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SHORT REPORT

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

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Kava, (*Piper methysticum* Forst. f., ''Intoxicating pepper''), is a consciousness-changing muscle relaxant consumed in the Pacific islands and, since 1982, by indigenous Australians in eastern Arnhem Land (Northern Territory, NT) using dried powder imported from Fiji or Tonga.[1] Very heavy use was widespread in Arnhem Land during the 1990s.[1] Circumstantial evidence suggests that kava consumption is associated with ischaemic heart disease (IHD) and sudden cardiac deaths among, particularly, young Aboriginal sportsmen in this population.[2]

METHODS

In a case-control study, cases comprised 83 people admitted to hospital for the first time during 1992–1997 from the region with a medical officer's confirmed diagnosis of IHD (ICD9 Codes 4100–4149). Of these, 25 were admitted on more than one occasion. Up to four randomly selected controls (n = 302) were matched with each case for age, sex, and home locality. NT registries indicated that a further 20 people with no record of hospital admission died with IHD during 1992–1997. These were matched with 75 controls. Comprehensive data were not available to identify IHD morbidity before 1992.

Methods to measure exposure to kava use, alcohol, tobacco, petrol sniffing, cannabis use, and other possible confounding factors, data analysis techniques, and ethics approvals have been described elsewhere.[3]

RESULTS

Adjusting for confounders, odds ratios (OR) for kava use before or during 1992–97 changed from 1.41 (95% CI 0.73 to 2.73, p = 0.303) to 1.51 (0.75 to 3.05, p = 0.247) (table 1). There was no residual confounding effect of age in the multivariate model (OR = 1.50, 0.74 to 3.04), ($\chi^2 = 0.23$, likelihood ratio test, p = 0.635). There was no association with kava use in just those communities where kava had been used for up to 15 years (adjusted OR = 1.75, 0.82 to 3.74, p = 0.140) or when those admitted on more than one occasion (n = 25) were compared with their matched controls (n = 132) (adjusted OR = 2.24, 0.65 to 7.68, p = 0.191).

Twenty who died from IHD without hospital admission and 75 matched controls were combined with 83 known admissions and 302 matched controls. No association with kava use was found (adjusted OR = 1.44, 0.78 to 2.66, p = 0.245) so the results of the analysis of IHD admissions alone were probably not influenced by survival bias.

While the expected association between IHD and tobacco use was not found in the univariate analysis (table 1), it appeared when 36 cases were compared with 158 controls who had no record of kava, alcohol, cannabis, or petrol use (OR = 3.96, 1.08 to 14.49, p = 0.021).

DISCUSSION

There was no clear evidence for an association between kava use and IHD. Twice the risk of IHD was the smallest detectable risk (80% power, 95%CI) in this study. Further research is warranted given the non-significant tendency for approximately 50% increased risk of IHD found in kava users and given the lack of information about kava's effects.

It is not known if an association between kava use and IHD would develop in time. Results of a recent cross sectional study in one community did not support this, as serum concentrations of markers of thrombolytic processes and carotid arterial wall thicknesses were no different in those who had used kava for 1–18 years (median 12 years) when compared with non-users.[1] Moreover, kava has been used for centuries by Pacific peoples with no evidence for an association with heart disease.

In Aboriginal kava drinkers, disrupted lipid profiles and tendencies for raised HDL-cholesterol and LDL-cholesterol,[1] are not clearly indicative of increased atheroma risk.

Aboriginal sportsmen who may already have established IHD may be at higher risk of cardiac events if heavy kava use accompanies vigorous exercise. Heavy kava users may become dehydrated and myocardial ischaemia and sudden cardiac death are possible attributable to abnormal coagulation with increased thrombosis and/or arrhythmias.[2]

Acute effects of kava may also be mediated by neurological mechanisms. Large doses of kava pyrones can lead to abnormal atrioventricular function, consistent with kava's well known muscle relaxing properties.[4] Actions of kava pyrones on voltage gated ion channels, with antagonistic effects on Na^+ and Ca^{2+} currents and modulation of K^+ currents, are of importance for their mood stabilisation properties and these cellular actions show a large overlap with the actions of established mood stabilising drugs.[4] Antipsychotic drugs prolong QTc interval and bind to the potassium rectifier channel, and their use may be associated with torsades de pointes and sudden cardiac death.[5] Similar outcomes with kava use should be considered especially in Aboriginal kava drinkers who may already suffer abnormalities in cardiac output or effective mechanical performance.

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Table 1 Odds ratios and 95% confidence intervals for the association between IHD and kava use and other substance use

Crude odds ratios 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			
	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	0.342
	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			
	Light	5	15	1.91	0.54	6.73	
	Moderate	6	31	0.86	0.29	2.51	0.679
	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			
	Up to 15 cigarettes a day	5	8	3.03	0.72	12.70	
	From 15 up to 25 cigarettes a day	2	33	0.26	0.05	1.29	
	25 cigarettes a day	36	105	1.80	0.80	4.06	0.019
	More than 25 cigarettes 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			
Odds ratios 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	50	199	1.00			
	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	0.469
	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	

*Likelihood ratio test.

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REFERENCES

1 **Clough AR**, Jacups SP, Wang Z, *et al*. Health effects of kava use in an eastern Arnhem Land community. *Intern Med J* 2003;**33**:336–40.
2 **Young M**, Fricker P, Thomson N, *et al*. Sudden death due to ischaemic heart disease in young Aboriginal sportsmen in the Northern Territory, 1982–1986. *Med J Aust* 1999;**170**:425–8.
3 **Clough AR**, Bailie R, Burns CB, *et al*. Validity and utility of community health workers' estimation of kava use. *Aust NZ J Public Health* 2002;**26**:52–7.
4 **Grunze H**, Langosch J, Schirrmacher K, *et al*. Kava pyrones exert effects on neuronal transmission and transmembraneous cation currents similar to established mood stabilizers—a review. *Prog Neuropsychopharmacol Biol Psychiatry* 2001;**25**:1555–70.
5 **Glassman AH**, Bigger JT. Antipsychotic drugs: prolonged QTc interval, torsades de pointes, and sudden death. *Am J Psychiatry* 2001;**158**:1774–82.