20) median(range) | 8(5-14) | 8(6-16) | 8.5(6-12) | 8(7-12) | 8(6-11) | 0.662 |
| Total protein(g/L)(62-80) median(range) | 80(3.9) | 80(4.8) | 81(3.0) | 80(5.2) | 81(5.2) | 0.534 |
| Albumin (g/L) (35-50) mean(sd) | 44(2.0) | 45(2.4) | 46(2.1) | 45(1.6) | 45(2.4) | 0.642 |
| Alanine transaminase(U/L) (<40) median(range) | 24(11-71) | 24(9-81) | 24.5(16-30) | 21(13-38) | 25.5(14-37) | 0.437 |
| γ-Glutamyl transferase (U/L)M(<60)F(<40) median(range) | 34(7-184) | 29(16-195) | 49(14-165) | 88(23-178) | 78(44-257) | <0.001 |
| Alkaline phosphatase(U/L)(35-135) median(range) | 109(61-240) | 105(77-152) | 130(86-163) | 123(99-212) | 165(102-204) | <0.001 |

$P < 0.001$, one-sided) (see Figure 5-1). Data were not sufficient for this comparison in heavy alcohol users.

The association between ALP and more recent kava use also remained statistically significant in multiple regression ($P < 0.001$) with the association remaining statistically significant in heavy alcohol users ($P < 0.001$) as well as in those who were not heavy alcohol users ($P < 0.001$). Those who were not heavy alcohol users and who last used kava 1-2 weeks prior to the study ($|t| = 2.3$, $df = 35$, $P = 0.015$, one-sided) and those who used it within the preceding 24 hours ($|t| = 2.7$, $df = 39$, $P = 0.005$, one-sided) had ALP levels significantly greater than non-users (see Figure 5-2).

Conversely, amongst those who were not heavy alcohol users, participants who had never used kava did not have statistically significant differences in ALP levels compared with those who had abstained from kava for 1-2 months ($|t| = 0.7$, $df = 35$, $P = 0.242$, one-sided), and they also did not have statistically significant differences in GGT levels compared with those who had abstained for 1-2 weeks ($|t| = 1.3$, $df = 35$, $P = 0.096$, one-sided).

Liver Function Tests: Associations with Other Measures of Kava Use

The comparative effects of different measures of exposure to kava use on levels of GGT and ALP in those who were not heavy alcohol users were assessed using multiple regression. Using a backward stepwise approach with ALP as the dependent variable, time since last kava use remained statistically significant ($P = 0.001$) while duration of kava use and quantity consumed per week were not significant. A similar result was found with GGT as the dependent variable ($P < 0.001$). Time since last kava use was therefore a key variable accounting for variation in observed levels of GGT and ALP.

Figure 5-1.

Increase in γ -glutamyl transferase (GGT) with more recent kava use in those who were not heavy alcohol users in an indigenous community in Arnhem Land (NT, Australia) and comparisons with a normal reference range (broken line).

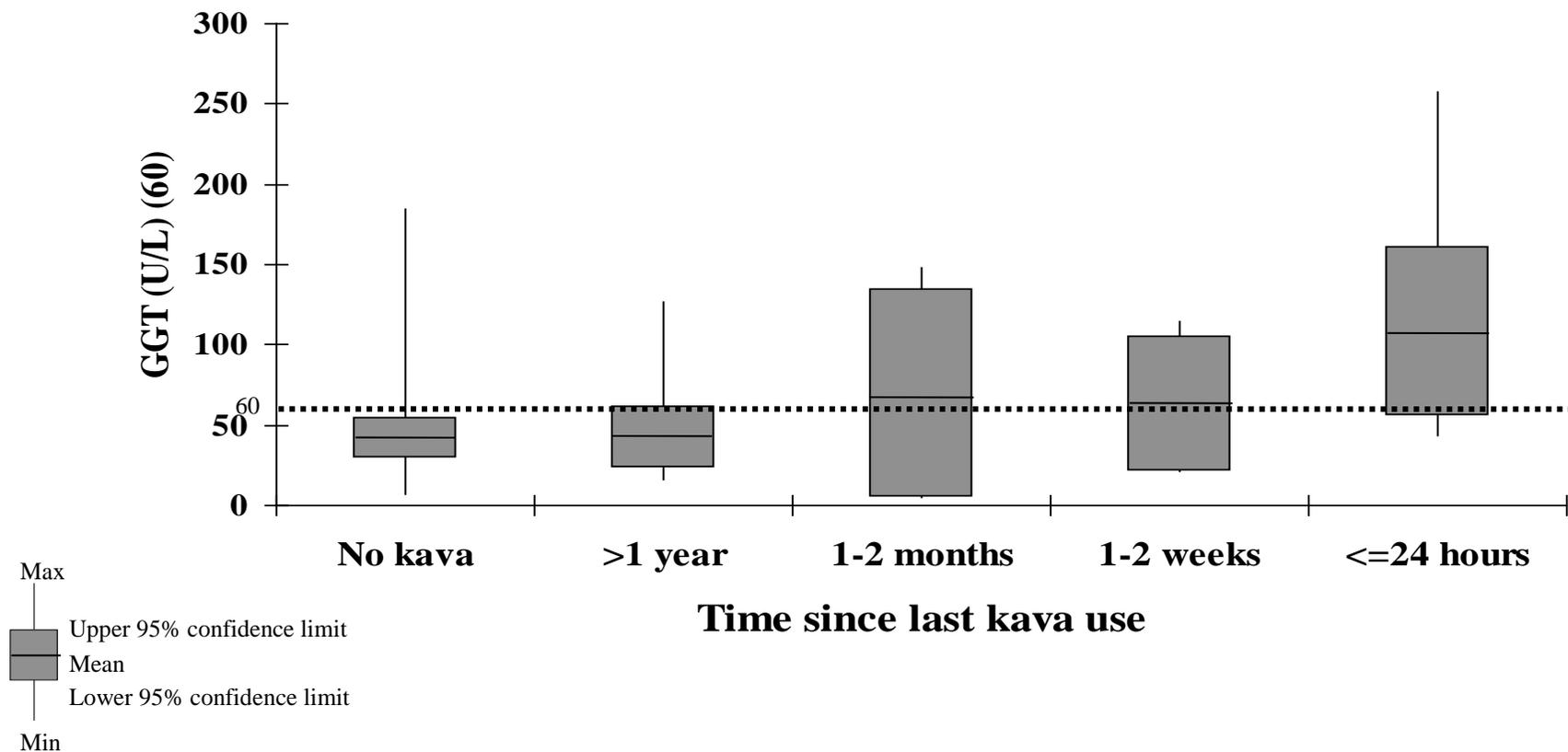
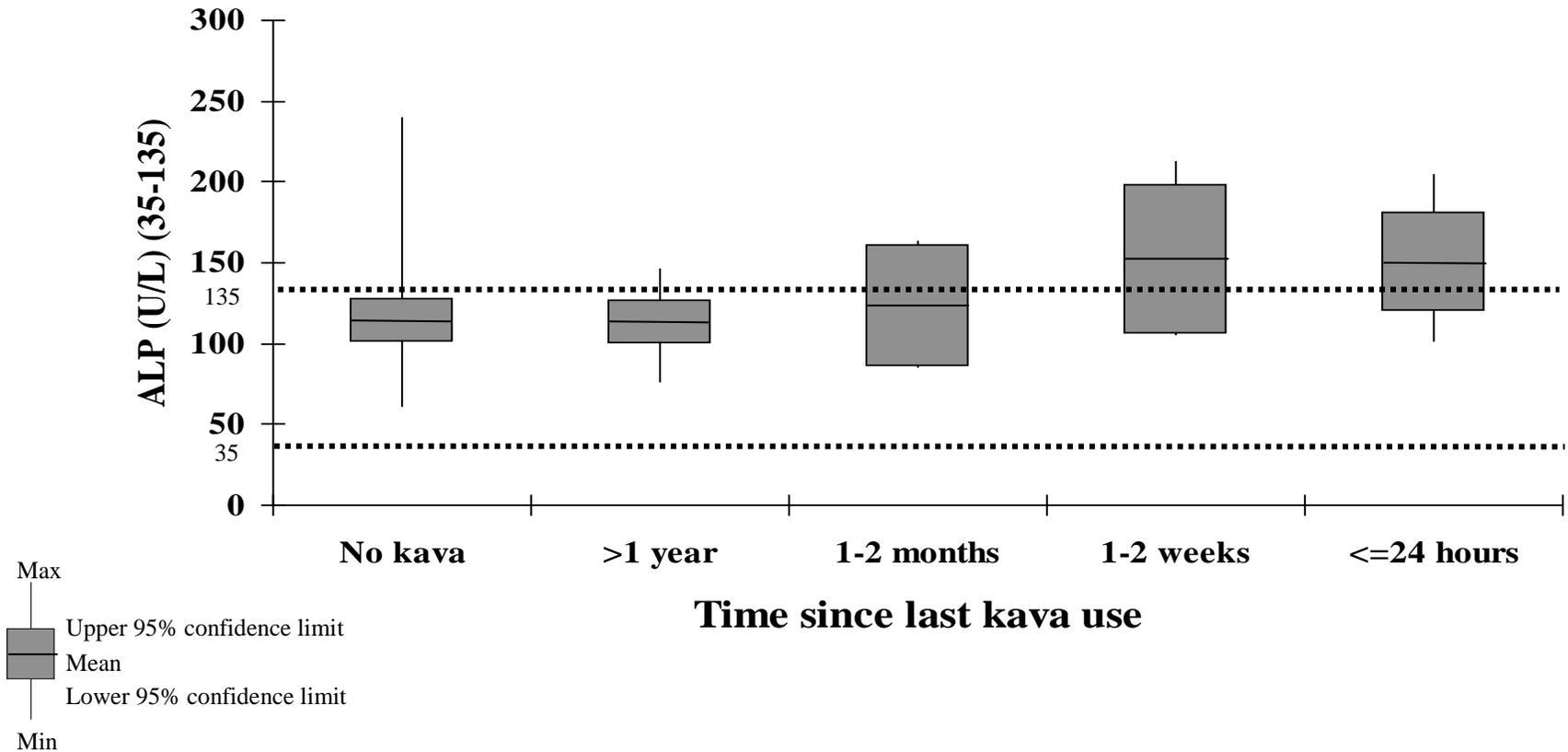


Figure 5-2.

Increase in alkaline phosphatase (ALP) with more recent kava use in those who were not heavy alcohol users in an indigenous community in Arnhem Land (NT, Australia) and comparisons with a normal reference range (broken lines).



Liver Function Tests: Associations with Other Substance Use

In the univariate analysis, there were no associations between measures of exposure to cannabis use and any of the outcome measures. While alcohol users as a whole had a higher GGT ($|t|=2.4$, $df=93$, $P=0.010$, one-sided), they did not have higher ALP ($|t|=2.2$, $df=93$, $P=0.410$, one-sided). Similarly, heavy alcohol users had higher GGT ($|t|=1.9$, $df=93$, $P=0.031$, one-sided) but not ALP ($|t|=0.6$, $df=93$, $P=0.271$, one-sided). Regression of outcome measures with number of years of alcohol use as an independent variable showed no associations except for a trend for increased ALP ($r=0.27$, $df=46$, $P=0.067$). Since there was no strong effect of heavy alcohol use on the outcome measures it seems unlikely that it was an important confounder of the effects of kava use.

DISCUSSION

These results confirm limited previous data showing that aqueous kava extracts can cause elevation of the liver enzymes GGT and ALP. Importantly however these liver function changes appear reversible,²³ with improvements evident from 1-2 weeks of abstinence from kava use at the moderate usage levels reported in this study. The majority of kava users had ALP and GGT levels return to normal levels after 1-2 months and 1 year of abstinence, respectively. These effects occur independently of effects of alcohol or other substance use. Most importantly, the use of aqueous kava extracts in this study was not associated with elevation of ALT. This is in contrast to the case reports of hepatic toxicity with fulminant hepatitis associated with herbal kava products.^{3,6}

The small sample size and lack of a random sample were unavoidable weaknesses of the study. Improved measures of exposure to kava use and time since last kava use in follow-up studies will assist to quantify more precisely the temporal relationships between development of liver function changes in kava users and their improvement when kava is discontinued.²⁸ One advantage of the present study was that possible confounding effects of alcohol and other substance use were quantified and controlled for whereas these were not clearly delineated in an earlier (1987) study.^{8,21,22,23}

Would these same results be observed at higher average levels of kava consumption and could the amount of kava consumed become a more important factor for irreversible liver injury at some critical level? Kava was used more heavily by Indigenous Australians in Arnhem Land in the recent past, but mortality data available for that time do not give cause for concern about death from liver damage in kava users. For the period 1985-1997, the period of heaviest kava use,³⁴ the Australian Bureau of Statistics (ABS) reported 9 deaths (5 males and 4 females over 15 years of age) in Indigenous people in eastern Arnhem Land with chronic liver disease as the primary cause (ICD9-code-571). When directly standardised to the 1991 Australian resident population, these deaths represent an annual rate (per 100000 population) of 18 (males) and 12 (females). While numbers of deaths are too small for meaningful

comparisons, rates are within ranges of age-adjusted death rates (per 100000 population) for chronic liver disease in indigenous people in the rest of the NT (5-91 in males, 6-39 in females) for a similar period (1982-1995).³⁵ Furthermore, close clinical surveillance of kava use within Arnhem Land, estimated by us as 30000 person-years of moderate to heavy kava consumption (unpublished data), has not documented any cases of severe hepatic toxicity which could be possibly attributed to kava.

It seems likely that the abnormal ALP and GGT seen with consumption of aqueous kava extracts reflects a different process to the hepatic toxicity documented from herbal products.^{3,6} It is possible that the ethanol or acetone extraction of kava lactones used in herbal products results in potentially hepatotoxic compounds not present in traditional aqueous extracts. It is also possible that genetic differences in liver metabolism between population groups may be important in determining whether hepatic toxicity occurs with various kava products.

In conclusion, the data confirm previous research and clinical observations that liver function test changes *do* occur in those who use kava in the form of aqueous extracts. However, the changes appear to only be in GGT and ALP and begin to return to normal after 1-2 weeks abstinence. There is currently no evidence for long-term liver damage from use of “traditional” preparations, even when people have used kava for 12 years or more. The reversibility of liver function changes outlined in this paper, together with the lack of reports of long-term liver damage in both Indigenous Australian and Pacific island populations are compelling. However, given consistent evidence for liver function changes in Arnhem Land, and since it is uncertain if these effects are reversible at higher levels of consumption, the possibility for long-term liver damage cannot be ruled out. Accordingly, until research further elucidates these issues, the promotion of moderate kava use together with clinical surveillance is required.

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Kava use, dyslipidaemia and biomarkers of dietary quality in Aboriginal people in Arnhem Land in the Northern Territory (NT), Australia

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ABSTRACT

Heavy kava use has been associated with sudden cardiac death in Indigenous Australians in Arnhem Land (Northern Territory, Australia) where poor diets and a high incidence of premature coronary heart disease are known. Heavy kava users may suffer additional risk if further malnourished.

Among 98 people (62 males, 36 females) in one community, 36 never used kava, 26 were past users, and 36 were continuing users. Across kava-using groups skinfold thicknesses, body mass index and body fat decreased. Total and LDL cholesterol were elevated in kava users compared to both former users and never users. HDL cholesterol was higher in current users versus never users. Across kava-using groups, triglycerides, homocysteine and diet-derived antioxidant vitamins α -tocopherol and retinol, did not vary. Plasma carotenoid levels (indicative of vegetable and fruit intake) were very low, but when adjusted for plasma cholesterol, did not vary between kava-using groups. An obsession for kava drinking may mediate kava's direct effects on nutritional status.

INTRODUCTION

In Pacific island societies, kava (*Piper methysticum* Forst. f.) has been used for centuries as aqueous extracts of the plant's crushed roots or lower stems.^{1, 2} Indigenous Australians in Arnhem Land (Northern Territory, NT) have used it in similar ways since 1982 when it was first brought from the Pacific.^{3, 4} A possible association between heavy kava use and sudden cardiac death in Indigenous Australians is an unresolved concern,⁵ although no clear evidence for increased risk of ischemic heart disease was found in a recent case-control study (unpublished data) or of atherogenesis in a cross-sectional study.⁶ Poor nutrition, which contributes to high rates of coronary heart disease (CHD) in Indigenous Australians, may add to this concern.^{7, 8}

In one Arnhem Land community, six years after kava was introduced, 'heavy' and 'very heavy' kava users showed signs of malnutrition with 20% lower body weight and 50% less subcutaneous fat.⁹ These same kava users had unusual lipid profiles with elevated high-density lipoprotein cholesterol (HDL).⁹ In a recent study, continuing kava users were compared with less regular users and a group of non-users. Lower body mass index (BMI), higher total cholesterol and a tendency for higher HDL were found in kava users while other CHD risk markers (C-reactive protein and homocysteine) were generally elevated.⁶

This short report seeks to clarify interrelationships between kava use, biomarkers of dietary quality and CHD risk, and nutritional status.

METHODS

In March 2000, a sample of 98 people (62 males, 36 females) was recruited in an eastern Arnhem Land Aboriginal community (340 people aged >15 years). Of these, 32 males and 4 females reported using kava. Seventeen males and nine females had ceased using it one year before the study while 13 males and 23 females never used kava. Self-reported kava consumption was confirmed by knowledgeable Aboriginal Health Workers and documentary evidence. Study setting, exposure methods and ethical approvals are described elsewhere.⁶ Participants gave written informed consent with procedures explained by Health Workers.

Sitting and standing heights (stadiometer) and skinfold thicknesses (calipers) were measured and body mass index (BMI) calculated.¹⁰ Percent body fat was measured using a Tanita Body Fat (TBF 521) instrument. Plasma samples (non-fasting) were analysed for lipids,⁶ carotenoids and micronutrients.⁸

Variations in continuous outcomes across kava using groups were tested using General Linear Modeling including tests for linear trend (SPSS V-11.0). Models included kava use (never, former and current) and gender as fixed factors and, for lipid-soluble antioxidants, plasma cholesterol as a covariate. Data for triglycerides, homocysteine, triceps and subscapular skinfold thicknesses, folate, α - and γ -tocopherol and carotenoids were skewed and were reported as geometric mean (95% confidence interval).

RESULTS

Fifty-three percent of continuing kava users and just 4% (n=1) of past users (P<0.001) showed dermopathy characteristic of prolonged use.¹¹ Adjusted for age, cholesterol levels beyond a reference range indicating increased CHD risk were more likely in continuing kava users than in never users for total cholesterol (OR=2.9, 1.2-7.0, P=0.005) and LDL (OR=2.5, 1.4-4.5, P=0.001) but less likely for HDL (OR=0.4, 0.2-0.7, P=0.001) (Table 5-4).

Table 5-4 shows the following. Total, LDL- and HDL cholesterol levels were highest in kava users and lowest in never users. Elevated HDL was less evident in kava users who had abstained for at least one year. BMI varied significantly by kava use with a non-significant tendency for a linear decrease. There were significant linear decreases in percent body fat and subscapular skinfold thickness, but not mid upper-arm circumference, across never, former and current kava use categories. Triceps skinfold varied significantly by kava usage, with no evidence of a linear trend. Compared to never users, current kava users had significantly lower percent body fat (P = 0.031) and subscapular skinfold thickness (P=0.027).

Table 5-5 shows that plasma levels of carotenoids were extremely low compared to other populations;^{12, 13} for example, they were approximately one-quarter of the carotenoid levels in non-Aboriginal subjects from Melbourne[unpublished data]. Average retinol and α -tocopherol concentrations were within normal ranges but not particularly low relative to other populations and did not vary by kava usage. After adjustment for plasma cholesterol levels, there were no differences between kava users and non-users for these biomarkers of dietary quality (with the exception of γ -tocopherol). High plasma homocysteine levels across all groups were consistent with low dietary folate intake and increased CHD risk.

Table 5-4: Coronary risk profile of survey participants stratified by kava use

| | Non-users | Past kava users | Continuing kava users | P_{ANOVA} | P_{linear trend} |
|--------------------------------------|------------------|------------------------|------------------------------|--------------------------|---------------------------------|
| | n=36 | n=26 | n=36 | | |
| male gender | 36% | 65% | 89% | <0.001 | <0.001 |
| age (yrs) | 36 (32-41) | 37 (32-42) | 36 (33-39) | 0.871 | - |
| Cholesterol total(mmol/L) | 4.2 (3.9-4.4) | 4.5 (4.2-4.8) | 5.3 (5.0-5.5)*†‡ | <0.001 | <0.001 |
| High density lipoproteins(mmol | 0.86 (0.76-0.96) | 0.97 (0.86-1.08) | 1.05 (0.95- 1.16)*‡ | 0.010 | 0.014 |
| Low density lipoproteins(mmol/L) | 2.8(2.5-3.1) | 3.1(2.7-3.4) | 3.7(3.4-4.0)*†‡ | 0.002 | <0.001 |
| Triglycerides(mmol/L) | 2.2 (1.8-2.6) | 2.2 (1.8-2.6) | 2.4 (1.9-2.8) | 0.824 | 0.567 |
| Body mass index (kg/m ²) | 23.2 (21.8-24.6) | 21.9 (20.3-23.6) | 21.5 (19.9-23.1) | 0.034 | 0.128 |
| Body fat (%) | 27 (24-30) | 25 (22-29) | 22 (19-25)* | <0.001 | 0.031 |
| Mid-upper arm circumference (cm) | 29.3 (28.1-30.5) | 29.4 (27.9-30.8) | 27.9 (26.5-29.2) | 0.265 | 0.144 |
| Skin fold thicknesses (mm) | | | | | |
| Triceps | 14.8(12.5-17.6) | 14.2(11.6-17.3) | 11.9(9.9-14.4) | <0.001 | 0.107 |
| Scapula | 26.5(22.4-31.4) | 24.4(20.0-29.8) | 19.6(16.2-23.7)* | <0.001 | 0.027 |

Data are estimated marginal mean (95% confidence interval); lipid and anthropometric data are adjusted for gender;

* significantly different from never users;

† significantly different from past users;

‡ elevated total cholesterol beyond the reference range for increased CHD risk (≥ 5.5 mmol/L) was found in 28% of continuing kava users and 6% of non-users, elevated LDL (≥ 3.4 mmol/L) in 65% of continuing users and 23% of non-users, with low HDL (≤ 0.9 mmol/L-M, ≤ 1.1 mmol/L-F) in 46% of current users and 85% of non-users

Table 5-5: Dietary markers stratified by kava use

| | Non-users n=36 | Past kava users n=26 | Continuing kava users n=36 | P_{ANOVA} | P_{linear trend} |
|---------------------------|--------------------------|--------------------------------|--------------------------------------|--------------------------|---------------------------------|
| Homocysteine (µmol/L) | 12.5(10.7-14.5) | 11.9(10.0-14.0) | 11.6(9.9-13.7) | 0.856 | 0.540 |
| Serum folate (ng/mL) | 6.3(5.0-7.9) | 7.7(5.9-10.1) | 5.0(3.8-6.5) | 0.086 | 0.203 |
| Retinol(µg/dL) | 51(46-56) | 53(47-58) | 55(49-61) | 0.706 | 0.406 |
| α-Tocopherol(µg/L) | 884(819-955) | 964(888-1045) | 980(899-1068) | 0.202 | 0.113 |
| γ-Tocopherol(µg/L) | 55(48-64) | 69(59-81)* | 82(70-97)* | 0.006 | 0.002 |
| β-Carotene (µg/dL) | 3.3(2.6-4.2) | 3.8(3.0-5.0) | 3.5(2.7-4.6) | 0.637 | 0.728 |
| α-Carotene (µg/dL) | 0.8(0.6-1.0) | 0.8(0.6-1.0) | 0.8(0.5-1.1) | 0.998 | 0.951 |
| Lycopene (µg/dL) | 6.5(5.0-8.3) | 6.4(4.9-8.3) | 8.1(6.1-10.7) | 0.440 | 0.291 |
| Cryptoxanthin (µg/L) | 1.7(1.4-2.1) | 1.6(1.2-2.0) | 1.4(1.1-1.7) | 0.467 | 0.225 |
| Lutain+Zeaxanthin (µg/dL) | 7.3(6.5-8.2) | 7.3(6.5-8.3) | 6.0(5.3-6.9) | 0.071 | 0.048 |
| Total carotenoids (µg/dL) | 20.8(17.8-24.3) | 20.9(17.7-24.7) | 21.4(18.0-25.5) | 0.974 | 0.828 |

Data are estimated marginal means (95% confidence interval); antioxidant data are adjusted for gender and plasma cholesterol;

* significantly different from never users.

DISCUSSION

The loss of total and upper body fat (indicated by low percent body fat and subscapular skinfold thickness respectively) was striking among current kava users compared to those who had never used it and this remained evident in former users. Although kava users had significantly lower body fat, low levels of biomarkers of dietary quality that were similar to those already reported for other Indigenous Australian populations,^{8, 14} were not further compromised in kava users. Loss of body fat in kava users has been described as similar to that in *anorexia nervosa* (Mathews et al., p.545).⁹ In Pacific island kava drinkers evidence for such weight loss, by contrast, is equivocal^{15, 16} which along with kava's association with feasting in Pacific traditions^{17, 18} implies the likely influence of social factors. Psychosocial factors are plausible causal agents for loss of body fat and are further indicated in Indigenous Australian kava drinkers. For example, some Arnhem Land Aboriginal people have used kava continuously for up to 18 years⁶ and unpublished participant-observations suggest that drinkers even constrain their eating in order to maximise kava's mood-altering effects. Therefore, notwithstanding possible economic deprivation and appetite suppression in kava users, an obsession with kava drinking may be a significant contributing factor.

While we could not identify associations of kava use with carotenoids, antioxidant vitamins, folate and homocysteine, concentrations of carotenoids and homocysteine were adversely low in all groups and suggested severe lack of cardioprotection associated with diets high in vegetables and fruit. We previously reported the tendency for elevated plasma cholesterol, including HDL, associated with kava use, but not triglycerides.⁶ However, the cholesterol levels in kava users were unremarkable relative to cholesterol levels in Caucasian populations. Tobacco was used by 83% of participants but was not associated with kava use⁶ (Fisher's exact P=0.110, data not shown) and generally low antioxidant levels did not vary across kava-using groups (Table 5-5). Nonetheless, greater risk of atherogenesis in kava users is biologically plausible if there is a greater proportion of small, dense LDL particles that may be more susceptible to oxidative modification, especially in the absence of adequate antioxidant levels, with possible additional tobacco-related CHD risk.

Confounding effects of excess alcohol use on lipid metabolism are possible,¹⁹ although unlikely since triglycerides were not elevated overall (Table 5-4). Drug-induced intrahepatic cholestasis is known to disrupt lipid profiles.¹⁹ But it is not known whether the obstructive hepatic response already reported in these same kava users⁶ could lead to atherogenesis with increased CHD risk.²⁰ Moreover, liver function changes are not necessarily accompanied by weight loss, unlike the dyslipidaemia of anorexia nervosa.¹⁹ Further research is warranted to consider the interrelationships of these factors with other measures of CHD risk.

In conclusion, we have found kava use to be associated with low body fat and dyslipidaemia. Biomarkers of fruit and vegetable consumption were also very low, but no more so than in other community members. These effects were less evident among former kava users. Thus while kava use among Aboriginal people is associated with malnutrition this effect is at least partly reversible.

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CHAPTER 6

CASE-CONTROL STUDY OF PNEUMONIA ADMISSIONS

INTRODUCTORY NOTES

Paper #7 in press

Clough AR, Wang Z, Bailie RS, Burns CB, Currie BJ. Case-control study of the association between kava use and pneumonia in eastern Arnhem Land Aboriginal communities (Northern Territory, Australia) [in press]. *Epidemiology & Infection*.

In Chapter 5 (paper#4) it was demonstrated that kava users showed lower lymphocytes, consistent with the findings of a previous cross-sectional study in another Arnhem Land community conducted 13 years earlier. Overall, it appeared that kava users in the recent study were not such heavy users as they were when the previous study was conducted. This is possibly an effect of a more restricted supply in what was an illegal trade in kava at the time the recent (2000) study was carried out.

The result of lower lymphocytes in kava users confirmed from the previous study and occurring even at more moderate levels of consumption suggests the possibility of an immune response in kava users. Does this immune response lead to an increased risk for serious infectious disease? To examine this question a case-control study compared admissions to hospital with a sample of age- and sex-matched controls selected from rolls prepared for each community. Paper#7 reports the results of this study. No evidence for an association was found.

Case-control study of the association between kava use and pneumonia in eastern Arnhem Land Aboriginal communities (Northern Territory, Australia)

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SUMMARY

Pneumonia causes significant morbidity and mortality in Aboriginal populations in Australia's Northern Territory (NT). Kava, consumed in Arnhem Land since 1982, may be a risk factor for infectious disease including pneumonia. A case-control study (n=115 cases; n=415 controls) was conducted in 7001 Aboriginal people (4217 over 15). Odds ratios (OR) were calculated by conditional logistic regression with substance use and social factors as confounders. Pneumonia was not associated with kava use. Crude OR=1.26 (0.74-2.14, P=0.386), increased after controlling for confounders (OR=1.98, 0.63-6.23, P=0.237) but was not significant. Adjusted OR for pneumonia cases being kava and alcohol users was 1.19 (0.39-3.62, P=0.756). In communities with longer kava-using histories, adjusted OR was 2.19 (0.67-7.14, P=0.187). There was no kava dose-response relationship. Crude ORs for associations between pneumonia and cannabis use (OR=2.27, 1.18-4.37, P=0.014) and alcohol use (OR=1.95, 1.07-3.53, P=0.026) were statistically significant and approached significance for petrol sniffing (OR=1.98, 0.99-3.95, P=0.056).

INTRODUCTION

In the 1980s in Australia's Northern Territory (NT), Aboriginal people accounted for 77% of hospitalisations for pneumonia and influenza with around a ten-fold risk of hospitalisation compared with non-Aboriginal people.¹ Death rates reported since then were more than ten times the Australian rate for the period 1981-1995, with little change during that time.²

Kava, the 'Intoxicating Pepper', (*Piper methysticum* Forst. f.) is a consciousness-altering muscle relaxant; a soporific with anaesthetic and analgesic properties.³ In the Pacific islands region, where the kava plant was domesticated, people have used it for centuries in a manner consistent with local cultural practices primarily in the form of aqueous emulsions of the crushed fresh or dried roots or lower stems.³ Kava powder mixed with water has been used by Aboriginal people in Arnhem Land since the early 1980s in eight settlements (isolated communities with from 200-1500 people) located along the 'Top End' coast of the NT.⁴ Kava was brought to Arnhem Land in part to minimise the harmful effects of alcohol use.^{5,6} However, kava use has been associated with abnormal liver function tests reported in published studies in the region.^{7,8} There have also been concerns of an association in Arnhem Land populations between kava use and various infectious diseases such as melioidosis.^{9,10}

Kava use in remote and isolated Aboriginal communities, which has continued despite NT Government efforts to restrict its use,¹¹ is now part of what has become a global controversy about the health effects of kava use. Serious irreversible liver damage has been reported in people using herbal extracts that contain kava lactones in several countries since the late 1990s.¹²⁻¹⁴ Consequently, manufactured kava-based products have become subject to medical alerts or bans on their sale in Europe,¹⁵ north America^{16,17} and Australasia.¹⁸

Amidst this broad international concern about kava's health effects, it is ironic that at the time of writing, the first kava legally available to Aboriginal communities under a

licensing regime introduced by the NT Government¹⁹ is being distributed in Arnhem Land. This study examined kava consumption as a potential independent risk factor for admission to hospital with pneumonia.

METHODS

Study Base

A case-control study was conducted in a base population that comprised all Aboriginal residents over the age of 15 years in the region's communities (2005 males and 2212 females in 1996²⁰).

Ethics

Ethical approval was provided by the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research which has an Aboriginal subcommittee and which works to National Health and Medical Research Council guidelines.

Cases

Data from Epidemiology Branch of Territory Health Services indicated that for ICD9 codes 4800 to 4879 'pneumonia and influenza', between 1994 and 1997 inclusive, there were 263 admissions (139 males and 124 females) to Gove District Hospital (GDH) or Royal Darwin Hospital (RDH) for Aboriginal people over the age of 15 from the regions' communities. These admissions were for 226 individuals (117 males, 109 females) with 37 people admitted more than once during the study period. GDH is the only secondary referral centre for the east Arnhem health region and the one to which most relevant cases are admitted initially. Some were admitted directly to the RDH and these were included in the cases.

Case Definition

An admission was included as a case in the study if there were symptoms of pneumonia plus an x-ray report consistent with the principal diagnosis of pneumonia recorded in hospital discharge summaries by the medical officer and reported in epidemiological data during 1994-1997.

Controls

Controls were selected at random from rolls prepared for each community. Controls (n=431, up to four per case) were matched with 120 cases for home locality, sex and age, in five-year groupings. The methods for selection of controls have been described elsewhere.²¹ Each community health centre in the region maintains a paper record of the medical history of local patients which is stored in files held in the health centre. This paper record is usually accompanied by a community list compiled from these files. The active file set held by the local community clinic together with a community list (where available) were used to compile the rolls used to select the controls. Inactive files for deceased persons were also included in the roll for possible selection as controls, i.e. any individual who had died after 1994. A reference date for enumerating ages across the study at the 1st of October, 1998 was used. Only those normally-resident in the local community were eligible for selection as controls.

Data Collection

For each case and control, consensus classification by health workers was used as already described.²¹ Data collection faced peculiar constraints. In accordance with the wishes of Aboriginal community representatives and their community controlled health service, face-to-face interviewing was not carried out with cases or controls. Additionally, a number of people were deceased among both cases (n=10) and controls (n=34). And, the status afforded kava, already highly stigmatised and controversial,

which became an illegal substance shortly after the study commenced,¹¹ also posed a significant challenge for data collection. These factors combined with the mobility of the region's population across the communities and nearby major centres in the 'Top End' of the NT, made interviewing of cases and controls infeasible. In order to overcome these difficulties, it was necessary to collect data by proxy using interviews with knowledgeable Aboriginal health workers combined with documentary sources.²¹

Aboriginal health workers in eastern Arnhem Land work in clinics in the communities in which they have lived for most of their lives and are held in high regard in those communities. They were often born in the communities where they work or in nearby communities that have close family and tribal affiliations. Most practising health workers in Arnhem Land are registered under the appropriate NT authority and have been trained on the job as well as through formal studies at an indigenous tertiary education institute at either diploma or certificate level. The senior health workers who provided information for this study have in some cases practised in the local clinic setting for 20 years.

For each case and control, consensus classification of kava use by health workers was compiled. Using their experience and knowledge without consulting clinic files and independently of each other, health workers evaluated whether cases or controls had ever used kava ('Yes' or 'No') and if they were using it during 1994-1997 ('Yes' or 'No'). To assess the occurrence of heavy drinking, we also asked health workers to describe the estimated time in a week a subject usually spent drinking kava and the time spent in activities where kava was consumed. Chart review data using local community clinic files assisted to confirm health worker consensus for both cases and controls. Similar methods were used to estimate exposure to other substance use and other related data. These methods for assessing exposure to kava use have been validated in an earlier study²¹ and the data collection procedures described in detail therein. To summarise, health workers concurred about an individual's history of kava use ($\kappa=0.83$), current use ($\kappa=0.43$) and also level of use ($\kappa=0.33$). We found good agreement between health workers' consensus and self-reported history of use ($\kappa=0.77$). Data from review of clinic patient notes and hospital discharge summaries

supported agreement between consensus classification and self-reported history and level of use ($\kappa=0.39$).²¹

Data Analysis

Analysis compared n=115 incident cases (60 males and 55 females) for whom kava use information was available with their matched controls (n=415, 225 males and 190 females). Conditional logistic regression was used to calculate crude and adjusted odds ratios (with 95% confidence intervals) with variable numbers of controls per case. Analyses were performed with Stata 7.0.²²

RESULTS

There were 106 out of the 226 individuals who were admitted with pneumonia as a subsidiary diagnosis and/or for whom x-ray findings were inconclusive or unavailable. These were excluded. X-ray findings were consistent with the medical officer's diagnosis and pneumonia was the principle diagnosis in 120 cases (63 males, 57 females). Kava use information was available for 115 of these.

The median age among 120 cases was 38 years (men 38, n=63 and women 39, n=57). Ninety-one cases (76%) originated from communities within the region where kava had been used since the early 1980s with the remaining 29 (24%) from communities with little or no kava using history. Based on these numbers, pneumonia admission rates during 1994-1997 were 1845 and 243 per 100000 in these communities respectively.

Kava Use and Pneumonia and the Effects of Alcohol and Other Substance Use

Kava users comprised 49% (56) out of 115 cases and 46% (190) out of 415 controls. There were more males among the cases who used kava (63%, n=38 males, 33%, n=18 females) as well as among the controls who used kava (58%, n=131 males, 31%=59 females).

There were similar proportions of kava and alcohol users among the cases and the controls (Table 6-1). In a regression model with just kava and alcohol, ORs for an association with pneumonia were 1.20 and 1.91 respectively but changed to 1.90 and 2.84 when the model included an interaction term for kava with alcohol (likelihood ratio test $K^2=2.11$, $P=0.147$) which, while non-significant suggested, that interaction effects should be considered in the multivariate model. No interaction of kava was found with either tobacco or cannabis use or petrol sniffing.

A crude odds ratio that those admitted for pneumonia were kava users was 1.26 (0.74-2.14, $P=0.386$) (Table 6-2a). In those who used no alcohol, after adjusting for the

Table 6-1 Kava and alcohol use among cases and controls

| | Cases | Controls |
|------------------------------------|-----------|------------|
| Kava used but no alcohol | 17%(n=19) | 16% (n=68) |
| Kava and alcohol used | 31%(n=35) | 29%(n=117) |
| Alcohol used but no kava | 27%(n=31) | 22% (n=90) |
| No kava and no alcohol used | 25%(n=28) | 33%(n=135) |

Proportions based on 113 cases and 410 controls for whom kava and alcohol use information were both available

effects of tobacco and cannabis use, petrol sniffing as well as body size, location of usual residence and a residual effect of age, the OR for kava use increased to 1.98 (0.63-6.23, $P=0.237$) (Table 6-3a). In those who used alcohol the OR for kava use was 1.19 (0.39-3.62, $P=0.756$). In a multivariate model that included confounders and an interaction term between kava and alcohol, OR for kava use was 2.22 and changed to 1.42 without the interaction (likelihood ratio test, $K^2=1.76$, $P=0.184$) indicating no significant interaction between kava and alcohol. There was no dose-response relationship (Table 6-2a and Tables 6-3a and 6-3b).

Among the cases normally resident in communities with a longer kava using history and who did not use alcohol, 19 were kava users and 13 were non-users with 68 users and 76 non-users among the controls. A crude OR for an association with kava use of 1.75 (0.65-4.72, $P=0.264$) in those who did not use alcohol in the kava using communities increased to 2.19 (0.67-7.14, $P=0.187$) in the multivariate analysis, i.e. little different from the OR using data for all communities.

There were crude associations between pneumonia admission and alcohol use (OR=1.95, 1.07-3.53, $P=0.026$), cannabis use (OR=2.27, 1.18-4.37, $P=0.014$) and a tendency for an association with petrol sniffing (OR=1.98, 0.99-3.95, $P=0.056$) (Table 6-2a).

Table 6-2a Crude odds ratios and 95% confidence intervals for the association between pneumonia and kava use and other substance use.

| "a" = reference category | Cases | Controls | OR | 95%CI | | P value* |
|---|-------|----------|------|-------|------|----------|
| Kava - use before or during admission period 1994-97 | | | | | | |
| Yes | 56 | 190 | 1.26 | 0.74 | 2.14 | 0.386 |
| a No | 59 | 225 | 1.00 | | | |
| Kava - level of use | | | | | | |
| a None | 59 | 225 | 1.00 | | | 0.282 |
| 1 night/week for a few hours | 6 | 8 | 3.01 | 0.95 | 9.58 | |
| > 1 night/week for a few hours | 3 | 20 | 0.68 | 0.18 | 2.48 | |
| about 2 nights a week | 10 | 43 | 0.90 | 0.40 | 2.05 | |
| Drink kava during the day as well as at night | 9 | 25 | 1.52 | 0.60 | 3.83 | |
| Sometimes drink for 24 hour sessions | 23 | 82 | 1.23 | 0.64 | 2.38 | |
| Alcohol - use before or during admission period 1994-97 | | | | | | |
| Yes | 66 | 207 | 1.95 | 1.07 | 3.53 | 0.026 |
| a No | 47 | 203 | 1.00 | | | |
| Alcohol - level of use | | | | | | |
| a None | 47 | 203 | 1.00 | | | 0.043 |
| Light | 14 | 31 | 2.51 | 1.09 | 5.79 | |
| Moderate | 9 | 53 | 1.11 | 0.44 | 2.78 | |
| Heavy | 40 | 114 | 2.19 | 1.13 | 4.24 | |
| Tobacco - use before or during admission period 1994-97 | | | | | | |
| Yes | 89 | 315 | 1.19 | 0.71 | 2.02 | 0.504 |
| a No | 25 | 95 | 1.00 | | | |
| Tobacco - level of use | | | | | | |
| a None | 25 | 95 | 1.00 | | | 0.214 |
| Up to 15 sticks a day | 3 | 11 | 0.96 | 0.22 | 4.25 | |
| 15-25 sticks a day | 3 | 29 | 0.41 | 0.11 | 1.49 | |
| One pack a day | 56 | 181 | 1.37 | 0.77 | 2.44 | |
| More than 1 pack a day | 22 | 85 | 1.06 | 0.50 | 2.22 | |
| Petrol - use before or during admission period 1994-97 | | | | | | |
| Yes | 16 | 30 | 1.98 | 0.99 | 3.95 | 0.056 |
| a No | 96 | 370 | 1.00 | | | |
| Cannabis - use before or during admission period 1994-97 | | | | | | |
| Yes | 26 | 57 | 2.27 | 1.18 | 4.37 | 0.014 |
| a No | 79 | 327 | 1.00 | | | |

* P-values reflect trend across categories.

Table 6-2b Crude odds ratios and 95% confidence intervals for the association between pneumonia and social and demographic characteristics.

| "a" = reference category | | Cases | Controls | OR | 95%CI | P value* |
|---|------------------------|-------|----------|------|-----------|----------|
| Employment | | | | | | |
| a Paid employment (not including CDEP) | In labour force | 16 | 57 | 1.00 | | |
| CDEP | In labour force (CDEP) | 31 | 146 | 0.62 | 0.30 1.29 | |
| Supporting parent Pension | | | | | | |
| Unemployment benefit | Not in labour force | 58 | 176 | 1.23 | 0.63 2.40 | 0.049 |
| No money | | | | | | |
| Time spent on outstations or community | | | | | | |
| a Mostly on community | | 67 | 250 | 1.00 | | |
| Divided between community and outstations | | 19 | 93 | 0.70 | 0.38 1.28 | 0.145 |
| Mostly on outstations | | 9 | 28 | 1.06 | 0.45 2.47 | |
| Live outside of the area | | 11 | 20 | 2.04 | 0.92 4.54 | |
| Condition of house | | | | | | |
| a Good | | 64 | 263 | 1.00 | | |
| Fair | | 11 | 47 | 1.32 | 0.58 2.99 | 0.693 |
| Poor | | 11 | 25 | 2.41 | 0.99 5.83 | |
| People living in the house | | | | | | |
| More than 10 | | 38 | 128 | 0.80 | 0.37 1.74 | 0.024 |
| 6 to 10 | | 27 | 139 | 0.47 | 0.23 0.99 | |
| a 5 or less | | 20 | 60 | 1.00 | | |
| Goes hunting regularly | | | | | | |
| Yes | | 60 | 229 | 1.04 | 0.62 1.77 | 0.872 |
| a No | | 34 | 127 | 1.00 | | |
| Diet includes bush food | | | | | | |
| Yes | | 68 | 289 | 0.67 | 0.37 1.18 | 0.168 |
| a No | | 24 | 71 | 1.00 | | |
| Active person | | | | | | |
| a Yes | | 62 | 291 | 1.00 | | |
| No | | 32 | 47 | 3.86 | 1.94 7.71 | <0.001 |
| Body size | | | | | | |
| Overweight | | 8 | 41 | 1.00 | 0.42 2.37 | 0.143 |
| a About right | | 65 | 295 | 1.00 | | |
| Underweight | | 37 | 74 | 2.33 | 1.42 3.82 | |

* P-values reflect trend across categories.

Table 6-3a Adjusted odds ratios and 95% confidence intervals for the association between pneumonia and kava use in those who do not use alcohol.

(Adjusted for a history of petrol sniffing, tobacco and cannabis use, usual residence, body size and age).

| "a" = reference category | Cases | Controls | OR | 95%CI | | P value* |
|---|-------|----------|------|-------|--------|----------|
| Kava - use before or during admission period 1994-97 | | | | | | |
| Yes | 19 | 68 | 1.98 | 0.63 | 6.23 | 0.237 |
| a No | 28 | 135 | 1.00 | | | |
| Kava - level of use | | | | | | |
| a None | 28 | 135 | 1.00 | | | 0.165 |
| 1 night/week for a few hours | 4 | 4 | 6.14 | 0.69 | 54.44 | |
| > 1 night/week for a few hours | 1 | 6 | 0.81 | 0.05 | 12.70 | |
| † about 2 nights a week | 2 | 14 | . | . | . | |
| Drink kava during the day as well as at night | 4 | 7 | 9.61 | 0.79 | 117.98 | |
| Sometimes drink for 24 hour sessions | 7 | 28 | 2.51 | 0.45 | 13.95 | |

* P-values reflect trend across categories.

† No information about effect because of sparse data

Table 6-3b Adjusted odds ratios and 95% confidence intervals for the association between pneumonia and kava use in those who use alcohol.

(Adjusted for a history of petrol sniffing, tobacco and cannabis use, usual residence, body size and age).

| "a" = reference category | Cases | Controls | OR | 95%CI | | P value* |
|---|-------|----------|------|-------|------|----------|
| Kava - use before or during admission period 1994-97 | | | | | | |
| Yes | 35 | 117 | 1.19 | 0.39 | 3.62 | 0.756 |
| a No | 31 | 90 | 1.00 | | | |
| Kava - level of use | | | | | | |
| a None | 31 | 90 | 1.00 | | | 0.146 |
| † 1 night/week for a few hours | 2 | 4 | . | . | . | |
| > 1 night/week for a few hours | 2 | 14 | . | . | . | |
| about 2 nights a week | 8 | 27 | 1.18 | 0.26 | 5.34 | |
| Drink kava during the day as well as at night | 5 | 18 | 1.19 | 0.20 | 7.03 | |
| Sometimes drink for 24 hour sessions | 15 | 51 | 0.94 | 0.24 | 3.76 | |

* P-values reflect trend across categories.

† No information about effect because of sparse data

Social and Contextual Data

Published data for 1996 for the region, showed that an average of between 8 and 9 people lived in each of the 839 available dwellings.²⁰ The modal category in our data was from 6 to 10 persons per dwelling among controls (Table 6-2b). Those living on outstations (smaller living areas with up to 120 people) were around 18% in 1996.²⁰ In our data 7% (n=28) of controls were outstation residents with another 24% (n=93) spending time in both communities and outstations. Five percent (n=20) were normally resident in the community but also resided in a town or major regional centre during the study period. Fifty-four percent (n=203/379) of controls were in paid employment or CDEP (Community Development Employment Program) with the balance not in the labour force at the time of the survey (Table 6-2b), somewhat higher than in the published data for 1996 (37%).²⁰ In our data, no people relied totally on a diet of 'bush food', while those who were active and who had superior diets were also regular hunters (Table 6-2b).

While the direction of the association is not clear, pneumonia admission appears to be associated with both participation in the labour force and the number of people living in the house where the case or control resided (Table 6-2b). There is an association of pneumonia admission with people who were regarded as 'inactive' by health workers (crude OR=3.86, 1.94-7.71, P<0.001).

DISCUSSION

To our knowledge, this is the first controlled study to consider kava use as an independent risk factor for pneumonia in any population. Admission for pneumonia was not associated with kava use.

Confounding Effects

A dynamic substance use complex exists in eastern Arnhem Land Aboriginal populations involving changing patterns of cannabis use, petrol sniffing, tobacco and alcohol use as well as kava.²³ Kava is usually consumed in the home community with groups of between 2 and 11 drinkers participating and with varying numbers of casual drinkers. Alcohol is consumed periodically in the home community, but usually in the nearby large towns or in one of two regional centres, where it is more freely available and where heavy drinking is a common pattern of consumption.²⁴ Petrol sniffing occurs sporadically with periodic resurgences.²³ Cannabis use (14%, n=57 controls) and a high prevalence of a history of tobacco use (77%, n=315 controls) was seen (Table 6-2a). The prevalence of cannabis use has increased rapidly over the past five years.²³

The multivariate analysis adjusted for confounders including tobacco, a known risk factor for pneumonia.^{25,26} A weak interaction between kava and alcohol use, also a known risk factor for pneumonia,²⁷⁻²⁹ was considered but found to be non-significant. Effects of alcohol use, if they exist, were controlled in the stratified analysis (Tables 6-3a and 6-3b). Body size, age and place of usual residence (a likely surrogate for heavy alcohol use) were also adjusted for in the multivariate model. Petrol sniffing, which is known to have occurred widely in the region for upwards of 30 years,³⁰ and cannabis use which may lead to respiratory abnormalities similar to those among tobacco users³¹ perhaps by compromising the lung's immune system³² were also included in the multivariate analysis. No comprehensive data were available describing the presence

or absence of chronic obstructive pulmonary disease or other chronic diseases in the cases or controls and so these variables could not be included in the analysis.

Strengths and Weaknesses of the Study

This study uses data for all cases of pneumonia admitted from eastern Arnhem Land for a defined period where the majority of kava users in the Aboriginal population reside and for whom diagnosis could be confirmed. It is possible that information bias²¹ occurred in this study because health workers may have known not only the kava using status of individuals in the study but also their hospital admission history and perhaps unwittingly reported substance use status for those known to have been admitted to hospital more readily than for those they knew had not been admitted. This prospect was minimised; except for appropriate identifiers, health workers were blinded to any information about cases and controls during interview. We have assessed the level of agreement that health workers demonstrated about kava using status of individuals known to them.²¹ It seems highly unlikely that all health workers who provided information would show the same pattern of information bias and that this bias would also occur between health worker consensus and self-reported data.

The higher admission rates for pneumonia in kava using communities compared with those with little or no kava use history may be due to a difference in the social and environmental factors affecting disease rates in each subregion. It is also possible, however, that these differences are due more to differential treatment regimes; clinics in one part of the region tending to treat pneumonia cases in the community rather than evacuating them to the regional hospital. There are also major cultural and historical differences between kava using communities, Yolŋu communities,³³ and those with little or no kava using history, Anindilyakwa and Nunggubuyu communities,³⁴ which may be reflected in the differential use of health services.

Imprecise measures of the level of kava use was an unavoidable weakness in this study, in part a consequence of the lack of reliable supply and consumption data given kava's

illegal or stigmatised status throughout the course of the data collection and study periods.¹¹ Kava supply and consumption had likely reached its apogee in 1996 and 1997 under the pervasive influence of an informal trade, but reliable supply information does not exist from 1994 onwards because the regulatory regime operating at that time was dismantled⁴ and not reinstated. For this reason, it is unlikely that more precise determination of consumption levels during the study period can be made. Future monitoring of kava's health effects may benefit from the availability of reliable community supply information with which to compare usage levels reported by proxy.

Plausibility of a History of Kava Use as a Cause for Pneumonia

There may be an immune system abnormality in kava users. In an earlier study heavy kava use was found to be associated with decreased lymphocytes.⁷ In a recent study we also found decreased lymphocytes (with 51% of kava users having levels below the normal reference range) along with a tendency for higher IgE among kava users.⁸ It is likely that decreased lymphocyte levels return to normal upon ceasing kava use, as do elevated liver enzymes with abstinence from kava.^{8,35} Lymphocyte levels in past kava users ($1.8 \times 10^9/L$, $sd=0.6$) tended to be higher than in current users ($1.6 \times 10^9/L$, $sd=0.6$)⁸ although the comparison was not statistically significant ($|t|=1.26$, $P=0.213$).

Despite the possibility of an immune abnormality, this study has shown that kava use alone is not clearly an independent risk factor for pneumonia as it appears to be for other serious infectious disease such as melioidosis.^{10,36} Information bias, incorrect diagnosis, and differential treatment regimes across the region may have confounded kava's effect if it exists.

The statistically significant associations between pneumonia admission and alcohol use, pneumonia admission and cannabis use and the tendency for an association with petrol sniffing (Table 6-2a), suggest that the effects of other substances, or combinations of substances, may also be important or may compound kava's effects in

ways not yet understood. These should be further examined as risk factors for pneumonia, and other serious infectious disease.

Environmental and biological processes that may increase pneumonia risk, such as lack of employment opportunities, poor nutrition and poor community living conditions, are likely to persist alongside the complex of substance use in Arnhem Land and its legacy. If Aboriginal people continue to drink kava in this context, it would be prudent to moderate consumption even though an association with pneumonia has not been shown.

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CHAPTER 7

CASE-CONTROL STUDY OF ISCHAEMIC HEART DISEASE ADMISSIONS

INTRODUCTORY NOTES

Paper #8: in press

Clough AR, Wang Z, Bailie RS, Burns CB, Currie BJ. Case-control study of the association between kava use and ischaemic heart disease in Aboriginal communities in eastern Arnhem Land (Northern Territory) Australia [in press]. *Journal of Epidemiology and Community Health*.

Although no clear indication of increased risk of atherosclerosis emerged in the cross-sectional study (Chapter 5, paper#4), which included kava users who had used it more or less continuously for up to 18 years (median 12 years), anecdote remained strong that young men suffering heart attacks were often known to be heavy kava users. While the cross-sectional study showed no convincing evidence of markers of increased risk of cardiovascular disease in kava users, the significance of the dyslipidaemia observed in kava users was not known.

To investigate a possible association between kava use and ischaemic heart disease, a case-control study was carried out. Admissions to hospital for ischaemic heart disease were compared with a sample of age- and sex-matched controls selected from rolls prepared for each community. Paper#8 reports the results of this study. Data were collected for this study concurrently with data for the case-control study for pneumonia, i.e. during 1999-2000. No evidence for an association between kava use and ischaemic heart disease was found.

The sample size in this study was much smaller than for the study of pneumonia admissions and so the power of the study was correspondingly reduced. Nevertheless, because of the dearth of information about these kinds of effects in kava users, the study provides a useful beginning.

**Case-control study of the association between
kava use and ischaemic heart disease in
Aboriginal communities in eastern Arnhem Land
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ABSTRACT

Study objective: This paper examines kava consumption as a risk factor for IHD. Ischaemic heart disease (IHD) causes significant morbidity and mortality in Indigenous Australians. Kava, the mood-altering drink from the Pacific, has been consumed in eastern Arnhem Land (Northern Territory, NT) communities since 1982. There is some circumstantial evidence of an association between kava consumption, heart disease and sudden cardiac death.

Design: A case-control study (n=83 cases; n=302 controls) was conducted. Main risk measure was exposure to kava use prior to or during the study period, 1992-1997. To control confounding, other substance use (alcohol, tobacco, petrol sniffing and cannabis) were also recorded. Crude and adjusted odds ratios were calculated by conditional logistic regression.

Setting: Eastern Arnhem Land has 7001 Aboriginal people.

Participants: The study was conducted in 4217 people aged over 15. Cases comprised those admitted to hospital during 1992-1997 from the region's communities. Up to four controls per case, were matched for age, sex and home locality.

Main results: A crude OR=1.41 (0.73-2.73, P=0.303) that those admitted with an ischaemic event were kava users changed little after controlling for confounders (OR=1.51, 0.75-3.05, P=0.247). In just those communities with a longer (up to 15 year) history of kava use, odds ratios for kava use among those admitted with an ischaemic event increased (OR=1.75, 0.82-3.74, P=0.140) but was not significant. Twenty-five cases were admitted more than once. Risk of readmission tended to be higher in kava users (adjusted OR=2.24, 0.65-7.68, P=0.191) but not statistically significant. Twenty people died with IHD as the principal cause without admission to hospital during 1992-1997. Even with these cases and matched controls included in the analysis, no association between IHD and kava use (adjusted OR=1.44, 0.78-2.66, P=0.245) was found.

Conclusions: This study provides no clear evidence for an association between kava use and IHD.

INTRODUCTION

Death rates from diseases of the circulatory system are 3.1 times greater in Indigenous males and 2.8 times greater in Indigenous females^{1,2} than in the rest of Australia and 7-12 times higher in 25-54 year olds,² with almost one-third of excess mortality in Indigenous Australians due to this cause.³ Fifty-seven percent of the deaths from circulatory disease are due to ischaemic heart disease (IHD).² In the Northern Territory (NT), from 1979-95, circulatory disease in Aboriginal people became a leading cause of death with death rates of up to 3454 among males and 2859 among females (per 100 000 population), rates 41% and 31% higher, respectively, than in the Australian population.⁴ Recently, death rates for IHD among NT Aboriginals have increased from 220 to 321 for males and from 72 to 177 (per 100 000 population) for females between 1981-95, being 40%-50% higher than in the wider Australian population.⁴

Kava, (*Piper methysticum* Forst. f., 'Intoxicating pepper') is a consciousness-altering muscle relaxant; a soporific with anaesthetic and analgesic properties.⁵ Pivotal to the economy, society and ritual traditions of Pacific Island nations,⁵ although perhaps becoming a contemporary drug of abuse in those societies,⁶ kava drinking is now part of broad lifestyle changes in the remote Arnhem Land region. Aboriginal people in Arnhem Land consume it as an infusion in cold water of dried powdered kava imported from Fiji or Tonga.⁷ It was first brought to eastern Arnhem Land in 1982 by local Aboriginal people who sought to enjoy its mood-altering and stress relieving properties, partly to minimise alcohol's devastating effects in their communities.⁷ Its use, however, soon became contentious⁸ and kava drinking is now implicated in serious infectious disease,^{9,10} hospital presentations for unusual neurological conditions including possible *grand mal* seizures,^{11,12} and sudden cardiac deaths among, particularly, young Aboriginal sportsmen.¹³ But there have been few systematic studies of kava's health effects.¹⁴ In particular the role of kava as a risk factor for IHD has not been assessed.

Possible harmful^{13,15} as well as protective^{16,17} mechanisms have been reported for kava and its lactones on IHD, and yet other evidence from Arnhem Land^{14,18} shows no clear indication of increased atheroma risk. This study seeks to determine if kava consumption alone is a risk factor for admission to hospital with IHD for Aboriginal people in Arnhem Land.

Kava supply and consumption remain controversial in Arnhem Land despite possible health effects and kava's illegal status since 1998.¹⁹ Adding to this controversy, manufactured 'herbal' remedies that include kava lactones, used worldwide as alternative anxiolytics,²⁰ are now implicated in cases of hepatitis and liver failure.^{21,22} Aboriginal people in Arnhem Land have no knowledge of manufactured kava-based products or herbal remedies. Yet, kava drinking practices in remote and isolated communities in northern Australia are now inescapably drawn into a global controversy about kava. When data for this paper was being analysed, the first kava legally available to Aboriginal communities under a licensing regime in the NT²³ was being distributed. At the same time (March, 2002) following international reports of hepatic toxicity from kava products, the Parliamentary Secretary for the Commonwealth Minister of Health and Ageing announced that the Therapeutic Goods Administration had issued an alert to health professionals in Australia.²⁴

METHODS

Study base

A case-control study was conducted in a base population comprising all Aboriginal residents over the age of 15 years in the region's communities (2005 males, 2212 females in 1996²⁵).

Cases

Between 1992-1997, 83 individuals (47 males, 36 females) were admitted for the first time to Gove District Hospital (GDH) or Royal Darwin Hospital (RDH) with a primary diagnosis of ischaemic heart disease (ICD9 Codes 4100-4149) and for whom kava use information was available. GDH is the only secondary referral centre for the east Arnhem health region to which most relevant cases normally-resident in the region's communities are admitted initially. Some are admitted directly to RDH, located in the NT's principal urban centre. Hospital discharge summaries were inspected to confirm medical officer's diagnoses coded in epidemiological databases. Twenty-five individuals were admitted on more than one occasion during 1992-1997.

Controls

Controls were selected at random from rolls prepared for each community. Controls (up to four per case) were matched for sex and age, in five-year groupings. Each community health centre in the region maintains patients' medical records in the health centre. Usually there is a community list compiled from these files. The local community clinic's active file set, together with a community list (where available), were used to compile the rolls. Inactive files for deceased persons were included for possible selection as controls, i.e. any individual who died after 1992. Ages were enumerated from the reference date of 1 October, 1998. Only those normally resident

in the local community were eligible for selection as controls. There were 302 controls used in the analysis.

Ethics

Ethics approval was provided by the Joint Institutional Ethics Committee of the Royal Darwin Hospital and Menzies School of Health Research which works to National Health and Medical Research Council (Australia) guidelines and which has an Aboriginal subcommittee.

Data collection

Data collection faced constraints not normally encountered in epidemiological studies making interviewing of cases and controls infeasible. Cross-cultural and language differences constrained data collection. Compounding this, Aboriginal community representatives and their community controlled health service instructed that face-to-face interviews were not carried out in their jurisdictions. A large number of people were deceased among both cases (n=20) and controls (n=38). Within the region and between the major centres in the 'Top End' of the NT the region's Aboriginal population is remarkably mobile. Kava drinking was already highly stigmatised and it became illegal shortly after the study commenced.¹⁹ To overcome these difficulties data were collected by proxy using interviews with knowledgeable Aboriginal health workers (who were also local community residents) combined with documentary sources.²⁶

Consensus classification of kava use was compiled for each case and control. Using their community experience and intimate knowledge, without consulting clinic files and independently of each other, health workers evaluated whether cases or controls had ever used kava ('Yes' or 'No') and if they were users during 1992-97 ('Yes' or 'No'). To categorise heavy drinkers, we also asked health workers to describe the estimated time in a week a subject usually spent drinking kava and the time spent in activities where kava was consumed. Clinic files and hospital discharge summaries assisted to

confirm health worker consensus. Similar methods were used to estimate exposure to other substance use and to compile other related data. These methods for assessing exposure to kava use have been validated and described in detail elsewhere.²⁶ To summarise, health workers concurred about an individual's history of kava use ($\kappa=0.83$), current use ($\kappa=0.43$) and also level of use ($\kappa=0.33$) with good agreement between health workers' consensus and self-reported history of use ($\kappa=0.77$). Data from clinic patient notes and hospital discharge summaries supported this agreement ($\kappa=0.39$).²⁶

Data analysis

Conditional logistic regression was used to estimate crude odds ratios (ORs) and 95% confidence intervals for the association between kava use and IHD (Table 7-1). Using conditional logistic regression, the independent contribution of kava use was assessed by estimating ORs adjusting for potential confounding factors of alcohol, tobacco use, petrol sniffing and body size (Table 7-2a). Since kava use was not uniform across the region, we further assessed the association between kava use and IHD using just the data from communities with more extensive kava-use history (Table 7-2b). Analyses were performed using Stata 7.0.²⁷

RESULTS

Kava use and IHD

Forty percent (n=33) of cases and 34% (n=103) of controls used kava. Twenty-four (51%) of 47 male cases and 9 (25%) of 36 female cases used kava. Among controls, proportions of kava users were similar (47%=79/169 males, 18%=24/133 females). Adjusting for the effects of alcohol use, petrol sniffing, tobacco and body size (health workers' estimate of body mass), the OR for kava use prior to or during the study period changed from 1.41 (0.73-2.73, P=0.303) to 1.51 (0.75-3.05, P=0.247) (Tables 7-1 and 7-2a); and was not statistically significant. A residual confounding effect of age was considered in the multivariate model but with little change in the OR for kava use (1.50, 0.74-3.04), ($K^2=0.23$, likelihood ratio test, P=0.635).

Four of the region's communities had little or no kava use history while five others had continuous access to kava supplies for up to 15 years. Since people normally resident in kava using communities have potentially had longer and perhaps continuous exposure to kava use, an association with IHD may exist in this group. Table 7-2b, shows that while the OR increased using data for just these communities (OR=1.75, 0.82-3.74, P=0.140), it was little different between males (OR=1.89, 0.66-5.40, P=0.216) and females (OR=1.57, 0.50-4.92, P=0.438). None of these ORs were statistically significant and there was no dose-response relationship (Table 7-1 and Table 7-2a).

Table 7-1. Crude odds ratios and 95% confidence intervals for the association between IHD and kava use and other substance use

| "a" = reference category | Cases | Controls | OR | 95%CI | P value |
|--|-------|----------|------|------------|---------|
| Kava – use before or during admission period 1992-97 | | | | | |
| Yes | 33 | 103 | 1.41 | 0.73 2.73 | 0.303 |
| a No | 50 | 199 | 1.00 | | |
| Kava – level of use | | | | | |
| a None | 50 | 199 | 1.00 | | 0.342 |
| 1 night/week for a few hours | 4 | 6 | 3.43 | 0.76 15.58 | |
| > 1 night/week for a few hours | 7 | 13 | 2.17 | 0.75 6.30 | |
| About 2 nights a week | 6 | 17 | 1.61 | 0.57 4.56 | |
| Drink kava during the day as well as at night | 2 | 14 | 0.63 | 0.12 3.29 | |
| Sometimes drink for 24 hour sessions | 13 | 48 | 1.11 | 0.45 2.75 | |
| Alcohol - use before or during admission period 1992-97 | | | | | |
| Yes | 41 | 143 | 1.21 | 0.61 2.39 | 0.590 |
| a No | 40 | 157 | 1.00 | | |
| Alcohol - level of use | | | | | |
| a None | 40 | 157 | 1.00 | | 0.679 |
| Light | 5 | 15 | 1.91 | 0.54 6.73 | |
| Moderate | 6 | 31 | 0.86 | 0.29 2.51 | |
| Heavy | 26 | 92 | 1.26 | 0.60 2.64 | |
| Tobacco - use before or during admission period 1992-97 | | | | | |
| Yes | 71 | 252 | 1.29 | 0.61 2.70 | 0.500 |
| a No | 11 | 50 | 1.00 | | |
| Tobacco - level of use | | | | | |
| a None | 11 | 50 | 1.00 | | 0.019 |
| Up to 15 cigarettes a day | 5 | 8 | 3.03 | 0.72 12.70 | |
| 15-25 cigarettes a day | 2 | 33 | 0.26 | 0.05 1.29 | |
| One pack a day | 36 | 105 | 1.80 | 0.80 4.06 | |
| More than 1 pack a day | 26 | 99 | 1.14 | 0.49 2.64 | |
| Petrol - use before or during admission period 1992-97 | | | | | |
| Yes | 5 | 14 | 1.32 | 0.41 4.22 | 0.648 |
| a No | 76 | 279 | 1.00 | | |
| Cannabis – use before or during admission period 1992-97 | | | | | |
| Yes | 5 | 33 | 0.46 | 0.16 1.35 | 0.137 |
| a No | 74 | 257 | 1.00 | | |

Table 7-2a. Adjusted odds ratios and 95% confidence intervals for the association between IHD and kava use (adjusted for a history of alcohol and tobacco use, petrol sniffing and body size)

| "a" = reference category | Cases | Controls | OR | 95%CI | | P value |
|--|---|----------|------|-------|------------|---------|
| Kava – use before or during admission period 1992-97 | | | | | | |
| Yes | 33 | 103 | 1.51 | 0.75 | 3.05 | 0.247 |
| a No | 50 | 199 | 1.00 | | | |
| Kava – level of use | | | | | | |
| a None | None | 50 | 199 | 1.00 | | 0.469 |
| | 1 night/week for a few hours | 4 | 6 | 3.48 | 0.74 16.30 | |
| | > 1 night/week for a few hours | 7 | 13 | 1.64 | 0.48 5.55 | |
| | about 2 nights a week | 6 | 17 | 1.83 | 0.62 5.44 | |
| | Drink kava during the day as well as at night | 2 | 14 | 0.63 | 0.12 3.38 | |
| | Sometimes drink for 24 hour sessions | 13 | 48 | 1.30 | 0.51 3.35 | |

Table 7-2b. Adjusted odds ratios and 95% confidence intervals for the association between IHD and kava use stratified by sex in communities with a significant kava using history (adjusted for a history of alcohol and tobacco use, petrol sniffing and body size)

| "a" = reference category | Cases | Controls | OR | 95%CI | | P value |
|--|-------|----------|------|-------|------|---------|
| Males | | | | | | |
| Kava - use before or during admission period 1992-97 | | | | | | |
| Yes | 24 | 76 | 1.89 | 0.66 | 5.40 | 0.216 |
| a No | 7 | 35 | 1.00 | | | |
| Females | | | | | | |
| Kava - use before or during admission period 1992-97 | | | | | | |
| Yes | 9 | 23 | 1.57 | 0.50 | 4.92 | 0.438 |
| a No | 11 | 53 | 1.00 | | | |
| Total | | | | | | |
| Kava - use before or during admission period 1992-97 | | | | | | |
| Yes | 33 | 99 | 1.75 | 0.82 | 3.74 | 0.140 |
| a No | 18 | 88 | 1.00 | | | |

Kava, alcohol and tobacco use

Table 7-3a shows that there were comparable proportions of kava and alcohol users and abstainers among cases and controls. Kava and tobacco use also showed similar proportions in cases and controls (Table 7-3b). Surprisingly, no crude association was found between tobacco use and admission for IHD (Table 7-1). However, it was possible to compare 30 cases with 122 controls who used neither kava, alcohol, petrol nor cannabis. In this analysis, the OR=4.37(0.91-20.99, P=0.036) that those requiring admission for IHD were tobacco users is more consistent with our knowledge of heart disease risk factors. Anecdotal reports from other studies in Indigenous Australians where the expected association between tobacco use and other major chronic diseases has not been found, or is even reversed, suggest possible unknown or unmeasured confounders.

The presence of chronic conditions

No comprehensive screening for chronic conditions was available. Among individuals admitted 24 had a history of hypertension, 19 diabetes, 12 renal disease, 11 rheumatic fever or rheumatic heart disease and 16 had cardiovascular problems (blood pressure, dyslipidaemia, chest pain) treated in the year prior to their admission. For controls the corresponding figures were 41, 42, 23, 5 and 9.

Risk of readmission for IHD

Those admitted on more than one occasion (n=25 individuals) were compared with their matched controls (n=132). There was no association with kava use (crude OR=1.68, 0.55-5.15, P=0.356). When adjusted for confounders (alcohol use, petrol sniffing, tobacco and body size) the OR increased to 2.24 (0.65-7.68, P=0.191) but was not statistically significant.

Table 7-3a. Kava and alcohol use among cases and controls

| | Cases* | Controls* |
|-----------------------------|---------------|------------------|
| Kava used but no alcohol | 12%(n=10) | 11%(n=32) |
| Kava and alcohol used | 27%(n=22) | 23%(n=69) |
| Alcohol used but no kava | 23%(n=19) | 25%(n=74) |
| No kava and no alcohol used | 37%(n=30) | 42%(n=125) |

* Proportions based on 81 cases and 300 controls for whom kava and alcohol use information were both available

Table 7-3b. Kava and tobacco use among cases and controls

| | Cases* | Controls* |
|-----------------------------|---------------|------------------|
| Kava used but no tobacco | 2%(n=2) | 2%(n=7) |
| Kava and tobacco used | 38%(n=31) | 32%(n=96) |
| Tobacco used but no kava | 49%(n=40) | 52%(n=156) |
| No kava and no tobacco used | 11%(n=9) | 14%(n=43) |

* Proportions based on 82 cases and 302 controls for whom kava and alcohol use information were both available

DISCUSSION

To our knowledge, this is the first study of kava as an independent risk factor for IHD in any population. No clear evidence for an association was found. Since the smallest detectable effect size was around two (80% power, 95%CI), the potential for confounding or measurement error to explain any association of less than two is likely to be high given the complexities of exposure measurement. Nevertheless, in the absence of previous studies in this area, we believe that excluding an effect size greater than two is a reasonable beginning. Since, the direction of effect was towards an increased risk of approximately 50%, the study provides an important basis for further research.

Confounding effects of other substance use

Difficulties in identifying heavy kava use in particular,⁷ the effects of alcohol use, a background of petrol sniffing and emerging cannabis use^{7,18} along with a high prevalence of a history of tobacco use (83%, n=252 controls, Table 7-1) made assessment of the independent effects of kava use difficult. The multivariate analysis adjusted for known risk factors including tobacco use, alcohol use and body size (Table 7-2a). The analysis also adjusted for a history of petrol sniffing which is known to have occurred widely in the region for upwards of 30 years.^{28,29}

The finding of no significant crude association between admission for IHD with the well established risk factor of tobacco use, however, raises concerns regarding exposure measurement even though an analysis comparing tobacco users with non-users and limited to cases and controls who used no other substances showed a stronger association between tobacco use and IHD. Data from NT Births, Deaths and Marriage Registrar indicated that during 1992–1997, 20 individuals from the region died with IHD as the principal cause with no record of admission to hospital. These 20 cases and their 75 matched controls were combined with the known hospital admissions (n=83) and matched controls (n=302). This analysis too showed no association between IHD

and kava use (adjusted OR=1.44, 0.78-2.66, P=0.245) indicating that the findings of the analysis of IHD admissions alone were not influenced by survival bias. With this data in an analysis using the 36 cases and 158 controls with no record of other substance use, the expected significant association between IHD and tobacco use alone emerged after all (OR=3.96, 1.08-14.49, P=0.021).

Strengths and weaknesses of the study

The study used data for all new cases of IHD admitted from a discrete region for a defined period where a majority of kava users have resided and with IHD the primary diagnosis. Methods used to assess exposure which relied on consensus classification by knowledgeable Aboriginal health workers may have overcome language and cultural barriers.²⁶ However, it is possible that information bias²⁶ occurred with health workers knowing kava using status and hospital admission histories of cases and controls and perhaps unwittingly reporting substance use status more readily for those admitted than for those they knew had not been admitted. This prospect was minimised since health workers were blinded to information about cases and controls during interview, except for appropriate identifiers. Moreover, the agreement between health workers describing individuals' kava using status was fair to very good.²⁶ Given this agreement it seems unlikely that all health workers providing information would show the same pattern of information bias.

Further reinforcing our confidence in the exposure measures used, it has been shown from a sample in one of the region's communities with 101 people interviewed face-to-face, that health workers' consensus (obtained separately) agreed with participants' self-reported history of tobacco use ($\kappa=0.82$), and alcohol ($\kappa=0.77$)¹⁸ with good agreement about current use of tobacco ($\kappa=0.79$) and alcohol ($\kappa=0.74$), and moderate agreement about current kava use ($\kappa=0.48$). There was universal agreement that there were no current petrol sniffers assessed but only fair agreement about those who had ever sniffed petrol ($\kappa=0.27$). Unpublished data from the same study used six images of male and female body size and showed fair agreement between health workers and self-reported assessments against these images ($\kappa=0.37$). When weighted for body-size

assessments that differed by just one category, good agreement was achieved ($\kappa=0.67$). Unpublished data from the same study also included measurements of body mass index (BMI) and a bio-electrical impedance analysis (BIA). BIA and BMI were significantly ($P<0.001$) associated with both health worker assessment ($F=16.45$ and $F=18.44$) and self-reported body size ($F=22.71$ and $F=31.46$).

We have argued elsewhere²⁶ that when health worker assessment is the only available data source in this difficult cross-cultural environment, i.e. when consensus classification must be relied upon as proxy for more directly-obtained data, we can do so with reasonable assurance. Finally, the method has the considerable advantage that the same data collection procedure can be applied systematically under repeatable conditions across a very diverse population.²⁶

Kava use and cardiac problems

The non-significant tendency for an association between kava use and IHD should be monitored, but there is no clear evidence for an association. It is possible that longer-term biological processes are still developing and perhaps an association between kava use and IHD would, in time, emerge. However, recent evidence from the cross-sectional study in one of the region's communities does not support this, since we found no association between serum levels of markers of potential thrombolytic processes or carotid arterial wall thickness among 101 study participants who had used kava for 1-18 years (median 12 years).¹⁸

Kava use may disrupt lipid metabolism^{14,18} but it is not clearly suggestive of any increased atheroma risk. In a study of possible causes of the characteristic rash among heavy kava users, Ruze³⁰ hypothesised that defects in cholesterol metabolism, and not niacin deficiencies, accounted for "acquired ichthyoses" similar to known reactions to cholesterol-lowering drugs. Ruze³⁰ emphasised that Mathews et al¹⁴ found increased HDL-cholesterol levels in heavy kava users. In a recent study we also observed a non-significant tendency for elevated HDL- along with LDL-cholesterol among kava users.¹⁸ These abnormalities increased with heavier use, but probably reversed upon

ceasing or moderating consumption.^{14,31} Antithrombotic effects of kavain (a kava lactone) on reducing aggregation in human platelets have been reported.¹⁶ Possible antithrombotic effects and elevated HDL-cholesterol¹⁴ imply the possibility of a myocardial protective effect of kava.

The acute effects of heavy use are not understood and further research is warranted. For example, kava's diuretic properties, never specifically measured but reported consistently in herbal medicine,³² make it plausible that those with established IHD could be at higher risk of cardiac events if they exercise while dehydrated from heavy kava use.¹³ An increased risk of myocardial ischaemia¹⁵ and sudden cardiac death¹³ among Aboriginal sportsmen could have been due to abnormal coagulation with enhanced thrombosis and/or the possibility of arrhythmia among recent heavy kava users.^{13,15} However, the evidence we have available for coagulation and thrombosis¹⁸ does not clearly support this suggestion.

Acute effects of kava may be mediated by neurological mechanisms. Kava's acute anaesthetic effects depress muscle action potentials.³³ And, it is possible that large doses of kava pyrones lead to abnormal atrioventricular function, perhaps caused by these same well-known muscle-relaxing properties.³⁴ It was recently reported that the actions of kava pyrones on voltage gated ion channels, with antagonistic effects on Na⁺ and Ca²⁺ currents and modulation of K⁺ currents, are of importance for their mood stabilisation properties and that these cellular actions show a large overlap with the actions of established mood stabilising drugs.³⁵ Given that the use of antipsychotic drugs is known to prolong QTc interval and bind to the potassium rectifier channel, and that their use may be associated with *torsades de pointes* and sudden cardiac death,³⁶ similar outcomes with kava use should be considered. The precise mechanisms for such a disastrous outcome are unclear given that there is preliminary evidence to suggest that kava extracts modify vagal control of heart rate in patients with generalised anxiety disorder¹⁷ implying a possible protective mechanism.

In conclusion, whether the chronic use of kava has harmful or protective effects, normal heart operation appears to be disrupted with kava use. Therefore it is plausible

that the risk of arrhythmia and a cardiac event may be higher in people with existing abnormalities in cardiac output or effective mechanical performance, as may be the case in many Aboriginal kava drinkers, especially with heavy exercise following kava use. Since these effects are likely to be dose-dependent, more precise determination of levels of consumption is also required. Ultimately, kava consumers will need to balance kava's desirable mood-altering qualities against these possible risks.

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CHAPTER 8

IMPLICATIONS FOR POLICY AND KAVA USE ELSEWHERE IN THE WORLD

INTRODUCTORY NOTES

Paper #9: published

Clough AR. Enough! or too much. What is excessive kava use in Arnhem Land? *Drug and Alcohol Review* 2002;22:45-53

Paper #10: in press

Clough AR, Jones PJ. Policy approaches to support local community control over kava supply and distribution in the Northern Territory (Australia) [in press]. *Drug and Alcohol Review*

Discussion based on the following published paper:

Currie BJ, Clough AR. Kava hepatotoxicity with Western herbal products: does it occur with traditional kava use? *Medical Journal of Australia* 2003; 178:421-422.

In response to wishes expressed by Aboriginal communities and after reviewing the possibilities for banning or controlling kava supply, the NT Government (with bipartisan support) decided to implement a system of controlled availability of kava in those communities where people wished to continue to use it. This led to legislation that simultaneously banned kava in the NT and established the environment where regulatory mechanisms could be designed to enable kava to be supplied with appropriate controls in those communities.

Recent amendments to the kava legislation have incorporated harm reduction principles which aim to encourage 'responsible' kava use. But there has been little information available until now that could be used to guide decisions on what is excessive kava use and what kava's health effects might be at different levels of consumption. Paper#9

attempts to address this gap by compiling existing information and incorporating new information that specifies, as far as practicable some of the health, social and economic effects of kava when it is consumed at different levels.

Paper#10 was invited as a result of paper#9. The journal requested an additional paper that provided details of the regulatory system, its history and its rationale. The paper documents approaches used in Aboriginal communities to control kava with respect to the new system and concludes by identifying risks to the regulatory system and its fragile nature. The regulatory system that has been developed reflects local needs and also the geographic and social diversity in Arnhem Land. If the rival illegal kava trade supplants the developing regulatory system health, social and economic impacts on Aboriginal communities may emerge once more.

Finally, a discussion included at the end of this chapter (page 8-62) attempts to address the issues confronting countries where kava has been an important item of export trade and the drug regulatory authorities in western countries, including Australia, where herbal remedies that include kava came to be marketed as complementary medicines especially during the 1990s. Cases of fulminant hepatic failure with some cases resulting in death, including one case recently reported in Australia, have led to the banning or restriction of products containing kava in a number of western countries. It has been reported that this has had a detrimental effect on the economies of Pacific island countries. This has also raised concerns for the hepatic toxicity of kava used in its traditional form in Pacific populations and in Indigenous Australians. The results of the studies reported in this thesis are incorporated into this discussion to try to inform this international controversy.

Enough! or too much. What is ‘excessive’ kava use in Arnhem Land?

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ABSTRACT

Objective: To describe parameters for use in monitoring health, social and economic effects of kava use in Arnhem Land Aboriginal communities in the Northern Territory (NT).

Setting: Kava has been used mainly in eight communities (population>200), and in smaller associated homeland areas since 1982 with a total population of approximately 6800.

Design: Cross-sectional description and comparison using data from three kava-using communities.

Study Procedures: Interview data combined with health worker assessments were compiled using: 1) a sample (n=136) aged 16-34 years in one community in 2001-02. 2) a sample (n=101) aged 16 years and over in 2000 where physical assessments and biochemical and haematological data were also collected. 3) participant-observation in one community (133 people aged 18 years and over) during 1989-91.

Results: Kava, supplied illegally, was still being used in Arnhem Land in 2001-02. In 2000 dermatopathy characteristic of heavy use, abnormally-low body mass index (BMI), low blood lymphocytes and abnormally-high γ -glutamyl transferase (GGT) occurred more frequently with increased kava use. These acute effects emerge at average consumption levels of from 310-440g/week of kava powder. When kava users in one community began to consume it at an average of 240-425g/week from mid-1990, 19% of available cash resources were spent on kava with 11% of cash resources leaving the local community economy. The proportion of men drinking kava reached 70% and women 62% from mid-1990 with 20% of the population spending unprecedented amounts of time (14+ hours/week) in activities where kava was consumed.

Discussion: These parameters may be useful to monitor kava's adverse health, social and economic effects. Their association with increased kava use suggests that

approaches to minimising harm from its abuse may fruitfully begin with controlling supply.

INTRODUCTION

Kava, the mood-altering drink prepared from crushed roots of the pepper plant (*Piper methysticum* Forst. f.), widely used in south Pacific countries on ceremonial occasions and in secular drinking,¹ was brought to Arnhem Land Aboriginal communities in the Northern Territory (NT) early in 1982.² Kava drinking was rapidly adopted in most coastal and island communities located across the NT's 'Top End' (Figure 9-1).

Kava soon became a challenge for policy,³ the object of public and institutional scrutiny that it remains today. Observers, service providers and regulatory authorities were unsettled by kava's coincidence with emerging community dysfunction soon after its introduction, concerns for health effects of 'excessive' use apparently without social controls, and economic hardships brought about by a vigorous informal, and later illegal, trade in the substance.⁴⁻⁸ This debate and commentary influenced policy and helped to engender both of the legislative attempts by the NT Government to control its supply and consumption; the first attempt in 1990,⁹ and the second in 1998.¹⁰

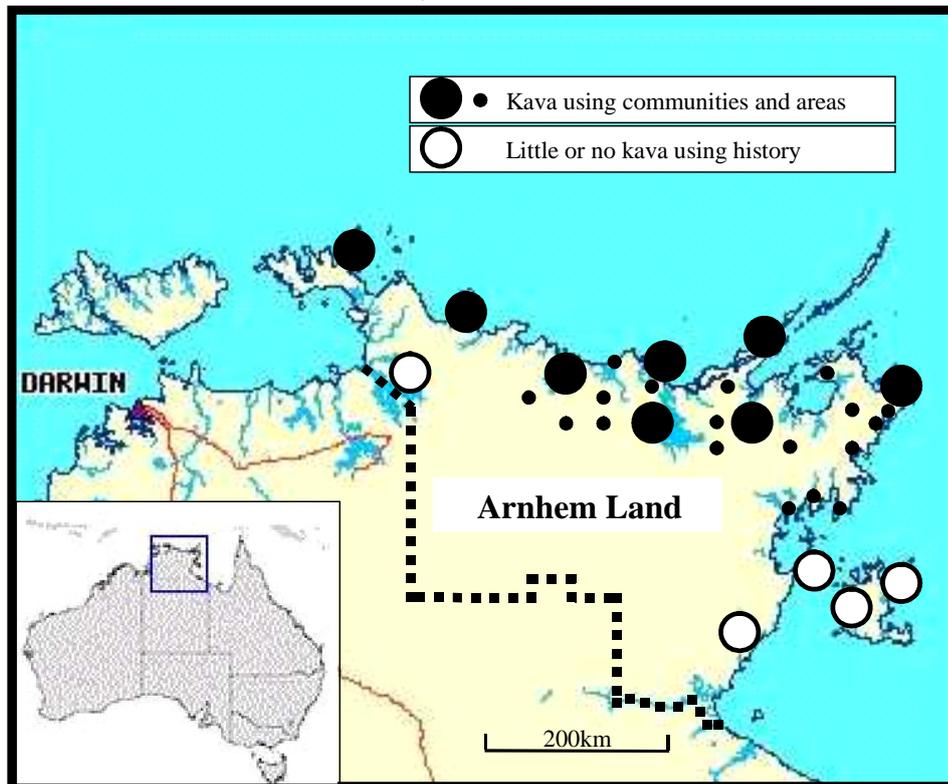
The explicit objectives of the recent legislation include harm minimisation,¹¹ and to encourage 'responsible' kava use under a licensing system which was implemented early in 2002. The aim of this paper is to describe 'excessive' use in terms of some known health effects along with aspects of kava's social and economic impacts to attempt to identify the harms that would be minimised.

In what way can the effects of using kava in Arnhem Land be described as 'responsible', 'excessive' or otherwise? How much kava is too much kava?

Firstly, data from a recent survey are presented that confirms that Aboriginal people in Arnhem Land continue to use kava. This is followed by a discussion of some of kava's health effects with increased kava use based on other recent studies and comparing these with results of previous studies. Finally, a detailed description is provided of social and economic changes that accompanied increased kava use in one community

after the first attempt to control kava in 1990. In the discussion and conclusions this information is combined to describe parameters that may be useful to

Figure 8-1. Kava using communities in Arnhem Land (NT, Australia)



monitor and manage the possible health, social and economic effects of the ongoing use of kava in this population.

Legislative efforts to control kava in the NT

A more complete description of the history of kava licensing will be the subject of a companion paper to the present one. To provide background information for the present paper, however, a brief summary history is included here.

Legal kava

The single wholesaler licensed to purchase and distribute kava in the NT was announced by the Chairman of the NT Licensing Commission on the 28th of March, 2001.¹² The wholesaler supplies kava, according to a wholesale management plan, to retailers who become licensed to sell kava to local people normally resident in approved licensed areas. Before kava came to be sold under the new system implemented in February 2002, four licensed areas were defined and retailing arrangements set in place. Management plans required for both wholesale and retail licensees included identification of strategies to minimise harm from excessive kava consumption, and the management of associated community issues.¹³

Twelve years earlier, in June 1990, the first management system was implemented under the NT's Consumer Protection and Fair Trade Act.¹⁴ For those communities who requested it of the NT Minister for Health and Community Services, kava could be sold only through the local elected community Council or other approved association. Sales were subject to an upper limit of 50g of kava powder per person per day (350g/week), sold only to persons aged 18 and over, and the kava sold obtained only from approved wholesalers with records kept of all transactions.¹⁵

Contradictions between the changing regulatory system of the Commonwealth with respect to kava and the objectives of the NT, from 1993 onwards destabilised the local licensing system and in 1994 it was dismantled.^{15,16} By 1995, however, regulatory realignment made it possible once again for the NT Government to consider legislating to control the sale, supply and possession of kava.¹⁵ On the 21st of November 1996 the Draft Kava Management Bill was tabled in the NT Legislative Assembly. Subsequently, during February-March of 1998, the NT Health Minister consulted with communities in person. Shortly afterwards, in June 1998, the Kava Management Act was enacted. The Minister, at several community meetings in Arnhem Land, undertook to implement a licensing system to enable those communities that wanted to continue to use kava to control its supply and consumption. At the same time, the Health Minister committed the Government to strong action to control illegal kava.

Illegal kava

During the regulatory hiatus from 1994 to early 1998, a large informal trade had emerged. The last available information based on supply records reported that the size of the kava trade in Arnhem Land was around 28 tonnes in 1992.^{15,16} The size of the kava trade in 1997, may have been between 27 and 36 tonnes and with a value of between \$6 million and \$8 million at known prices to consumers.¹⁷ At the end of 1999, the size of the, by this time illegal, trade was estimated to be 20 tonnes and worth about \$5 million. In 2000, the trade was probably around 15 tonnes and worth perhaps \$3.8 million.¹⁷

INFORMATION USED IN THIS PAPER

The information used here comes from three studies conducted in Arnhem Land communities at different times between the late 1980s through to the present. These include a recent survey of drug use among young people in eastern Arnhem Land, a cross-sectional study of kava's health effects in one eastern Arnhem Land community conducted in 2000¹⁸ and, a participant-observation study conducted during 1989-1990 in a western Arnhem Land community.¹⁹ Data on health effects and kava consumption patterns from a previous 1987 cross-sectional study²⁰ are used to compare with results of the second and third studies.

- **Recent data on kava use collected in eastern Arnhem Land 2001-2002 (n=136).**
Data is available from interviews about drug use recently conducted with individuals and key informants (local Aboriginal health workers) in an eastern Arnhem Land community. These data were collected prior to the implementation of Kava Management Plans, i.e. when the kava available was being supplied illegally. A random sample of people aged between 16 and 34 years was selected. Some of these folk (n=15) were interviewed and asked about their drug use, including kava use. For everyone selected in the random sample, including those interviewed, health workers independently assessed whether a person was currently using kava and whether they had ever used it.
- **Cross-sectional study of health effects of kava use in one community in eastern Arnhem Land in 2000 (n=101)**
Selected data from a recent cross-sectional study carried out in one community is used to describe kava's health effects.¹⁸ The community had a total population of 702 and of these 101 adults (approximately 30% of the population over 15 years of age, including 65 males and 36 females) participated in the study.¹⁸ In the results section, comparisons are made with a similar cross-sectional study carried out earlier, in 1987, by other researchers in another eastern Arnhem Land community of similar size (881 people).²⁰

- **Observations of social and economic effects of kava use in one community in western Arnhem Land, 1989-91**

The author conducted participant-observation research in this community from January 1989 to June 1991. The community, located on an island near the western Arnhem Land coast, then had a population of 229, including 133 people aged 18 years or more, normally resident there.²¹ Data from field notes and diaries, community Council records and kava supplier records from 30 months continuous residence in this community¹⁹ are used to describe kava's social and economic impacts and patterns of supply and consumption. These observations were made before and during the implementation of the first attempt to control kava supply in 1990.

Methods

The methods used to collect the data presented have already been described in published papers to which the reader is referred. These papers describe methods for assessing exposure to kava use from interviews and health worker assessments,²² participant observation¹⁹ and methods for measuring health outcomes reported in the cross-sectional study.¹⁸

Approvals

This series of studies received ethical approval from the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research.²² A memorandum of understanding negotiated for the cross-sectional study in the community concerned guided this research.¹⁸ The local Council approved access to community records for the participant-observation study and the relevant wholesaler provided kava supply data.¹⁹

RESULTS

Kava use in 2001-2002 (n=136)

Table 9-1 shows that kava (supplied illegally) was still being used in Arnhem Land recently. Kava use had declined from 1998 up to 2000.²³ However, its continuing use suggests that this decline may have stabilised. Anecdote supports the continued availability and use of kava in most of those communities where it was being used prior to 1998 despite recent attempts to eliminate illegal supplies. At the time of writing, the pattern of kava use may still be changing as legal kava becomes more widely available. For example, one locality is using legal kava exclusively, in others it is likely that only illegal kava continues to be available while in others both types are available. Monitoring is being implemented to try to document these changes.

Cross-sectional study of kava's health effects in one community in eastern Arnhem Land (2000) and comparisons with a previous study

This study¹⁸ found that, in males who did not use alcohol, kava users more frequently showed dermopathy characteristic of heavy kava use, and a lower body mass index (BMI). They also had increased levels of liver enzymes (γ -glutamyl transferase, GGT) while blood lymphocytes were decreased.

Results of the earlier (1987) pilot survey had also suggested similar health burdens for kava users in the nearby community in eastern Arnhem Land.²⁰ The two communities studied were similar in size with a similar kava using history. Anecdotal reports suggest that kava continues to be available in both.

The associations between kava use and signs of poor health found in both studies conducted 12 years apart in two similar communities lend considerable weight to their conclusions.^{18,20} The results of the two studies are summarised in Figures 9-3 and 9-4 which show the increased frequency of occurrence of abnormal values and the stronger

effect on health measures with increased kava use. Kava dermatopathy was found with greater frequency with increased kava use (Figure 9-3). A lower average BMI at levels below a normal reference range occurred in the earlier study amongst the ‘heavy’ and ‘very heavy’ users but to a slightly lesser degree in the recent study (Figure 9-4a). A higher average GGT occurred amongst these categories in the earlier study but GGT increased with kava use in both studies (Figure 9-4b). Figure 9-4c shows that even though blood lymphocyte levels in both studies were largely within but at the lower end of a normal range, the heavier kava users showed decreased levels especially in the recent study.

Table 8-1. Kava users in a random sample of people aged 16-34 years (n=136, 70 males and 66 females) from one community in eastern Arnhem Land (2001-02)

| | Age group (years) | | | |
|-------------------------------|--------------------|--------------------|--------------------|--------------------|
| | 16-19 (17m,17f) | 20-24 (17m,18f) | 25-29 (19m,18f) | 30-34 (17m,13f) |
| Kava – ever used | | | | |
| Males, % of sample | 24% | 41% | 47% | 71% |
| n= | 4 | 7 | 9 | 12 |
| Females, % of sample | 6% | 50% | 50% | 46% |
| n= | 1 | 9 | 9 | 6 |
| Kava – currently using | | | | |
| Males, % of sample | 12% | 35% | 47% | 71% |
| n= | 2 | 6 | 9 | 12 |
| Females, % of sample | - | 44% | 44% | 46% |
| n= | 0 | 8 | 8 | 6 |

Figure 8-2. Kava supplied to one community in western Arnhem Land, 1989-91

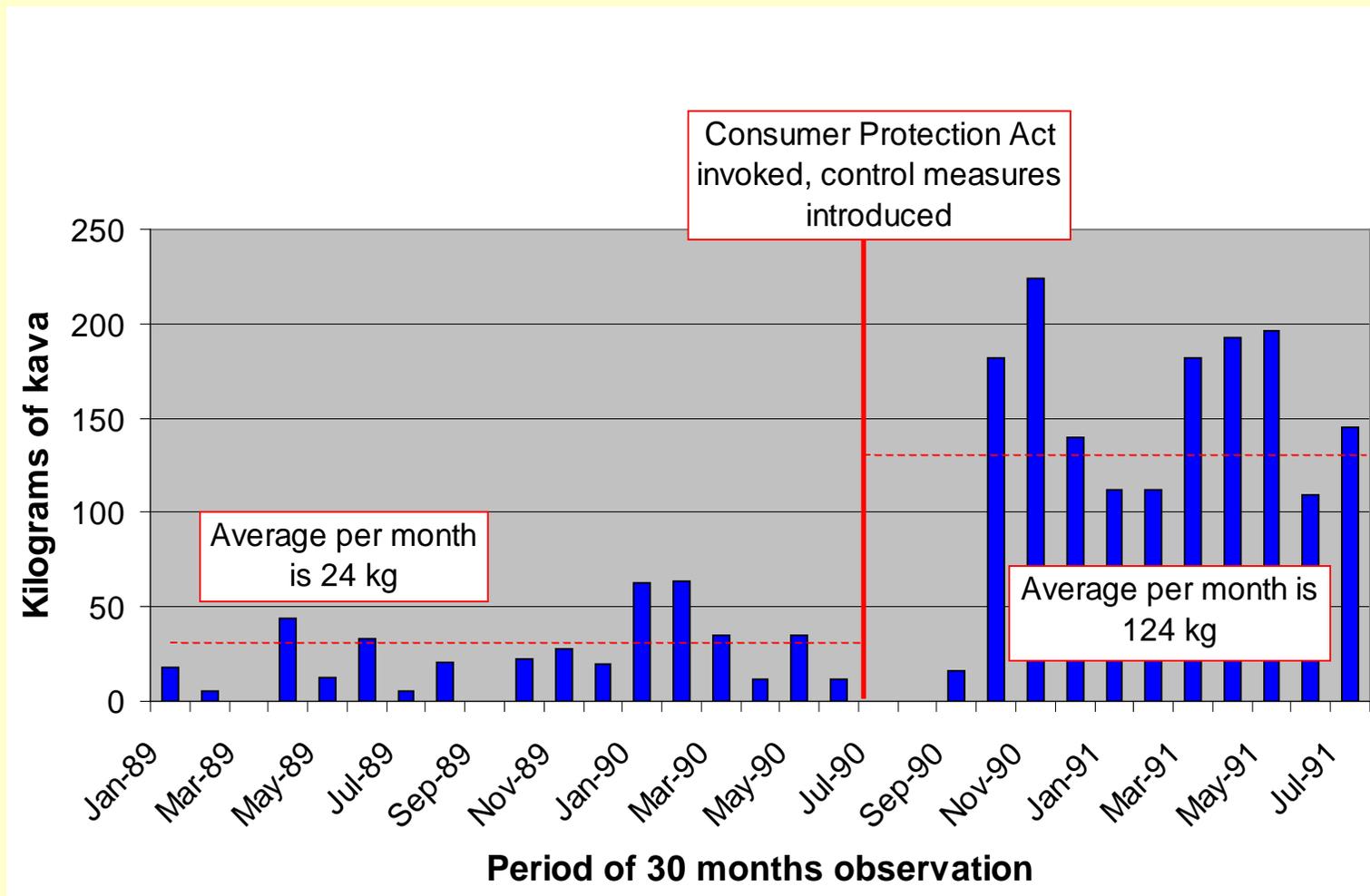


Figure 8-3. Two studies of kava use comparing skin rash

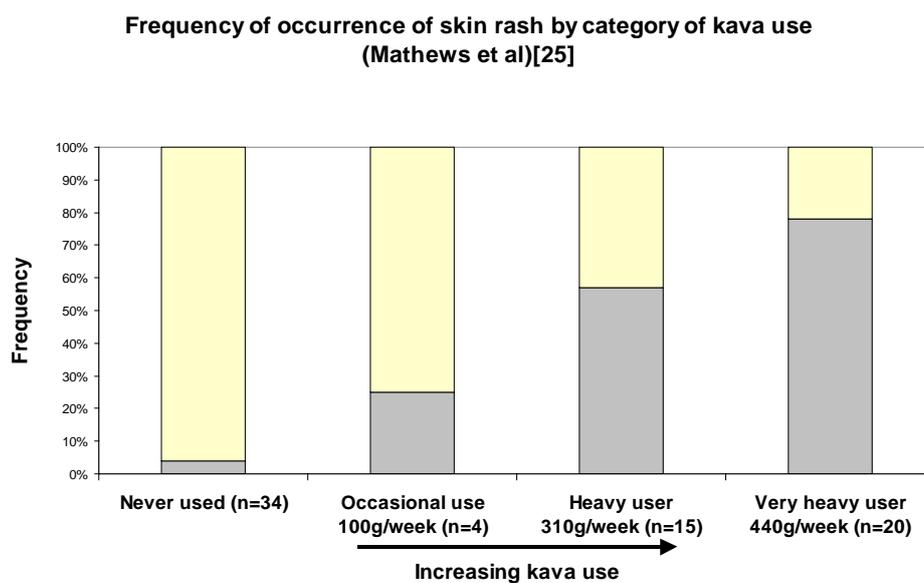
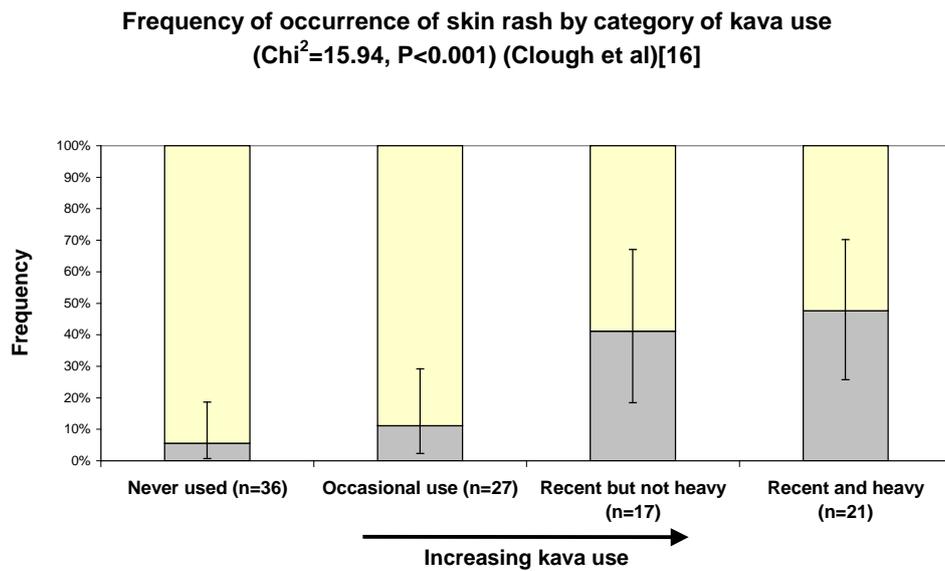
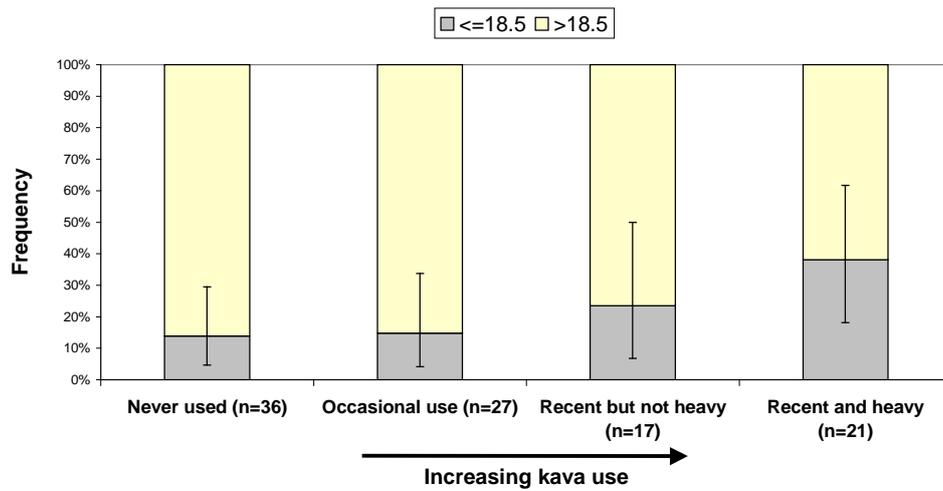


Figure 8-4a. Body mass index (kg/m²)

Frequency of occurrence of abnormal values of body mass index
by category of kava use (Chi² =3.99, P=0.046)[16]



Differences in body mass index by categories of kava use
(F=3.40, P=0.021)

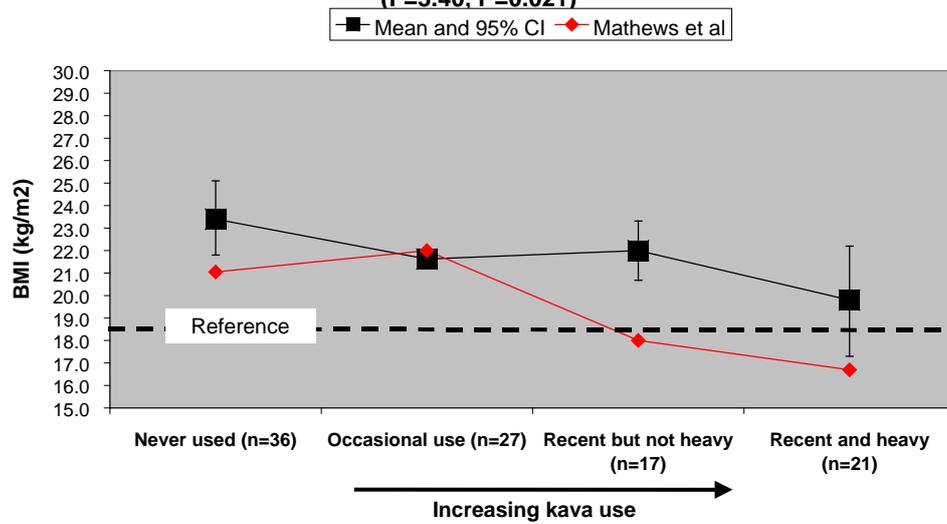
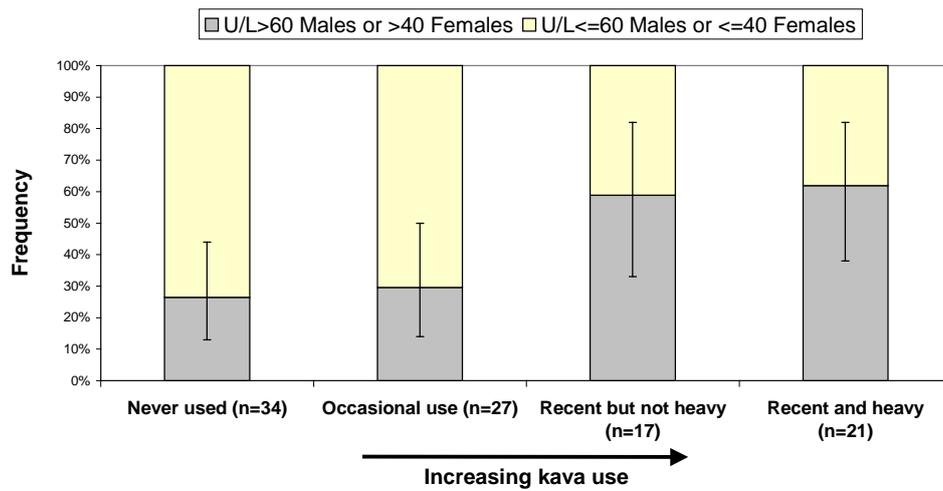


Figure 8-4b. Liver enzymes (GGT)

Frequency of occurrence of abnormal values of gamma-glutamyl transferase by category of kava use ($\chi^2=8.15$, $P=0.004$)[16]



Differences in gamma-glutamyl transferase by categories of kava use ($F=7.37$, $P<0.001$)

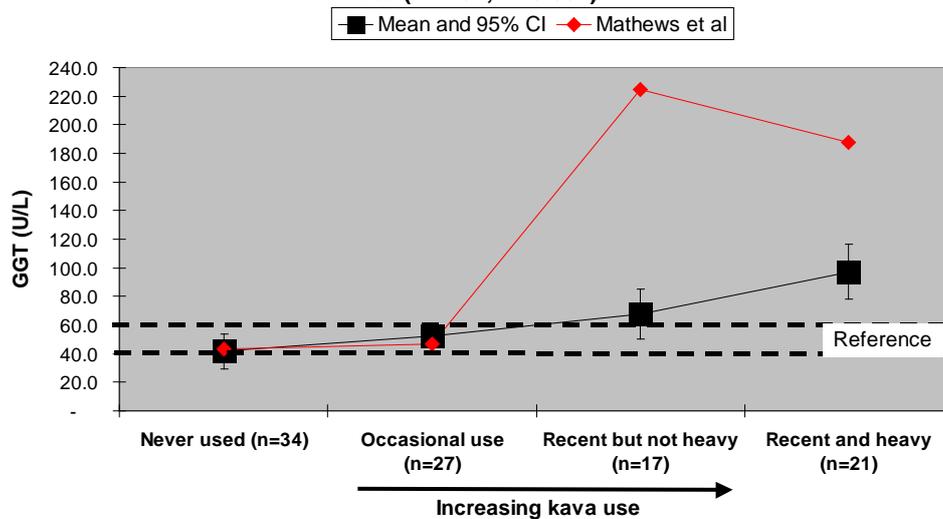
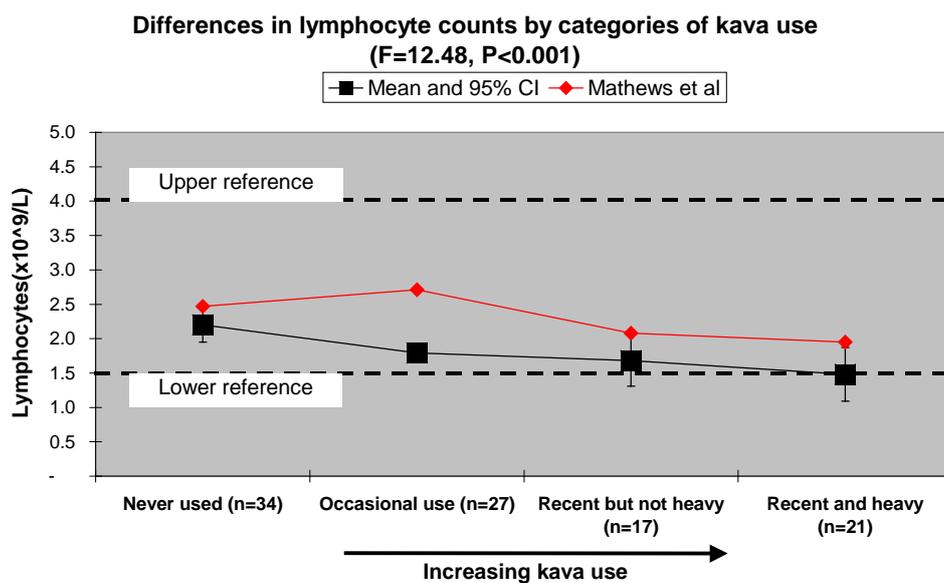
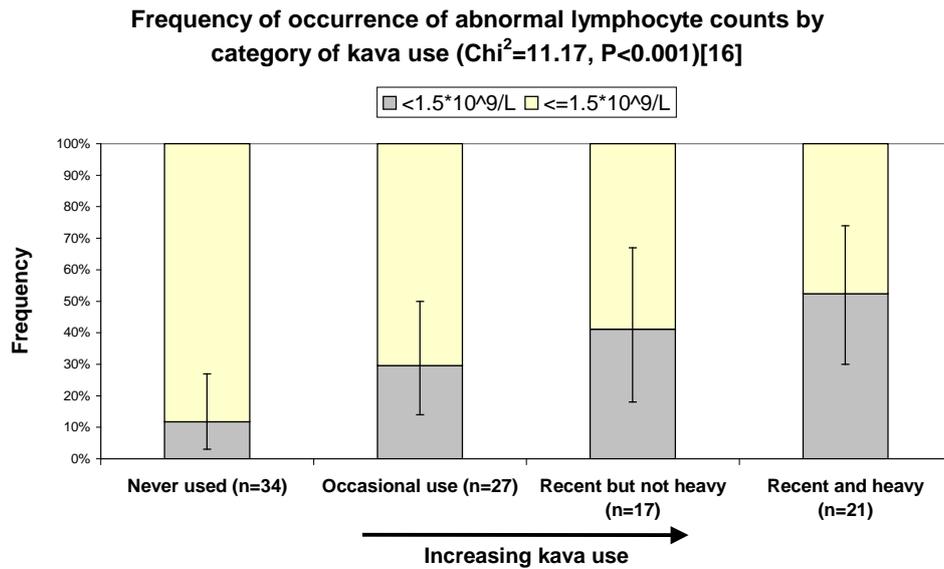


Figure 8-4c. Lymphocytes



Social and economic changes in one community in western Arnhem Land 1989-91

Between 1989-90 to 1990-91 there was a five-fold increase in the amount of kava used in this community (Figure 9-2).¹⁹ There were two main reasons for this large increase.

Firstly, the structure and rationale of the kava supply system changed between the two periods. In the earlier period, prior to regulation, one local man (or his brothers) purchased kava for local redistribution by sending cash in advance to a dealer in Darwin, the NT's capital city approximately 200km distant. Sometimes the kava purchased was exchanged in the community for cash, but more often it was quickly consumed within social networks closely affiliated with the buyer or it was distributed to other clan groups. Profits were often used to meet immediate family cash needs such as goods from the store, repairs and maintenance and fuel and tyres for a four-wheel drive vehicle; and cash for children at boarding school in Darwin. Setting aside some profit for the purchase of more kava rarely occurred. When the next supply of kava was to be ordered, considerable effort was usually required to gather together enough cash. The kava supply was accordingly irregular.

Additionally, under the first licensing regime, the local community Council became the official local supplier after 1990. The community Council established a committee that included some elected members and some representatives of the kava users in the community, including the man who managed kava sales before the regulations. The committee had a bank account but no funds. The first licensed shipment of kava was purchased using the council's creditor system and, after recovering the cost from sales of the initial order, it was possible for the kava committee to continue trading using recurrent profits without indebting the licensed association.

The management of cash and credit through the committee and the local Council made it possible for a regular supply of kava to be available and for broader interests in the community to benefit from the profits. All those people over the age of 18 years in the community who had the cash to purchase it and who wished to drink it could do so. Council records for the period January through May of 1990 indicate that around half of the profits from kava sales went to airline companies servicing the community.

Another 25% went to the local community store. The use of profits to purchase food and transport to support social, family and ceremonial requirements benefited a broader cross-section of the community, notwithstanding disagreements about the distribution of benefits. The supply of kava, and kava drinking, surged in September 1990 (Figure 9-2).

Changes in the patterns of kava use

Data on changes in consumption patterns reflect these changes (Table 9-2). It is informative to compare the data in Table 9-2 to patterns of consumption observed at about the same time in the community in eastern Arnhem Land where the earlier (1987) cross-sectional survey was conducted.²⁰ In this community, Mathews et al described 'occasional' (100g/week), 'heavy' (310g/week) and 'very heavy' (440g/week) kava use with extreme use at 900g/week.²⁰ In 1989-90 in the western Arnhem Land community, kava users as a group at that time could be described as 'occasional', but by 1990-91, they had become 'heavy users' overall (Table 9-2). The proportion of drinkers increased from just over 30% (34/112) to 66% (83/125) in this community. Men drinking kava nearly doubled over the period while women drinking increased five-fold. In addition, the time spent drinking kava increased dramatically with 21% (mostly men) spending more than 14 hours a week in kava drinking activities and drinking in excess of 425g/week a level comparable to the 'very heavy' category described by Mathews et al.²⁰ Average consumption amongst kava users grew to around 370g/week of kava powder in 1990-91 from a level of 145g/week in 1989-90 before the management regime.

Table 8-2. Patterns of kava consumption in one community in western Arnhem Land between 1989 and 1991

| Categories of kava consumers | | | | | | | |
|------------------------------|--------------|--------------------------------------|------------------------------------|-------------------------------------|---------------------------------------|----------------------------------|-------------------------------------|
| | Non drinkers | 2 hrs/week (up to 3.5 hrs)* or | 5 hrs/week (3.5-6.5 hrs)* or | 9 hrs/week (6.5-11.5 hrs)* or | 14 hrs/week (11.5-16.5 hrs)* or | 24 hour (>16 hrs/week)* or | Total drinkers + non drinkers |
| | Nil | <130g/week† | 130-240g/week† | 240-425g/week† | 425-610g/week† | >610g/week† | |
| 1989-90 | 78 70% | 14 13% | 20 18% | - | - | - | 112 100% |
| males | 32 54% | 10 17% | 17 29% | | | | 59 100% |
| females | 46 87% | 4 8% | 3 6% | | | | 53 100% |
| 1990-91 | 42 34% | 15 12% | 11 9% | 32 26% | 17 14% | 8 6% | 125 100% |
| males | 21 30% | 2 3% | 4 6% | 19 28% | 16 23% | 7 10% | 69 100% |
| females | 21 38% | 13 23% | 7 12% | 13 23% | 1 2% | 1 2% | 56 100% |

* Based on hours per week spent in the activity of drinking

† Based on levels of consumption of 34g/hr (1989-90) and 39g/hr (1990-91) [19]

Social and economic impacts

Community records from 1990-1991 showed that the gross cash income for local people was around \$655,000 and that there were 12 individuals in the workforce and 129 others most of whom received income support from either unemployment benefits, or pensions and child support. These estimates derived from fieldwork are in line with estimates derived from later census figures for 1996 that indicate that the community's gross cash income was around \$700,000.²¹

During the period of observation from 1990-91, the value of kava purchased from the wholesaler was \$73,300; a total of 1446kg delivered to the community at a wholesale price of \$50/kg. The known proceeds from sales for the year were at least \$52,200. The investment in kava by consumers, therefore, was at least \$125,500 (= \$73,300 + \$52,200) during 1990-91 with \$73,300 leaving the community economy to the benefit of the wholesaler. This represents 11% of people's income for 1990-91 with at least another 8% (\$52,200) of the profits concentrated or redistributed locally; at least 19% of the cash available to community members in all. By contrast, during 1989-90 the trade probably represented a cash drain of just 2% on the community's resources.

Between 1989 and 1991, the author observed or participated in 26 social gatherings where kava was consumed. Eleven of these gatherings were household groups at home or out hunting and kava was consumed as part of these activities; another eight involved elders conferring about ceremonial and related matters and four were major public funeral events at which kava was consumed. Kava was an adjunct to these activities and not their principal focus. There were no differences, between 1989-90 and 1990-91, in the range of activities in which kava drinking was included. The principal difference was, as reported earlier,¹⁹ more time was spent by more people in activities that involved drinking kava. Kava drinking, however, took place inside the same kinds of social settings (Table 9-2).

CONCLUSIONS AND RECOMMENDATIONS

It is ironic that the first attempt to control kava's sale and distribution created the conditions for the expansion of its use. Existing social controls on supply and consumption were probably marginalised at that time by the new, more formal management system and by the urge to generate profits. Further discussion of this prospect and its consequences is taken up in another paper. However, the unique observations made during the implementation of regulations in 1990 suggest that an approach to minimising harm from kava abuse may be fruitfully based on controlling its availability which in turn is likely to limit opportunities for excessive use. Reinforcing the need to focus attention on controlling supply is that, whether under the circumstances of regulation or without regulation of the trade, all kava supplied to the community studied in western Arnhem Land was usually consumed with expedience. This pattern of consumption of kava and other commodities has also been described in other Arnhem Land communities in the late 1980s.²⁴ In sum, this suggests that the more kava is available the more it is likely to be consumed and possibly abused. But how much kava is too much?

A level of consumption of kava from 310g/week and up to 440g/week is correctly regarded as a range of use where acute health effects can occur, as Figures 9-3 and 9-4 show. The results of the recent cross-sectional study indicate that these occur amongst more than 50% of the using group in the 'recent and heavy' use category with a steady increase of the effect with increased kava use (Figures 9-3 and 9-4).¹⁸ In the earlier study by Mathews et al, kava's effects on health measures increased markedly in the 'heavy use' category, i.e somewhere between 310g/week and up to 440g/week ('very heavy' use)²⁰ (Figures 9-3 and 9-4). Some point within this range then may be referred to as 'excessive' kava use in terms of these acute health outcomes.

Other studies have investigated kava's possible longer-term health effects. For instance, kava has been implicated in serious infectious disease²⁵ and sudden cardiac death in young Aboriginal sportsmen.²⁸ However, data not yet published shows that we cannot confidently assert that kava use is associated in the long term with increased risk of ischaemic heart disease morbidity and mortality nor pneumonia, [Clough et al, unpublished data]. Excessive use may be associated with unusual neurological episodes.²⁶ For example, there is evidence suggesting that the occurrence of *grand mal* seizures, probably related to kava toxicity and/or withdrawal, coincided closely with increased kava availability during 1994-97 when there were no constraints on kava supply.²⁷ However, kava use does not appear to produce long-term neurological effects.²⁹ There is no evidence of permanent liver damage in Aboriginal people who use it. The available information suggests that kava's health effects in Aboriginal kava drinkers are largely reversible including the effects on GGT.^{18,30} There is no evidence in kava users in Arnhem Land of serious liver failure as has been reported in Europe³¹⁻³³ in some people who have used commercial kava-based products.

Effects on community social and economic life began to emerge in the western Arnhem Land community when the median kava use climbed to within the range of from 240g/week to 425g/week (Table 9-2). The average consumption in this community was 370g/week in 1990-91 in 83 kava drinkers.¹⁹ The sales limit under the first management regime at the time was 350g/week.^{16,19} The participant observation data suggest that use of kava at this level is also likely to be reflected in the following social and economic features in a community.

- around two-thirds of the males and more than half the females in the community population drinking it (Table 9-2), and
- around 20% of the population spending 14 hours or more in a week in activities where it is consumed (Table 9-2), and
- 20% of the available cash in a community is used to purchase it.

In sum, combining these results, it is suggested that average kava consumption in a community from 240g/week and up to 440g/week is a level at which health and/or social effects may begin to appear. These simple parameters require further refinement. But they begin to quantify some of the harmful outcomes that may need to be addressed for the controlled use of kava in small community settings where local people have now taken on the responsibility for controlling kava under their own management plans and associated license conditions.

While it is necessary to closely monitor any adverse effects that may emerge in the long term, controlled use may allow people to have enough kava to meet their desire to use it but to avoid adverse effects by moderating their consumption below the range of 240-440g/week. Health promotion programs may benefit from the inclusion of this kind of message.

How much is too much is one issue, and many people already have experience of extreme kava use during the mid-1990s as the informal kava trade expanded with few controls. On the other hand, how much kava is enough to maintain a stable legal trade managed by local community organisations must be balanced not only against prevalence and use levels and likely health, social and economic effects but also against the persisting threat of a rival and aggressive illegal trade.

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**Policy approaches to support local community
control over kava supply and distribution in the
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ABSTRACT

Health consequences of kava abuse in Arnhem Land Aboriginal populations (Northern Territory, NT, Australia) and the persistence of an illegal kava trade, with scarce cash resources used to purchase it and few benefits returned to communities that use kava, have been a cause for concern for 20 years. Despite these concerns, some Arnhem Land groups seek to continue using kava and to control its sale and distribution and profits from the enterprise. In response, policy makers in the NT have embraced principles of harm reduction and created regulatory mechanisms to address broader public concerns and to support local management of kava supply while reinforcing control over the consequences of its use. This paper describes the kava regulatory system now being implemented in the NT which features kava management plans that specify how kava is to be controlled. Kava management plans were developed in consultation between Aboriginal communities and licensing authorities. Pricing of kava and licensees' use of profits are negotiated through the licensing process. The lucrative illegal trade continues to threaten the regulatory system. The illegal trade and community responses to it are described. Evidence presented shows that some Aboriginal groups have consistently tried to control kava since it was introduced in 1982 for community benefit, in part to reduce health problems, but also to permit the enjoyment of a mood-altering substance that did not have the harmful effects of heavy alcohol use and because cash could be generated from kava sales. The new regulatory system holds the prospect for these efforts to be realised. However, continued support is required to enable those Aboriginal organisations now licensed to trade in kava to achieve the objectives of their kava management plans within a unique regulatory system.

INTRODUCTION

Kava

A companion paper to this described the principal health, social and economic harms from kava abuse in Arnhem Land Aboriginal communities and proposed parameters for monitoring its impacts.¹ Kava (*Piper methysticum* Forst. f.), used in south Pacific societies for its mood-altering qualities,² has been used in Arnhem Land Aboriginal communities since early in 1982.³ Kava abuse and profiteering from its trade remain significant public health issues in Arnhem Land. To try to control kava sales and consumption, two legislative attempts by the NT Government were implemented; firstly in 1990⁴ and again in 1998 with the passage of the *Kava Management Act*.⁵ This paper describes the origins and rationale for the more recent regulatory system currently being implemented.

While this regulatory system was being developed in the NT, kava became internationally controversial because, from 1998, evidence began to appear in the literature suggesting that the use of manufactured products based on kava is associated with serious irreversible liver damage with medical alerts issued or bans on kava products imposed in Europe,⁶ north America⁷ and Australasia.⁸ Aboriginal people have consumed kava as dried powder mixed with water;⁹ not as manufactured tablets, capsules or extracts. No evidence for serious liver damage in kava users in Arnhem Land has emerged,¹⁰ however, this controversy has made for greater urgency to control kava use and to monitor its health effects.

The first part of the paper describes the implementation of the current regulations following the *Kava Management Act* in May, 1998.¹¹ Highlighted in this discussion are the kava management plans being established in kava retailing communities.

The second part relates to 1994-1998, when the 'black market' trade in kava consolidated its control over kava supply.¹² The nature and scope of this trade and some community responses to it are described.

From 1982-1990, informal supply networks were established and kava use was consolidated with no regulation.^{13, 14} Then, from 1990-1993, the first attempt to regulate kava operated.¹⁴ Evidence presented in the third part of the paper highlights the congruence between regulatory mechanisms being implemented through kava management plans and local mechanisms that Aboriginal people used to try to control kava from 1982-1993.

Finally, in the discussion section, possible risks to the stability of the new regulatory system are outlined.

INFORMATION USED IN THIS PAPER

The authors have observed kava use and related social developments in Arnhem Land communities for 13 years (AC) and 19 years (PJJ) respectively in community development and support roles in the fields of local government (AC) and education (PJJ). One of us (AC) also conducted formal studies of kava's health and social impacts from 1989-1991 and also from 1998 to the present. The other (PJJ) is the Project Officer for Kava Licensing in the NT and has been directly involved in consultations to develop the current regulatory system since 2000. Insights presented here are derived from this combined experience. Our method is based on understandings of local situations developed in ongoing consultations with government and non-government agencies, community stakeholders and local Aboriginal groups. It recognises that in Indigenous Australian societies local social order is more often sought through self-regulation and consensus and less from the coercive influence of the state.¹⁵ Understanding how day-to-day activities are to be arranged and what constitutes acceptable or unacceptable forms of conduct or regulation helped to negotiate a realistic and workable regulatory system containing the peculiar features required in each community and supported by the power of NT Government law. No information pertaining to any community is used here other than that which is already publicly available.

Setting

By 2001, the eight major communities (200-1500 people) and associated homelands (up to 100 people) and their associated family outstations that have a significant kava-using history in Arnhem Land included approximately 7700 people.¹⁶ They are located along the 'Top End' coast of the NT (see locality map in the companion paper¹). Kava continues to be used in most of these communities today.¹⁷ Some communities are clearly associated with an area incorporating the immediate environs of the settlement, whereas other smaller communities are affiliated with an Aboriginal organisation that has service responsibilities

to a group of homeland localities scattered over a much wider area. These people comprise 11 major language groups¹⁸ with land ownership and rights vested in patrilineal descent groups.¹⁹ Traditional Indigenous cultural practices coexist with contemporary administrative structures responsible for management of local community services and infrastructure.²⁰ This demographic and social diversity poses unusual challenges for management of all kinds of community matters including kava.

CURRENT ARRANGEMENTS FOR THE REGULATION OF THE SALE AND SUPPLY OF KAVA IN THE NT

On 21 November 1996, the NT Attorney General and Minister for Health tabled in the NT Legislative Assembly a draft *Kava Management Bill* and at the same time moved that the broader issue of kava regulation be referred to the Assembly's Sessional Committee on Use and Abuse of Alcohol by the Community.²¹ Included in the terms of reference was the requirement that the Committee should consult with relevant stakeholders and make recommendations as to the desirability and extent of kava availability in the NT. As a result the *Draft report on the inquiry into the issue of kava regulation*, was prepared by Menzies School of Health Research in 1997,¹² and this became the principle resource document to develop the current kava regulation scheme. The draft report considered an enforcement model based on a complete ban meaning that responsibility for kava control would rest entirely with NT Police. This approach was not recommended since it had little community or agency support. Instead the report recommended a system of controlled availability, with local control over the distribution of profits to communities with provision for funding education, research and evaluation.¹²

In May 1998, the *Kava Management Act* was implemented making the possession and supply of more than 2kg of kava illegal in the NT unless in accordance with a licence issued by the NT Licensing Commission. In October 2000, the NT Government, with bipartisan Parliamentary support, chose to make explicit a commitment to harm reduction principles by amending the Act's long title thus. "*An Act to prohibit the cultivation, manufacture, production, possession and supply of kava, to encourage responsible practices and procedures in relation to the possession, supply and consumption of kava and for related purposes.*"²² Further amendments to the objects of the Act were recently introduced to the NT Parliament to specifically encourage communities to participate in controlling kava availability.²³ While 'controlled availability' and 'responsible practices and procedures' for example require precise definition, these amendments set overall objectives for the new regulatory system.

The major mechanism within the licensing framework designed to give effect to these objectives and to Aboriginal peoples' wishes to control kava is the requirement for kava management plans. Under Section 58 of the Act, a kava management plan must be approved by the NT Licensing Commission before it can recommend to the Minister that a kava licensed area be declared.¹¹ Kava management plans approved by the Licensing Commission may vary but are required to address main headings including: boundary of the licensed area, any locations within the boundary from which the possession of kava should be excluded, times and place of sale, purchase limits, community expectations or rules and actions to monitor and modify kava's negative impacts.²⁴ The views of the NT Department of Health and Community Services and NT Police are formally considered as part of this process.

The successful applicant for the wholesale licence to purchase and distribute kava in the NT was announced by the Chairman of the NT Licensing Commission on the 28th of March, 2001.²⁵ Since May 2002, the wholesale licensee has supplied kava to three

retailers in the region who are each licensed to sell it in their respective licensed areas in accord with each area’s management plan (Table 9-3).²⁶

Table 8-3. Kava in Arnhem Land: summary of current status of kava licensed areas and licensees at 1 March, 2003

| Licensees | Licensed area declared | Date declared | Licensed to sell kava | | Licence issue date |
|---|--------------------------------|----------------------|------------------------------|-----------|---------------------------|
| Ganybu Wholesalers | Licensed wholesaler for the NT | Not applicable | To licensed kava retailers | Wholesale | Jan 2002 |
| Laynhapuy Homelands | Yes | Nov 2001 | To community residents | Retail | Jan 2002 |
| Yirkala Dhanbul Community Association Inc | Yes | Nov 2001 | To community residents | Retail | Feb 2002 |
| Warruwi Community Inc | Yes | Nov 2001 | To community residents | Retail | Mar 2002 |
| Ramingining Community Inc | Yes | Nov 2001 | No licence to sell kava | - | - |

These three communities include around 1500 people or approximately 19% of the population in communities that have had a significant history of kava use. Around 53% of males and 11%-27% of females may be drinking kava in these communities,¹⁷ and so there could be 280-330 people aged over 15 years drinking it in three licensed areas at present.

Before kava can be sold in a licensed area, retailing arrangements must also be approved by the NT Licensing Commission including selling price and proposals for the use of

profits for community benefit.²⁶ At the time of writing, retailing arrangements were still being negotiated in the fourth licensed area. The wholesale licensee and three retail licensees are each local Aboriginal community-based organisations with elected committees and executives, or trading entities of those organisations, with their business facilities situated in the communities they service.²⁶ Two communities where kava has been used have chosen not to seek the declaration of a licensed area while others are considering their position.

Two of the four kava management plans have proposed to allow kava sales from Monday to Saturday with trading restricted to the afternoon or evening hours.²⁶ The other two proposed to trade on specific days during the working week avoiding trading on a pay day or on days when social security payments are made. Three proposed to maintain a community register of kava users and all four proposed daily sales limits of 400g/person (2x200g packets) and weekly sales limits of 800g/person. Wholesale and retail licence applicants in their applications were asked to take into account information in the companion paper to this which suggested that health and social effects begin to emerge in Aboriginal communities when kava users consume an average of from 240g-440g/week of kava powder.¹

To monitor and modify kava's impacts, licensees proposed to involve key community-based service agencies including the community school, store, health centre and council operations. Approaches to discourage kava abuse also varied. In one community, a community forum approach for public discussion of kava misuse issues was proposed. The remaining three all opted to counsel heavy kava users in a manner compatible with family or clan structures. All licensees emphasised the need for local health service involvement for clinical surveillance and possible treatment and for the community store to alert the licensee to changes in store turnover and purchasing patterns.

As part of licensing approval processes applicants were requested to provide details of programs and/or activities they would fund to contribute to responsible kava retailing and

consumption and to community amenity. This criterion had the effect of encouraging applicants to allocate funding to support education, monitoring and community development projects. The successful wholesale licensee allocated 100% of kava profits to these kinds of activities. While retail licensees will allocate profits using local decision-making structures, all three have proposed their own local monitoring and education programs to ensure that kava is not abused.

Decision-making structures vary. In one, the local Community Council is the decision-making body. In another, a Council sub-committee of key community people, a renewed version of a previous 'kava social club',¹² will deal with kava issues and decide on allocation of profits for community benefit. In the third, local clan structures are being used to allocate profits in smaller isolated homelands. This variation parallels the different management infrastructure available to the retailers which ranges from full support of a financial accounting infrastructure to one with almost no infrastructure at all. In the latter licensed area, the work of managing kava purchases and sales is done entirely by hand in an environment remote from normal communications services available in larger communities.

All licensees expect that those who flout licence conditions will be reported for investigation by Police or Licensing Inspectors. One licensee has stipulated that if kava consumption interferes with traditional cultural practices such as ceremonies and mortuary rites, the kava committee can take remedial action that it deems compatible with customary law. Other controls embodied in license conditions include such features as: the exclusion of sites of special historical or public significance inside the licensed area; driving motor vehicles whilst under the influence of kava; and where the local store is owned by the community, requiring that people spend one-third of the value of a cheque cashed by the store on food. Such peculiarly local regulations about kava are feasible within kava management plans.²⁶ The licensing system gives these apparently humble rules legal effect since they form part of the Ministerial declaration of the licensed area and also the licence

conditions under which the retail licensee is obliged to operate and since they are approved by the NT Licensing Commission in accordance with the *Kava Management Act*.

The consultation process to develop these mechanisms was lengthy. It began in February 1998, when the NT Minister for Health consulted in person with major kava-using communities. At six large community meetings, the Minister advised that his Government would first make kava illegal and then, in those communities where people wanted to continue using it, Government officers would consult about how this could be done in accordance with their wishes and with NT law.

Following completion of the Minister's consultations, the *Kava Management Act* became operational in May 1998. Consultations with communities that had expressed a wish to continue to use kava were continued from March 2000. During 2001, a total of 21 consultation visits were made to six communities that were considering kava licensing. In those communities who wanted to proceed, the next step involved outlining the boundaries of a licensed area. This part of the process provided the opportunity for landowners in localities within proposed licensed areas who did not wish to be part of the local licensing scheme to ensure their country was not included. Further deliberations at several community meetings involved internal discussions among community organisations and community leaders. In six months this process yielded firm decisions about clearly defined boundaries of the four licensed areas and these were communicated to the NT Licensing Commission. The next part of the process required that the relevant community organisation develop a management plan for kava in their licensed area. This step was facilitated having already been set in train through consultation for each licensed area.

There was a change of Government in the NT in August, 2001, but bipartisan support for kava regulation continued. In November 2001, four kava licensed areas were declared by the Minister for Racing, Gaming and Licensing based on management plans approved by the Licensing Commission.²⁷ Having established licensed areas, the wholesale licence was

issued in January 2002 (Table 9-3) with the first legal kava supplied to retailers in May 2002.

In summary, this system to manage kava has been developed to provide a legal instrument with regulations and licensing conditions created by local people to suit their particular geographical and social circumstances. At the same time kava remains illegal outside the licensed areas, viz. the rest of the NT. The regulations have the force of law with transgressors losing vehicles conveying illegal kava and risking prosecution.²⁸ It provides for monopoly control of the trade by local Aboriginal community organisations and for return of benefits from the trade to support community development and community maintenance initiatives consistent with each community's objectives. Finally, it offers the prospect for controlling supply and making kava available under conditions specified by the local community. In the following sections, evidence is presented to demonstrate the congruence between the mechanisms employed in these regulations and some earlier efforts of communities to control kava themselves. Firstly, a description of the illegal kava trade and responses to it is provided since this is a key concern for the regulatory system.

AN ILLEGAL TRADE IN KAVA AND COMMUNITY RESPONSES TO IT

Illegal kava

When regulations were introduced to control kava in 1990, kava supply to Arnhem Land communities as a whole dropped from 23 tonnes in 1989 to 18 tonnes in 1990.^{1, 14} But within six months of implementing controls, most kava using communities had arranged for legal supplies by requesting to do so to the NT Minister for Health and Community Services.^{14, 29} By 1993, kava supply had climbed to unprecedented levels; 28 tonnes a year.¹⁴ Although there were attempts to develop an illegal trade by circumventing the licensed suppliers during this time, most kava was supplied by two licensed importers, one a community-based enterprise in eastern Arnhem Land and the other a proprietary limited company located outside Arnhem Land but still in the NT.¹⁴

Contradictions between the Commonwealth's and the NT Government's regulatory approaches that emerged from 1990 assisted to nullify the first NT licensing system and later afforded unlicensed kava importers the opportunity to import kava at little risk of prosecution or competition after March 1994.¹² Unfortunately, this occurred when demand for kava was the highest then known. With demand so strong, an unregulated trade quickly became established and then flourished in subsequent years.¹² Proceeds from this lucrative trade were repatriated to beneficiaries from outside Arnhem Land and the NT or were concentrated into the hands of a few local agents.¹²

Because of the illicit and stigmatised nature of the illegal trade, accurate information about its structure and operations since 1993 remains scarce. One of the authors (AC) was told in early 1998 by one trader while discussing the imminent kava regulations that "*Kava tells us who we are. Nothing can stop the kava. We will just go underground.*" This illustrates the determination of those involved in the trade. At about the same time (late 1997) it was estimated that the kava trade in Arnhem Land amounted to 27-36 tonnes a year with a

retail value of over \$6 million at then known prices to consumers (\$230-\$270/kg).³⁰ The Alcohol and Other Drugs Section of the NT government health agency reported that kava use had declined up to the end of 1998 but, by 1999, it was being consumed in most communities where it had been used prior to implementation of the Act.³¹ At the end of 1999, the illegal trade was perhaps 20 tonnes and worth about \$5 million and in 2000, was around 15 tonnes and worth perhaps \$3.8 million.³⁰ The recent (January, 2003) seizure of a single shipment of 776kg of kava,^{32,33} worth approximately \$180,000-\$210,000, suggests that the illegal kava trade threatens to remain a heavy economic burden. More comprehensive estimates of the overall size of the illegal trade are not possible. Prices in the legal trade are currently set through the licence approval process at \$140/kg i.e. 40%-50% lower than illegal prices, suggesting that the legal trade holds the potential to alleviate some of this large financial drain.

In the illegal trade, kava is supplied to selling agents located with bases in towns in or near Arnhem Land and then sold to agents living in or near kava-using communities. At this level kava may be sold by people who have contributed cash to a pool to commission the delivery of kava from either a supplier based in the NT or even interstate. When kava is supplied to consumers it may be in exchange for cash or it may be given or shared amongst family and community members along lines of social and family obligations.

From May 1998 to the end of June 2002, the total amount of illegal kava seized in almost 100 successful policing operations was around five tonnes and this had reached 5.8 tonnes by February 2003.²⁸ Thirty-seven of these seizures were of 'commercial' quantities (more than 25kg) with a retail value of at least \$6000 and potentially attracting the most serious penalties (imprisonment for eight years) under the Act.¹¹ Since 1998, two-thirds of the total amount of kava seized was discovered within or entering Arnhem Land (NT Licensing Commission, unpublished data). While the amounts seized represent just a small proportion (around 5%) of the illegal market, anecdote suggests that policing of the Act has assisted to bring about a reduction in illegal kava supply. It is too soon to tell whether the licensed kava trade has assisted to bring about a decrease in the demand for illegal kava

as well. Despite the decline, the illegal trade persists; it remains vigorous and is supported by an intransigent group of dealers that have strong local affiliations along with interstate and international connections.

Community attempts to control illegal kava supply

Although the illegal trade has been sustained by demand for kava among Aboriginal kava drinkers, during the 1990s, some communities rallied support from within to try to ban or stigmatise its use or to restrict its availability.^{14, 34} Reports of unusual clinical presentations for 'fits' or 'seizures',^{35, 36} the general poor health of the population^{37, 38} and observations of weight loss and skin changes alarmed community leaders in some parts of Arnhem Land.¹² One community Council responded during 1996-97 by exerting pressure on local dealers. Council resolutions were passed implementing workplace rules against its sale by Council employees and community residents. Tenancy regulations were used to try to evict dealers, and kava users in the community were counseled to try to reduce overall demand. Information about cash earned from local kava sales by one supplier operating from Sydney was provided to the Australian Tax Office for investigation. These skirmishes served to disrupt supply temporarily, but with no strong legal instrument pertaining to kava itself, no lasting impact was made on kava availability in that place. The size of the kava trade and its easy availability afforded by entrenched local traders overwhelmed this community's efforts, illustrating once again the determination of those involved.

Another community led by a strong and charismatic elder in eastern Arnhem Land had greater success. A trade in kava was becoming established for the first time in his community in 1997. In response, the elder demanded that kava use cease and in the process stigmatised its use by suggesting that the dried, cracking skin appearing in kava users looked like the crust of dried algae that appears on rocks near the shore at low tide, simultaneously alluding to the inertness and lethargy of kava users. Still today this community has no regular access to kava nor is it seeking to become a licensed area.

CHANGING CONTEXT OF KAVA USE SINCE THE 1980's

While efforts to control kava supply may not have received unanimous support from within, it is clear that the will to try to control the illegal kava trade has remained, and has emerged from time to time, in some kava using communities. We now present information which demonstrates that both the intent and capacity has existed to manage kava supply along with the distribution of profits for local community benefit. The role that kava use has played in community life is also illustrated in this section by reference to the way in which kava use and alcohol use have interacted since the early 1980s when kava was first introduced. This helps to clarify some of the original rationale for kava use and how this rationale is compatible with the new regulatory system.

Local community enterprises

At the end of 1981, the first of several visits to Fiji to inspect village development projects was undertaken by Aboriginal people from one community in eastern Arnhem Land. Uniting Church personnel working in Arnhem Land communities, themselves Fijian, facilitated these visits,³⁹ and this partly accounts for the fact that kava drinking was taken up mainly in those communities that had a Uniting Church affiliation.⁴⁰ After returning home, community people arranged kava supplies for their own local use.³⁹ At first, kava supplies were purchased and distributed by community members or employees of local community organisations and the modest profits were used mainly to provide transport services.^{39, 41} Kava was purchased from a Sydney wholesaler.³⁹ The demand for kava quickly grew locally and so did its regional scope (see Figure 9-1 in the companion paper to this¹), well beyond the capacity of the local community organisations to service and manage. By 1985, it was being used in most of the communities that today have a history of ever having used kava.¹³ Expansion of kava use was also facilitated by European traders who were already selling general merchandise operating from vessels visiting communities along the 'Top End' coast and who assisted to meet this rising demand.^{12, 14}

They too initially purchased it from interstate wholesalers. In response, the Aboriginal community organisations that originally took up supplying kava sought to limit supplies by requesting that their wholesaler sell exclusively to them and by urging traders to stop selling kava. However, the wholesaler and the European traders had neither the regulatory compunction nor was there any benefit to their unregulated enterprise in doing this, so the trade continued to expand rapidly during the 1980s. The possibility for monopoly control by local community organisations over the wholesale supply of kava was thus short-lived, and very early, kava supply became determined as much by commercial imperatives as community needs.

The companion paper to this describes some social and economic impacts of kava in one community between 1989 and 1991.¹ A committee of the local community Council managed kava supply and the distribution of the proceeds from its sale from 1990 onwards with a broad cross-section of the community sharing in the cash benefits. The kava committee supported cultural activities largely by supplying transport and food to participants in traditional ceremonies.

In other communities, similar committees also invested kava profits in ceremonial activities although few details of amounts involved are now available. In one in western Arnhem Land, a kava social club operated which supported sport and recreation in addition to traditional cultural activities.¹⁴ This committee is the same committee recently re-established, including some original members, to manage kava issues under the new regulatory system and described in the previous section. Another community in eastern Arnhem Land used part of kava profits to support a housing maintenance program.⁴² This particular community is still considering its current approach to kava under the new regulatory framework.

Kava used to control alcohol abuse during the 1980s

When people first brought kava to Arnhem Land, they sought to enjoy its mood-altering qualities, partly in the hope that the harmful effects of emerging patterns of alcohol abuse in their communities could be reduced. This was described in several reports in the early 1980s.^{39-41, 43-45}

These events occurred during a period of accelerated social and economic change in people's lives in Arnhem Land. In eastern Arnhem Land, during the 1970s, a bauxite mine and processing plant were developed on the Gove Peninsular along with a residential and service centre where around 3350 non-Aboriginal people now reside.⁴⁶ The response to these developments⁴⁷ had already become a focus for a national land rights movement during the 1970s, so people in the area have a long history of dealing with significant social and economic issues besides kava.⁴⁸ During the 1970s the regular availability of alcohol from liquor outlets in the service centre, a service which local people were struggling to deal with for the first time, had become a significant and immediate challenge.⁴⁹⁻⁵¹ It is interesting to note that the liquor licence application for the first public house in the service centre was unsuccessfully opposed in 1970 by sections of the same Aboriginal group that first brought kava to Arnhem Land 12 years later.^{3, 49, 51}

The control of alcohol issues, however, today remains a perennial problem in this community and others nearby. Yet, at the same time, the view that kava is a challenge to alcohol continues to be held. For example, kava is seen by some elders as an important adjunct to traditional ceremonies and as a way of maintaining enthusiasm for participation while also reducing the likelihood of having participants in the more secret aspects of ritual dangerously uninhibited by alcohol. There are few published reports that can assist us to understand the social context of kava use, especially with respect to alcohol during the 1980s and 1990s. Anthropological literature on Arnhem Land societies says little about kava or its social effects. Fieldwork for the more significant ethnographies was mostly carried out prior to kava's arrival.^{46, 52-55} One study reported diverse opinions about kava use along with optimism for its controlled consumption as an alternative to alcohol and for

the local use of proceeds from its sale.⁵² Another study in a kava using community where alcohol was already periodically available, reported that kava was easily incorporated into daily life.⁵⁴ This same community opted for no kava in 1990¹⁴ but its population began to use it heavily from the middle of 1996 combined with continued access to alcohol.¹²

When it was first introduced to eastern Arnhem Land, kava appeared to offer the alternative to alcohol people were seeking.^{39, 56} Sanctioned by the Uniting Church, kava drinking practices in the early 1980s fostered religious fellowship and facilitated a public expression of spiritual commitment.³ Kava use appeared to be supervised and controlled in these settings. For young people engaging with the social changes emerging since the 1970s, kava drinking practices provided opportunities for more relaxed social interactions that would normally have not been sanctioned under restrictive kinship guidelines rigidly enforced by elders. Such interactions were, and still are, more problematic with alcohol in the environment. Many of the negative impacts of alcohol use were reported to decrease significantly when kava was first used; a matter commented on by observers at the time.^{39, 44, 56} However, these changes were also short-lived and alcohol abuse continues to be a significant community issue. Data on exposure to kava use in 1999 indicated that 47% of kava users were also alcohol users (Clough AR, unpublished data). More recent (2000-2002) data from community surveys shows that 43%-56% used kava and alcohol (Clough AR, unpublished data). It seems likely that kava and alcohol use will continue to co-exist in the region.

Drinking style and kava abuse

Observations of Fijian kava drinking practices created the conditions for a local variant of these styles of kava use to be adopted in Arnhem Land, viz. a steady tempo of drinking brews of dried kava powder mixed with water.⁹ This style of consumption continues to characterise kava drinking in Arnhem Land today and it has fitted easily with community life.³⁹⁻⁴¹ Just one drinking cup tends to be used amongst a group to share kava in turn among drinkers gathered around a bowl with the kava dispensed by the person who mixed

the brew.⁹ The etiquette of one person serving a cup at a time turns out to be a very effective constraint on consumption. Notwithstanding the few drinkers who double up in a round, opportunities to guzzle more than anyone else are limited by manners. This pattern of drinking is both a constraint on the total amounts of kava individuals are able to consume and a kind of enforced sharing of the available supply.⁹ When kava is scarce, everyone tends to share the reduced consumption.

Conversely when kava is plentiful everyone tends to drink more because of this same structure of sharing. While all may drink at a steady tempo, when more kava is available, as has been demonstrated, more people will spend more time drinking it in the same settings until there is no more kava.⁹ So, the structure of drinking itself, while potentially a vehicle leading to kava abuse, also affords some assurance that controls on total supply under a regulatory regime are likely to be a very effective way to control *everyone's* consumption.

DISCUSSION AND CONCLUSIONS

We have presented evidence to demonstrate the congruence between structures and mechanisms for controlling kava and managing the proceeds from its sale that already existed in kava using communities prior to 1998 on the one hand, and the recent regulatory measures developed in consultation with these same communities on the other. Communities have once again demonstrated the desire for a local monopoly in the trade in kava for community benefit and for the development of community enterprise and they have also demonstrated their intent to eliminate the illegal trade. At the same time it is clear people want to continue to use kava in order to enjoy the positive social aspects of its consumption. Also, there is evidence that community concern exists about health and social effects of excessive kava use and that community groups recognise that such effects need to be addressed by appropriate local action from time to time. Kava management plans backed by the authority of NT Government law have the potential to realise these harm reduction objectives.

In the debate about kava use in Arnhem Land, it has often been presumed, and it continues to be reported, that Aboriginal people have no social controls of their own for kava use. The particular form this criticism has usually taken is that Aboriginal kava drinkers lack a moderating, formal, ceremonial context of kava use as exists in Pacific island societies.^{52, 57-60} There is no evidence to suggest that a formal ceremonial context alone would be sufficient to prevent kava abuse and, in any event, there is enough concern emerging from Pacific island societies to suggest that kava is becoming a contemporary drug of abuse there too.⁶¹ Given that the common experience of these Indigenous societies is rapid social change in the context of economic modernisation, a broader view of what constitutes social controls on consumption of any drug in this context is required. The information presented here demonstrates that the development of the regulatory system for kava in the NT has been open to incorporating Aboriginal views about what constitutes appropriate controls

which do, in fact, exist and that the regulatory system has provided the unusual opportunity for these to be supported by the power of NT law.

Possible threats to a fragile regulatory system

What we have described amounts to a very large effort in policy making to try to control a drug used for a comparatively short time in remote Arnhem Land where perhaps no more than 1800 to 3100 people have ever used it. And if 53% of males and 11%-27% of females are currently use kava,¹⁷ perhaps just 1900 people are using it today including the 280-330 who are doing so in accordance with a licence. This extraordinary effort reflects how serious is the concern that continues to exist about the health, social and economic effects of kava misuse in Arnhem Land and about exploitation of Aboriginal people with limited resources by the illegal trade. There is a view that this regulatory effort imposes a system of rules foreign to Indigenous social systems, one that is repressive or unworkable.^{62, 63} The evidence we have presented demonstrates that the current system was developed in consultation with affected communities and incorporates their wishes in a novel way. This now parallels other developments in similar areas of recognition of customary law in the NT.^{64, 65}

However, it should be recognised that such systems are fragile and vulnerable to collapse, as occurred with kava licensing efforts in the early 1990s. Accordingly ongoing consultation and consideration are required. A principal threat appears to lie in the resurgence of an illegal kava trade. Continued successful policing is required to discourage illegal traders. Objective assessment and monitoring of the kava management system by Government and non-Government agencies and kava licensees is also required to enable prompt remedial action to be taken if adverse outcomes are recognised. Commitment to harm reduction sentiments underpinning kava regulation and the managerial capacity to fulfill their responsibilities are required by licensees. It will be important that the NT Licensing Commission continues to maintain the criterion that wholesale and retail licensees use profits from kava sales for local community benefit and

for monitoring, education, research and evaluation as opposed to private or corporate gain. It will also be important that licensed retailers adhere to their respective kava management plans that form the conditions of their licence and that they implement their strategies to reduce harm from excessive kava use. Finally, national regulatory authorities will need to consider likely impacts of their future decisions on the developing system for kava regulation in the NT.

How much kava is too much kava is an issue addressed in the companion paper to this¹ which concludes that a successful legal trade must be balanced against the persisting threat of the rival illegal trade and the health and social effects of its use. The capacity to decide how much kava is too much now lies more in the hands of local communities. Recognising, acknowledging and strengthening local control mechanisms outlined here and marshalling their potential to manage supply and distribution using global constraints on legal supply, combined with greater restrictions on the illegal trade, may afford unusual strategies to reduce harm from too much kava.

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**The global controversy about the hepatic toxicity
of kava (*Piper methysticum*):
a discussion.**

INTRODUCTION

Over the last decade there has been an expanding global market for herbal preparations manufactured in western countries and containing kava extracts.¹ These products have been marketed for the treatment of anxiety, insomnia, premenstrual syndrome and stress and sold over the counter as complementary medicines or dietary supplements.² Kava is now internationally controversial and this is of direct relevance for this thesis since Aboriginal people continue to use kava while concerns about its possible hepatic toxicity have emerged in western countries. The present discussion outlines the significance of the controversy about kava's liver toxicity for the continued use of kava in Arnhem Land and also uses the data in the thesis to inform the international debate about its causes.

Since 1999, cases of severe hepatic toxicity in people using kava-containing herbal products have been reported from Europe and the United States. Subsequently, kava-based herbal products have been banned in some European countries including the United Kingdom.²⁻⁴ In February 2002 a practitioner alert and a consumer advisory were issued in Australia by the Therapeutic Goods Administration (TGA) concerning hepatic toxicity possibly related to kava-containing products.^{5, 6} By late 2002 eight liver transplant cases following hepatic failure associated with the use of kava-containing products had been reported from Europe and two from the United States.² Recently, one Australian patient who had used a herbal remedy containing kava died soon after liver transplantation.⁷ As a result of this case the TGA initiated a voluntary recall of all complementary medicines containing kava extracts on August 15th 2002.⁸ The TGA has 87 products containing kava on its Australian Register of Therapeutic Goods.⁹

Kava: herbal panacea or liver poison?

In a recent contribution to the debate about this controversy, Moulds and Malani discussed the paradox that fulminant hepatic failure has not been documented with traditional kava use in Pacific countries.¹⁰ They also emphasised that the kava used in

western countries is not the kava used in the Pacific where the water extract of powdered roots is the common traditional drink.¹⁰ As reported in Chapter 5, paper#4 and #5, no evidence for serious irreversible liver injury has emerged in Aboriginal kava drinkers in Arnhem Land, who also use kava in a similar style, as an aqueous brew of kava powder imported from Pacific island countries.

At a symposium held to discuss the issue of international bans on kava held at the Pacific Forum Secretariat in Suva in November, 2002, participants from several Pacific countries expressed their frustration that an item of trade of considerable significance to Pacific island economies¹¹ was suddenly banned in countries that had been major markets for Pacific kava exports. A range of views is reportedly held on this by some Pacific-based kava marketing agencies.¹⁰ Moulds and Malani note that some have expressed outrage and consternation that a Pacific icon which has been used for centuries without any suggestion of liver toxicity can be suddenly banned, while others have expressed mistrust and suspicion that commercial motives exist for banning a successful natural therapy that threatened the profits of the multinational pharmaceutical industry.¹⁰ These strong views from the Pacific are reflected in the views of some practitioners in the natural therapies industry in Australia, for example:

“How is it that a substance safely used as a beverage on a daily basis by a large percentage of the population in one country suddenly becomes so dangerous that it has to be totally banned in another? Clearly this is more an issue of politics than public health. Kava’s popularity could be its downfall. Whose interests are being served with the recommendation coming out of Germany that physicians there should revert to prescribing benzodiazepine drugs?”¹², p.143

While such commercial and political intrigues appear plausible, the demand for kava is also likely, in the medium term, to be subject to changes in the wider economic context which are known to lead in turn to sudden changes in the conditions that operate in the niche market for exotic agricultural products, such as kava, exported from small peripheral economies to large and complex ones.¹¹ Data to expose the truth of the situation may be difficult or impossible to collect, although further objective study of these issues could be revealing. Nevertheless, the clinical concern remains. Despite the

poor documentation of some of the reported cases of liver toxicity and despite the possibility that the liver toxicity in most of these cases may have been caused by co-ingested drugs or herbs,¹³ there is sufficient evidence that kava preparations available as manufactured products and sold in the natural therapies industry in western countries cause severe liver toxicity in some patients.^{2, 7, 14-17}

In this debate, Moulds and Malani have posed two interesting and challenging questions.¹⁰ Firstly, have people in western countries been saved from an epidemic of liver toxicity by vigilant regulators, or are they being denied the benefits of an effective alternative therapy with few side effects? Secondly, they ask whether Pacific islanders have been unwittingly exposing themselves to a nasty liver toxin, and should they now change their traditional habits?¹⁰ Their second question is also of particular relevance to Aboriginal kava drinkers in Arnhem Land since they have come to use kava in the same way as Pacific islanders.

Consideration of the issues

Moulds and Malani raise several unresolved issues of contention in this debate.¹⁰ Some of these issues can now be addressed using the information provided in the studies conducted for this thesis.

*Issue 1. How relevant is the Pacific island (and Indigenous Australian) experience to the interpretation of the cases of liver toxicity in western countries.*¹⁰

A major deficiency in the information available about the Pacific experience¹⁰ and that of Indigenous Australians who drink kava (Chapter 5, paper#4 and paper#5) is that neither population has been systematically examined for liver toxicity related to kava use. Moulds and Malani also concede that since the population exposed to risk of liver toxicity from kava use is small, a low frequency of occurrence is likely to go undetected even in the Pacific islands with a total population of just over one million people.¹⁰ In Arnhem Land, this problem is magnified since the population of Arnhem Land Aboriginal communities where kava has been used is less than 10 000, and of these only

from 1800 to 3100 people may have ever used kava (paper#10, this Chapter). Moulds and Malani suggest that this is a major public health concern in Pacific island countries and needs to be addressed.¹⁰ The issue also remains a concern for Indigenous kava users. However, paper#4 and paper#5 (Chapter 5) shed some light on the issue.

Firstly, clinical observation over a period of 20 years has not documented any cases of fulminant hepatic failure attributable to kava use in Indigenous Australian kava drinkers.¹⁸ This does not imply, of course, that liver injury may have occurred but did not come to the attention of clinicians, underscoring the need for continued surveillance.

Secondly, the reversibility of raised levels of the liver enzymes γ -glutamyl transferase (GGT) and alkaline phosphatase (ALP) without an elevation in levels of alanine aminotransferase (ALT) does not suggest acute inflammation and is not consistent with the changes documented in the cases of hepatic toxicity associated with herbal products, where aminotransferase levels were especially high.^{2,7,14} Paper#5 (Chapter 5) reported reversibility of GGT and ALP elevation independently of the effects of alcohol and other substance use. Similar studies are required for Pacific island populations to describe the nature of any liver function abnormalities and to make comparisons with those found in Indigenous Australians. If similar results are found in these unrelated populations, this would add weight to the suggestion that liver function changes in those who use kava in the traditional manner do not reflect the same pathological process.¹⁸

Issue 2. Comparative doses from different extraction procedures

Chapter 2 (paper#1) suggested that varying approaches to estimating dosages of kava lactones in controlled studies of kava's health effects make it difficult to compare the results of pharmacological studies with kava's health effects in Indigenous kava users who use kava brewed in a traditional form. Aboriginal kava drinkers in Arnhem Land may consume dosages of kava lactones 10-50 times greater than recommended therapeutic dosages in manufactured herbal remedies. This has been largely overlooked or misinterpreted¹³ in the debate about kava's liver toxicity until Moulds and Malani raised the issue for discussion.¹⁰

While it is not suggested that all of kava's health effects are caused by kava lactones, the apparent incongruity between dosages suggests the effects of other factors leading to hepatic injury in those who use herbal extracts containing kava. This is further reinforced by the estimate of exposure time for Aboriginal kava drinkers in Arnhem Land, viz. approximately 30 000 person-years of moderate to heavy kava use, and by the lack of evidence of deaths from liver disease in Aboriginal kava drinkers during a 15-year period (1985-1997) when kava drinking in Arnhem Land was known to be heaviest (Chapter 5, paper#4).¹⁹

Issue 3. Genetic differences in liver metabolism

In two patients with liver failure reported in Europe, phenotyping of the activity of cytochrome P450 isoform CYP2D6 showed that they were "poor metabolisers", and it was postulated that genetic differences in liver metabolism of kava lactones may be important.¹⁶

In many populations it has been shown that a small proportion may have inherited deficiencies in glutathione-synthesising enzymes.^{20, 21} This is of relevance since it is also known that drug-related idiosyncratic hepatic reactions can occur in individuals with inherited deficiencies in the capacity to synthesise the P450 family of phase I drug metabolising enzymes, in particular the CYP2D6 enzyme which is estimated to be involved in metabolising around one-quarter of all prescribed drugs.²² It is also known that this enzyme is inactive in around 6-9% of Caucasian populations²² while, interestingly, some indigenous Pacific island populations and south-east Asian groups (no data for indigenous Australians) show little or no deficiency in CYP2D6 activity.^{21, 23}

Glutathione is a tripeptide, which consists of the amino acids *gamma*-glutamic acid, cysteine and glycine, and is known to play a role in the metabolism of a wide range of compounds including xenobiotics.²⁴ Glutathione is concentrated in the liver and is known to have potent antioxidant action and by directly quenching reactive hydroxyl free radicals is known to be an extremely important cell protectant.²⁵ For example, it

has been shown in the laboratory that the cytotoxicity of the sesquiterpene group of lactones, which includes kava lactones, is markedly diminished in the presence of glutathione.²⁶ Glutathione is also an essential cofactor in enzymatic detoxification and may have an important role in the phase II conversion of kava lactones into waste products that can be excreted.²⁵ The homeostatic redox cycle in humans normally keeps glutathione replete although oxidative stress can outpace glutathione synthesis resulting in depletion of glutathione reserves especially if dietary intake is limited.²⁵ Of interest is a recent report that glutathione is present in the 'traditional' aqueous kava extract¹³ and that manufactured ethanol or acetone extracts contain reduced amounts or no glutathione at all^{13, 27} suggesting that glutathione from kava itself may play an additional protective role in the hepatic toxicity of kava lactones.

We do not know how many people are exposed to kava as a herbal remedy in western populations. However, based on the low incidence of hepatic toxicity in users of other herbal remedies²⁸ it seems likely that the incidence is also very low in users of kava products. This together with the putative lack of glutathione in manufactured kava products²⁷ suggests one plausible explanation for why liver damage occurs in some users of these products and not in all who have used them and not, as far as we know, in users of aqueous extracts (Chapter 5, paper#5).^{10, 18} If the liver is challenged by kava lactones without the added benefit of a natural source of glutathione, it is possible that the reserves of protective glutathione in hepatocytes may become depleted leading to cell damage or death. It is also possible that a small proportion in the Caucasian populations who have used the manufactured products have inherited deficiencies in the glutathione synthesising enzymes or the CYP2D6 and related P450 enzymes, or deficiencies in both. These deficiencies would predispose such an individual to adverse drug reactions with hepatic stress occurring during the metabolism of kava lactones. There is some support for part of this hypothesis since both cases of liver failure noted by clinicians in one report were found to be deficient in the CYP2D6 enzyme, an occurrence which has a probability of less than 0.01% in a population where the prevalence of CYP2D6 deficiency was 9%.¹⁶

Issue 4. Manufacturing Processes and Extraction procedure

Several commentators have suggested that the liver toxicity from herbal remedies containing kava lactones is the result of extraction procedures.^{13, 29} One interesting hypothesis (Peter Whitton, personal communication) is that at least a component of kava lactones in kava may be broken down in a pH-related reaction²⁶ by bonding with glutathione (amongst other agents) to form water soluble molecules that are less-directly dependent on the liver for their metabolism. This means that a component of the kava lactones in a water soluble state could be absorbed both in the mouth and oesophagus, possibly accounting for the characteristic numbing of the tongue and throat when kava is consumed. This also may mean that in traditional kava extracts the molecule challenging the body is somewhat different from that in the manufactured extracts which will not be water soluble and possibly more toxic to the liver if depletion of the cellular glutathione stores occurs and renders the individual liable to damage during oxidative stress. Further research into these interesting aspects of kava lactone metabolism may also shed light on the wider issue of the hepatic toxicity of herbal remedies.³⁰

It has been suggested that poor control over the parts of the kava plant harvested and used to manufacture herbal remedies has led to the introduction of a liver toxin into manufactured products to which Pacific populations have not been exposed. This may be possible, but no convincing data has been published. A related issue is that, due to the varied approach to the regulation of complementary medicines in different countries, herbal remedies have not required the same rigorous testing or manufacturing controls as new pharmaceuticals.¹⁰ So the toxicological studies have not been performed that are necessary to make assessments of liver toxicity in humans.¹⁰ The possibility of reactions in some individuals to poor quality products in the natural therapies industry because of defective manufacturing processes was highlighted recently by the large scale recall by the TGA of an Australian company's products,³¹ many of which were natural therapies or herbal remedies. While only two of these products contained kava,⁹ and neither was the product reported to be associated with the recent death of an Australian patient from liver failure,⁷ this episode helps to emphasise the variety of factors that may effect the hepatic toxicity of all drugs.

Conclusions

It is clear that a risk-benefit assessment for herbal medicines that contain kava would not be completely reliable since much knowledge is still lacking.¹⁷ While it seems likely that the frequency of the occurrence of severe liver toxicity in western countries where manufactured kava products are used is very low, data are not available to compare the risks with established anxiolytic drugs such as the benzodiazepines, for instance.

Although a rigorous systematic review found kava to be an effective symptomatic treatment option for anxiety,¹ it seems prudent to conclude that herbal preparations containing kava should not be used until the mechanism for hepatic toxicity is clearly ascertained and until an adequate risk-benefit profile can be established. The fate of kava as a component in the natural therapies industry, however, will also be linked with a wider emerging controversy about the safety of herbal remedies generally.³⁰

Moulds and Malani suggest that in the meantime people in Pacific island countries can probably continue using kava in the traditional manner to which they have become accustomed.¹⁰ Similarly, Aboriginal people in Arnhem Land can probably continue using kava in the same way since there is no evidence that kava has caused severe liver damage in this population. However, while the risks may be small they are nonetheless unknown. It may be that the abnormal but reversible GGT and ALP levels seen in Aboriginal kava drinkers in Arnhem Land do not reflect the same pathological process. However, further clinical surveillance and close monitoring is required to ensure that if hepatic injury becomes evident in Aboriginal kava users in Arnhem Land, regulatory authorities can take appropriate action. Similar surveillance may be appropriate in Pacific countries.

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CHAPTER 9

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

INTRODUCTION

This chapter presents a summary and interpretation of the findings of the thesis in relation to the research questions set out in Chapter 1 (page 1-14) which can now be addressed. Also provided are suggestions for further research indicated by the thesis findings and some of recommendations for policy and practice they imply. The strengths and limitations of the evidence available to address the questions posed in the study are also discussed.

Question 1. Pneumonia and ischaemic heart disease

Is kava use associated with an increased risk of pneumonia? Is kava use associated with an increased risk of ischaemic heart disease? And if so, are these risks increased with increased kava use? Furthermore, do these risks occur independently of alcohol or other substance use?

The study found no convincing evidence that kava is associated with an increased risk for admission to hospital with pneumonia (Chapter 6, paper#7). However, the non-significant tendency for an association is in the direction of an increased risk. Crude odds ratio, OR=1.26 (0.74-2.14, P=0.386), increased after controlling for confounders (OR=1.98, 0.63-6.23, P=0.237) but was not significant. Adjusted OR for pneumonia cases being kava and alcohol users was 1.19 (0.39-3.62, P=0.756). In communities with longer kava-using histories, adjusted OR was 2.19 (0.67-7.14, P=0.187). There was no kava dose-response relationship.

The multivariate analysis adjusted for confounders including tobacco use. A weak interaction between kava and alcohol use was considered in the analysis but was found to be non-significant. Effects of alcohol use, if they existed, were controlled in the stratified analysis. Despite the possibility of an immune abnormality in kava users suggested by the

results of the cross-sectional study (Chapter 5, paper#4), this study showed that kava use alone is not clearly an independent risk factor for pneumonia as it appears to be for other serious infectious disease such as melioidosis.^{1,2}

Similarly in the case-control study of kava use and ischaemic heart disease (Chapter 7, paper#8), while the tendency for an association between kava use and admission to hospital with IHD was not significant, the direction of the association suggested an increased risk of around 50%. A crude OR=1.41 (0.73-2.73, P=0.303) that those admitted with an ischaemic event were kava users changed little after controlling for confounders (OR=1.51, 0.75–3.05, P=0.247). In just those communities with a longer (up to 15 year) history of kava use, odds ratios for kava use among those admitted with an ischaemic event increased (OR=1.75, 0.82-3.74, P=0.140) but were not significant. Twenty-five cases were admitted more than once. Risk of readmission tended to be higher in kava users (adjusted OR=2.24, 0.65-7.68, P=0.191) but not statistically significant. Twenty people died with IHD as the principal cause without admission to hospital during 1992-1997. Even with these cases and their matched controls included in the analysis, no association between IHD and kava use (adjusted OR=1.44, 0.78-2.66, P=0.245) was found. This study provided no clear evidence for an association between kava use and IHD (Chapter 7, paper #8).

Question 2. Cross-sectional study

In a sample of Aboriginal people living in one community in the Miwatj region where kava has been used, do we find that increased exposure to kava use is associated with abnormal biochemical, haematological, immunological, cardiovascular and neurocognitive findings? Furthermore, do these risks occur independently of alcohol or other substance use?

In the cross-sectional study (Chapter 5, paper#4), kava use was associated with dermatopathy, liver function abnormalities (GGT and ALP) and decreased lymphocytes.

These results were also found in the earlier cross-sectional study carried out by Mathews et al.³ Markers of cardiovascular risk (fibrinogen, plasminogen activator inhibitor-1, C-reactive protein and homocysteine) were increased across the population, but were not higher in kava users; and neurocognitive tests were not different between kava use categories. IgE and IgG antibodies were elevated across the whole group possibly due to a heavy pathogen burden. The interaction between kava, alcohol and other substances was examined in terms of kava's association with liver function abnormalities (Chapter 5, paper#4). The small sample size in the cross-sectional study and multiple substance use common among the population made this difficult. To elucidate the confounding effects of other substance use for all characteristics measured on the cross-sectional study would require further research with a larger sample size, ideally with participants matched for age and sex with similar numbers of substance users and abstainers, for instance alcohol users and non-users.

There was no evidence for any long term neurological damage in kava users. This was briefly reported in the cross-sectional study (Chapter 5, paper#4) and described in more detail in the neurocognitive studies published from that data.⁴ However, acute toxicity and withdrawal effects of kava use are possible. In other published papers^{5,6} not included in this thesis, the occurrence of 32 unusual neurological presentations to community clinics during the 1990s was recorded during the course of conducting the research. These presentations were for "seizures" or "fits" (n=32). More than half of these episodes occurred during 1994-1997, the period when kava availability may have reached its peak⁵ and when kava drinking is likely to have been heavy. A majority of individuals experiencing "seizures" were heavy users described by local Aboriginal health workers as people who were known to drink kava in continuous sessions for 24 hours and more.

Question 3. Specific effects of kava

What evidence is there for effects of kava use on liver function, cardiac function and nutritional status? Are these effects associated with increased exposure to kava use? Furthermore, do these risks occur independently of alcohol or other substance use?

Liver function abnormalities were found to be reversible in kava users and to begin to return to baseline after 1-2 weeks abstinence from kava (Chapter 5, paper#5). No evidence for irreversible liver damage was found even in those who had used kava more or less continuously for up to 18 years. The occurrence of serious irreversible liver damage in some people in western societies who have used natural therapies containing kava stands in stark contrast with the lack of evidence for serious liver damage in Indigenous Australian kava users and in those who use kava in the Pacific region in a traditional manner.

Chapter 5 (paper#6) described trends across kava-using groups with weight loss, lower skin fold thicknesses, lower body mass index and body fat in continuing kava users compared with past users and non-users. Total cholesterol, high density- and low density lipoproteins were increased but not triglycerides. Antioxidant levels that were already very low were not further compromised even in heavy kava users. Weight loss and altered lipid profiles may be more a result of an obsession with kava drinking than to oxidative stress induced by kava itself. The implications of the dyslipidaemia observed for a possible increased risk of cardiovascular disease require further study.

The case-control study of IHD concluded that longer-term biological processes may still be developing and perhaps an association between kava use and IHD would, in time, emerge. In addition, a plausible mechanism for a specific acute effect of kava was proposed. Kava's strong muscle-relaxing properties may increase the risk of arrhythmia or abnormal atrioventricular function. It is plausible that a cardiac event may occur in people with existing abnormalities in cardiac output or effective mechanical performance, as may be the case in many Aboriginal kava drinkers, especially with heavy exercise following kava use.

Question 4. Patterns of kava use and social and economic impact

To what extent has kava been used in Arnhem Land communities and what social and economic impacts have occurred in association with its use?

The available evidence suggests that kava has been used by perhaps as many as seven out of ten adult males and around half of the adult females in some Arnhem Land Aboriginal communities (Chapter 4, paper#3). During the 1980s, about half the total adult population was using it in those Arnhem Land communities where it was available. In the early 1990s, 70% of males and 62% of females were using kava in one western Arnhem Land community (Chapter 8, paper#9). Kava supply to Arnhem Land may have peaked between 1994 and 1997 but community supply data during this time was not available to confirm this. It is possible, however, that from 27-36 tonnes of kava were being used per year during 1994-1997 (Chapter 8, paper#9).⁵ In 1999, 53% of males and 27% of females were continuing to use it in eastern Arnhem Land kava using communities (Chapter 4, paper#3). The most recent data available, which was collected in 2001-2002 in the community in eastern Arnhem Land where kava was first introduced in 1982, suggest that kava, supplied illegally, was still being used there by a similar proportion of the population (Chapter 4, paper#3).

Recently, the pattern of kava supply has become more complicated with kava supplied legally in accordance with a licence under the regulatory scheme implemented in 2002 in three communities. Illegal kava is still available in some localities (Chapter 8, paper#10). Data describing more recent consumption patterns in this complex picture are not available and further research and monitoring of kava use levels is required.

In 2000 in the cross-sectional study, the individuals interviewed reported their current consumption of kava powder at an average of 118g/week, ranging from <40g/week to >195g/week (Chapter 5, paper#4). This is less than 368g/week (ranging from <130g/week to >610g/week) described in western Arnhem Land in 1990-91 (Chapter 2, paper#1). It is

also less than the range described by Mathews et al in eastern Arnhem Land in 1987 (from 100g/week to 900g/week) with most kava users surveyed using it at around 440g/week and described by Mathews et al as ‘very heavy’ users.³

When the cross-sectional study was conducted in 2000, median duration of kava use was 12 years (range 1-18 years) in the individuals interviewed. One-quarter of kava users had used it continuously for 16 years or more (Chapter 5, paper#4).

When kava supply to Arnhem Land reached its peak during 1994-1997, its value may have reached \$6-8 million per year in an unregulated trade (Chapter 8, paper#9). To put this into perspective, this amount of money was equivalent to the total capital and operational budgets for 1996 of the biggest community Council in Arnhem Land which, at the time, had a population of around 1350 people and, of which this author was the chief executive officer (unpublished observations). As pointed out in Chapter 1 (page 1-9), the quantity of kava imported in the early 1990s represented 10% of the total amount of kava exported to all countries from Fiji, the major kava exporter in the Pacific at that time.⁷ The structure of the kava trade in Arnhem Land communities described in Chapter 8 (paper#9 and #10) implies that a proportion, perhaps as much as half of the profits from the kava trade can be redistributed or redirected for local community or family purposes. The balance is usually removed from the local community economy in an unregulated or illegal trade. Information presented in Chapter 8 (paper#9) showed that when kava users in one community began to consume it at an average in the range 240-425g/week from mid-1990, 19% of available cash resources were spent on kava with 11% of those cash resources leaving the local community. The proportion of men drinking kava in this community reached 70% and women 62% from mid-1990 with 20% of the population spending unprecedented amounts of time (14+ hours/week) in activities where kava was consumed. In eastern Arnhem Land, recent data show that the economic impact of illegal kava use was estimated to be equivalent to that for tobacco (Chapter 4, paper#3). Further data on the social and economic effects of this pattern of kava usage are not available or consist

largely in anecdote and unpublished observations and commentary. Further study is required.

Question 5. Policy consequences

What kinds of policies and interventions have been implemented to minimise the harmful health and social effects of kava use? How have these policies and interventions developed, what has informed their development and, what can be learned from this experience for development of future policies and interventions?

Concerns about the health consequences of kava abuse in Arnhem Land Aboriginal populations and the persistence of an illegal kava trade, with scarce cash resources used to purchase it and few benefits returned to communities that use kava, have been a cause for concern for 20 years. Despite these concerns, some Arnhem Land groups seek to continue using kava and to control its sale and distribution and profits from the enterprise. In response, policy makers in the NT have embraced principles of harm reduction and have created regulatory mechanisms to address broader public concerns and to support local management of kava supply while reinforcing control over the consequences of its use (Chapter 8, paper#10).

Two legislative attempts by the NT Government were implemented; firstly in 1990 and again in 1998 with the passage of the *Kava Management Act*. From 1982-1990, informal supply networks were established and kava use was consolidated with no regulation. Then, from 1990-1993, the first attempt to regulate kava operated. During this time, there occurred a very large increase in the amount of kava supplied to Arnhem Land communities. At least two factors seemed to be involved in this rapid expansion of kava use. Firstly there was improved reliability of kava supply with cash and credit managed through conventional trading mechanisms. Secondly, social control mechanisms that were already in place were marginalised when the authority to purchase and supply kava shifted to community Councils (Chapter 8, paper#9 and #10). For local Aboriginal community

members, control over these contemporary institutions in their traditionally oriented communities has been problematic in many instances.

The kava regulatory system now being implemented in the NT under the *Kava Management Act* features kava management plans that specify how kava is to be controlled (Chapter 8, paper#9). Kava management plans have been developed through consultation between Aboriginal communities and the licensing authority representing the NT Government. These consultations were more comprehensive than the consultation efforts made when the first regulatory system was established in 1990. Since the plans developed reflect local social and geographical circumstances, they may provide improved opportunities to reinforce local control over kava. Under the new regulatory system, pricing of kava and licensees' use of profits are negotiated through the licensing process. The lucrative illegal trade continues to threaten the regulatory system but has declined markedly since the end of 1997 (Chapter 8, paper#9).

The process of licensing now appears to be more consistent with the will that has prevailed among some Aboriginal groups who consistently tried to control kava since it was introduced in 1982 for community benefit, in part to reduce health problems, but also to permit the enjoyment of a mood-altering substance that did not have the harmful effects of heavy alcohol use and because cash could be generated from kava sales. The new regulatory system holds the prospect for these efforts to be realised. However, continued support is required to enable those Aboriginal organisations now licensed to trade in kava to achieve the objectives of their kava management plans within a unique regulatory system (Chapter 8, paper#10).

While this research was being prepared for publication its results had some influence on the recent policy approaches to kava in the NT. The successful applicant for the wholesale licence to supply kava sought advice from myself on the health effects of kava, the only applicant to do so. As a result, the applicant proposed in its licence application a program to monitor the health effects of kava and the impact of its continued use on community life

(Chapter 8, paper#10). It also proposed to implement an education program about kava in order to encourage its responsible use. Each of the successful retail licensees was subsequently asked by the licensing authority to address these same matters in their applications. To control supply of kava, retail licensees were also asked to provide in their applications details of how they would control the quantities that could be purchased in their communities. The licensing authority (NT Licensing Commission) used the guidelines for monitoring that were developed in Chapter 8 (paper#9) of this thesis to guide the applicants. Controlled availability of kava to Arnhem Land Aboriginal communities remains an important underpinning principle of the new licensing system.

Question 6. International implications

What are the implications of this research for the use of kava as a mood-altering substance in other parts of the world?

The international controversy about kava's liver toxicity is of direct relevance for this thesis since Aboriginal people continue to use kava while concerns about its possible hepatic toxicity have emerged in western countries (Chapter 8, discussion).

The recent cross-sectional study (Chapter 5, paper#4) confirmed findings of the 1987 study where abnormal liver function tests (elevated GGT) were found in Aboriginal kava drinkers. Abnormal GGT and ALP were found in 61% and 50% of kava users respectively. Importantly however alanine transaminase (ALT) was not raised in any of the kava drinkers. By contrast, in the cases of irreversible hepatic injury possibly caused by herbal remedies containing kava in Europe, north America and Australia, ALT was found to be markedly raised.⁸⁻¹⁰ Furthermore, the abnormalities in liver function in Aboriginal kava users have usually returned to normal within one to two months after stopping kava use (Chapter 5, paper#4 and #5). The elevated GGT and ALP but normal ALT in Aboriginal kava users are not suggestive of acute inflammation and are not consistent with the changes documented in the cases of hepatic toxicity associated with

herbal products, where aminotransferases are especially high. Clinical surveillance in the NT over 20 years has not documented any fulminant hepatic failure attributable to kava use (Prof Bart Currie, personal communication). This is despite Aboriginal kava drinkers consuming kava lactones in doses estimated to be 10-50 times the recommended therapeutic doses for herbal products (Chapter 2, paper#1).

The abnormal but reversible GGT and ALP seen in heavy kava drinkers (Chapter 5, paper#5) does not appear to reflect the same pathology as in the cases of liver injury¹⁰ and whether the apparently idiosyncratic fulminant hepatic failure documented with herbal kava preparations can also occur with traditional aqueous extracts requires further study.

Strengths and limitations of the research

Measuring individuals' kava consumption proved to be a major challenge for this study. Measuring variations in daily and long term use retrospectively at different levels of kava consumption was particularly difficult. In the earlier studies, community supply information was available to assist to corroborate estimates of consumption. But this was not the case in the cross-sectional study in 2000 (Chapter 5) and for the case-control studies reported here (Chapter 6, paper#7 and Chapter 7, paper#9).

Classification by proxy respondents using assessments of the time people spent drinking kava in different social settings assisted to identify kava users and to distinguish the heavier kava users among them (Chapter 2, paper#1). By studying the social settings in which kava was consumed and with close observation in one community, it was possible to develop a classification of drinking settings and to estimate the amount of time people would spend in those settings where kava was consumed (Chapter 2, paper#1). In those admitted to hospital with pneumonia and ischaemic heart disease, when non-users were compared with moderate users and with heavier users identified by health workers, pathology reports on admission showed that GGT and ALP increased across the groups

(unpublished data). Also in the cross-sectional study, those classified by health workers as heavy kava users had higher GGT ($|t|=4.32$, $P<0.001$) and ALP ($|t|=3.44$, $P=0.001$) than those classified as more moderate kava users (data not shown). Heavier kava users were identified both by self-reported kava use as well as by health workers' consensus classification independent of self-report in the cross-sectional study and in the case-control studies and other reports of admissions. Since these measures of exposure were independently associated with elevated liver enzymes (GGT and ALP) in the cross-sectional study and in the hospital admissions, confidence in the validity of using health workers' assessments of kava use as proxy exposure measures is reinforced.

In my continuing studies, in a different group of communities in Arnhem Land (unpublished data), stronger validation of the use of data from proxy respondents emerged. Random samples of 194 and 176 individuals were selected in two communities and assessed by up to five health workers for their substance use including alcohol, tobacco, cannabis, kava and petrol sniffing. In addition, sub-samples of $n=77$ and $n=55$ in each of the two random samples, respectively, were opportunistically recruited for interview. Proportional agreements among health workers and agreement between health workers and self-report were analysed. Under-reporting in interviews, especially for current use, was found in both communities as was found in this study (Chapter 3, paper#2). The data not yet published, show that consensus classification by health workers yielded 'moderate' to 'good' agreement for all substances for both a history of use and current use except for petrol sniffing for which 'fair' agreement was achieved.

Proxy respondents have been used in socio-demographic studies of educational level, height, weight and employment status, studies of medical history including chronic and acute conditions, epidemiological studies of surgical events, medication use and family health history, and research into diet and life habits including alcohol and drug use, for example.^{11, 12} Proxy respondents are used when the index subject, the preferred source of information, is not available for questioning. Recent reviews discuss the validity and utility of this approach in, for instance, geriatric studies,¹³ studies of symptom burden in

palliative care¹⁴ and chronic disease.¹⁵ Data collected by proxy has typically relied on a ‘significant other’, usually a close member of the family, or a health care provider.¹⁵ Aboriginal health workers in remote communities are both health care providers and family members, i.e. they are ‘significant others’, but for a much wider family group. Moreover, their profound knowledge of their own peoples’ health and habits, hitherto little recognized in epidemiological studies, is likely to be useful and valid for large numbers of community members and not just a few individuals. This comes about because of the traditional structure of extended family relationships in small isolated localities where interactions and behaviours are often close and well known (Chapter 3, paper#3).

Health worker knowledge assisted to overcome language and cultural barriers and to deal with the issue of under-estimating in self-report, well known in estimating exposure to illicit or stigmatised substance use.¹⁶ An additional advantage is that a consensus of Aboriginal health workers may reduce the rate of “non-response”, i.e. the occurrence of missing values. This stands against the usual disadvantage of using proxy assessments in epidemiological studies where missing information reduces the data available for multiple logistic regressions resulting in a tendency for the odds ratio to be biased towards a value of one.¹⁷

A major hazard with using proxy respondents is the prospect of misclassifying exposure status. It is possible that health workers systematically misclassified individuals in the studies reported here, especially since there was consistent *disagreement* between consensus classification and self-report. However, when proxy respondents consistently agree with each other, and make ‘errors’ in the same direction about most of the substances assessed in different communities and with different groups of health workers making assessments at different times, systematic under-reporting in interviews, a well-known tendency in the drug and alcohol field,¹⁶ seems more plausible.

The small sample sizes were an unavoidable weakness in the study. In the case-control studies (Chapter 6, paper#7 and Chapter 7, paper#8), the smallest detectable effect size was

around two (80% power, 95%CI). The potential for confounding or measurement error to explain any association of less than two was likely to be high given the complexities of exposure measurement. Nevertheless, in the absence of previous studies in this area, excluding an effect size greater than two is a reasonable beginning. Since, the direction of effect was towards an increased risk of approximately 50% in the study of IHD and almost 100% in the study of pneumonia, this research provides an important basis for further study and monitoring. Reliable data for IHD cases prior to 1992 was not available, so increasing the sample size in the study was not possible. It would have been possible to increase the sample size in the study of pneumonia admissions by including those admitted during 1992-1993. This may have assisted to increase the power of the study which would have allowed for a stronger analysis for kava using communities only.

In the study of admissions for ischaemic heart disease, an association between admission for IHD and kava was probably confounded by other substance use. An association between the well established risk factor of tobacco use and IHD emerged only when cases who died from IHD without admission during 1992-1997 and their matched controls were included in the analysis (Chapter 7, paper#8).

In the cross-sectional study (Chapter 5), a further weakness was brought about because of the requirement to work in the clinic and this meant that a random sample was not possible in this study. Participants were opportunistically recruited. This weakness may not have been serious given the overall congruence between the results of an earlier cross-sectional study and the one conducted as part of this thesis. The lack of appropriate controls for comparing characteristics across groups was an unavoidable weakness. Small numbers and the widespread use of a number of substances made it difficult to find suitable controls in the community studied. The earlier cross-sectional study was criticised for not controlling for the effects of alcohol and other substance use. In the study reporting liver function abnormalities associated with kava use (Chapter 5, paper#5), these effects were quantified in more detail and better controlled.

The case-control studies (Chapter 6, paper#7 and Chapter 7, paper#8) used data for all cases of pneumonia and ischaemic heart disease admitted from eastern Arnhem Land for defined periods where the majority of kava users in the Aboriginal population reside and for whom diagnosis could be confirmed. To the author's knowledge, these are the only studies of kava as an independent risk factor for these illnesses in any population. The 1990s, the period during which most of these studies were conducted, included the times of heaviest kava availability and consumption known in Arnhem Land (Chapter 8, paper#9 and paper#10).^{5, 18}

Recommendations for further research

Chapter 2 (paper#1) suggested that varying approaches to estimating dosages of kava lactones in controlled studies of kava's health effects make it difficult to compare the results of pharmacological studies with kava's health effects in Indigenous kava users who use kava brewed in a traditional form. Aboriginal kava drinkers in Arnhem Land may consume dosages of kava lactones 10-50 times greater than recommended therapeutic dosages. This has been largely overlooked in the debate about kava's liver toxicity, for instance, until recently.¹⁹ While it is not suggested that all of kava's health effects are caused by kava lactones, the apparent incongruity between dosages strongly suggests the effects of other factors leading to hepatic injury in those who use herbal extracts containing kava. This requires further study.

Improved methods of quantifying exposure to kava use are required. Research should focus on further investigating the utility and validity of using key informants, such as Aboriginal health workers, as proxy respondents. Some parameters that assisted to identify heavy kava use were developed in this study for use in proxy assessments (Chapter 2, paper#1 and Chapter 3, paper#2). Further work could validate these against more objective measures including changes in levels of liver enzymes with estimates corroborated by community supply information for kava which is now available under the

new regulatory system in Arnhem Land. The validity of using the same approach with proxy respondents to estimate other substance use and other aspects of the health of Aboriginal community people, such as weight loss or gain and the presence of chronic conditions, for example, also requires further investigation.

The hepatic toxicity of kava should be further studied with a focus on the temporal associations between exposure to kava use in its different forms at different dosage levels in samples matched for age and sex. Continued clinical surveillance among Indigenous Australian kava users is required for early detection of the emergence of any cases of serious liver injury. Such surveillance should also be considered in Pacific island populations. Further investigation of the causes of liver toxicity in users of manufactured herbal remedies containing kava is required. While studies of the prevalence of the use of herbal remedies containing kava in western populations may no longer be relevant, such studies could assist to provide information about the use of herbal remedies generally. A focus on pharmacogenetics and the risks of hepatic injury from xenobiotics may be fruitful.

The case-control study of ischaemic heart disease, while providing no clear evidence for an association with kava use (Chapter 7, paper#9) nevertheless suggested the possibility of an increased risk of the order of 50%. Since an association may yet emerge in the population, further study is required. Although kava users showed no evidence for accelerated atherosclerosis (Chapter 5, paper#4), the unusual lipid profiles of kava users who may be obsessed with the practice of drinking kava (Chapter 5, paper#6), and the relationship of these unusual lipid profiles with nutritional indicators, warrant further study as a possible increased risk of cardiovascular disease, especially since generally raised levels of risk factors such as C-reactive protein were observed in this study.²⁰

Further research into the association between serious infectious diseases and kava use is required. In particular, further data on the lymphocyte levels in those admitted to hospital with pneumonia should assist to clarify a possible immune response in kava users which

may predispose them to increased risks (Chapter 5, paper#4 and Chapter 6, paper#7 and #8). Lymphocyte levels in past kava users ($1.8 \times 10^9/L$, $sd=0.6$) tended to be higher than in current users in the cross-sectional study ($1.6 \times 10^9/L$, $sd=0.6$) although the comparison was not statistically significant ($|t|=1.26$, $P=0.213$) (Chapter 5, paper#4). The temporal relationship between kava use and lower lymphocyte levels warrants further study. The statistically significant associations between pneumonia admission and alcohol use, pneumonia admission and cannabis use and the tendency for an association with petrol sniffing (Chapter 6, paper#8), suggest that the effects of other substances, or combinations of substances, may also be important or may compound kava's effects in ways not yet understood. Hospital based case-control studies of IHD and pneumonia may be worthwhile in Pacific island populations.

Further study is required to more comprehensively describe the social and economic role of kava in Arnhem Land than was possible in this study. In particular, such research should focus on documenting the extent to which continued use of licensed or illegal kava impacts upon the cash resources available in a community and the dispersal of profits made from kava sales. Such studies should also examine impacts of kava use on community life and work patterns as well as on the food purchasing patterns of community members. In addition, since Pacific island countries have suffered economic loss and possible social disruption because of the international controversy about kava,¹⁹ this also warrants systematic study. Clearly, drug regulators in western societies cannot allow unrestricted use of a substance which may be associated with serious liver injury. Research to establish a risk-benefit profile for kava and kava-based products will be required if Pacific island kava exporting countries will be able to propose the possible re-establishment of a valuable and unique export industry.

The effects of an obsession with kava drinking need to be considered in further studies of possible toxicity and withdrawal seizures in kava drinkers and also of possible effects on nutritional status. A major differential diagnosis of a toxicity or withdrawal "seizure" is syncope.²¹ This is plausible among heavy kava users who suffer loss of body fat³ if food

intake is low. “Seizure” episodes described may well represent syncope rather than true seizures. However, the occurrence of seizures from apparent withdrawal effects after ceasing heavy kava use does suggest an independent effect of the substance itself, analogous to alcohol.

Recommendations for practice and policy

1. Since most of the health effects of kava use appear to be reversible with moderate consumption or abstinence, if Aboriginal people wish to continue to use it, policy approaches should be implemented that provide for the controlled availability of kava.
2. Policy approaches should ensure that local community control is reinforced and maintained over the availability of kava and of the profits acquired from the trade in kava and that the illegal trade is eliminated.
3. Licensees’ should be encouraged to modify their kava management plans to ensure that the controls on kava availability they contain are strictly observed.
4. While no clear evidence for long-term irreversible effects of kava use by Indigenous Australians emerged in this study, some results are suggestive of an increased risk of cardiovascular disease, serious infections, weight loss and poor nutrition and acute effects such as toxicity and withdrawal seizures. The possibility of cardiac events associated with kava use cannot be ruled out. Close monitoring for these potential adverse effects of kava use in Aboriginal communities is recommended in addition to initiatives encouraging moderation in consumption.

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APPENDIX A

APPENDIX A

PUBLICATIONS

APPENDIX B

Northern Territory University's "Rules for the presentation of theses submitted for a professional doctorate or higher degree by research" (section 4.4).

APPENDIX C

**Letter from Director Postgraduate Research Studies
approving thesis format.**

APPENDIX D

APPENDIX D

ETHICS APPROVALS