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SHORT COMMUNICATION

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

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Heavy kava use has been associated with sudden death in Aboriginal 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 thickness, 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 *vs* 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.
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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 (Brunton, 1989; Lebot *et al*, 1997). Aboriginal Australians in Arnhem land (Northern Territory, NT) have used it in similar ways since 1982 when it was first brought from the Pacific (Cawte, 1985; Clough *et al*, 2000). A possible association between heavy kava use and sudden death in Aboriginal Australians is an unresolved concern (Young *et al*, 1999). While clinical observation suggests that sudden deaths have occurred in Arnhem Land among heavy kava users, no clear evidence for increased risk of ischaemic heart disease was found in a recent case–control study (unpublished data) or of atherogenesis in a cross-sectional study (Clough *et al*, 2003). Poor nutrition, which contributes to high rates of coronary heart disease (CHD) in Aboriginal Australians (Thomson, 1991; Rowley *et al*, 2001), may add to this concern.

In one Arnhem Land community, 6 y after kava was introduced, 'heavy' and 'very heavy' kava users showed signs of malnutrition with 20% lower body weight and 50% less subcutaneous fat (Mathews *et al*, 1988). These same kava users had unusual lipid profiles with elevated high-density lipoprotein (HDL) cholesterol (Mathews *et al*, 1988). In a recent study, continuing kava users were compared with less regular users and a group of nonusers. Lower body mass index (BMI), higher total cholesterol and a tendency for higher HDL-cholesterol were found in kava users, while other CHD risk markers (C-reactive protein and homocysteine) were generally elevated (Clough *et al*, 2003).

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

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Methods

In March 2000, a sample of 98 people (62 males, 36 females) was opportunistically recruited in an eastern Arnhem

Land Aboriginal community (340 people aged >15 y). Of these, 32 males and four females reported using kava; 17 males and nine females had ceased using it 1 y 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. Details of the study setting, methods for assessing exposure to kava and other substance use (alcohol, tobacco, and cannabis), and ethical approvals are described elsewhere (Clough *et al*, 2003). Participants gave written informed consent with procedures explained by health workers.

Sitting and standing heights (stadiometer) and skinfold thicknesses (calipers) were measured and BMI was calculated (Wang *et al*, 2001). Per cent body fat was measured using a Tanita Body Fat (TBF 521) instrument. Plasma samples (nonfasting) were analysed for lipids (Clough *et al*, 2003), carotenoids and micronutrients (Rowley *et al*, 2001).

Variations in continuous outcomes across kava using groups were tested using general linear modelling including tests for a 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

In all, 53% of continuing kava users and just 4% ($n=1$) of past users ($P<0.001$) showed dermopathy characteristic of prolonged use (Norton & Ruze, 1994). Adjusted for age, cholesterol levels beyond a reference range indicating increased CHD risk (Table 1) 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 low-density lipoprotein (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 1 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 1 y. BMI varied significantly by kava use with a nonsignificant tendency for a linear decrease. There were significant linear decreases in percent body fat and subscapular skinfold thickness, but not midupper-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 a significantly lower percent body fat ($P=0.031$) and subscapular skinfold thickness ($P=0.027$).

Table 2 shows that plasma levels of carotenoids were extremely low compared to other populations (Ito *et al*, 1990; Ford & Giles, 2000) for example, they were approximately one-quarter of the carotenoid levels in non-Aboriginal subjects from Melbourne (unpublished data). The 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 nonusers 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.

Discussion

The loss of total and upper body fat (indicated by low percent body fat and subscapular skinfold thickness,

Table 1 Coronary risk profile of survey participants stratified by kava use

	Kava use				
	Never (n = 36)	*Past* (n = 26)	*Current* (n = 36)	P_{ANOVA}	$P_{linear\ trend}$
Male gender (%)	36	65	89	<0.001	<0.001
Age (y)	36 (32–41)	37 (32–42)	36 (33–39)	0.871	–
Total chol. (mmol/l)	4.2 (3.9–4.4)	4.5 (4.2–4.8)	5.3 (5.0–5.5)*†‡	<0.001	<0.001
HDL chol. (mmol/l)	0.86 (0.76–0.96)	0.97 (0.86–1.08)	1.05 (0.95–1.16)*‡	0.010	0.014
LDL chol. (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
BMI (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
Midupper-arm circ. (cm)	29.3 (28.1–30.5)	29.4 (27.9–30.8)	27.9 (26.5–29.2)	0.265	0.144
Triceps skinfold (mm)	14.8 (12.5–17.6)	14.2 (11.6–17.3)	11.9 (9.9–14.4)	<0.001	0.107
Subscapular skinfold (mm)	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 nonusers, with low HDL (≤0.9 mmol/l-M, ≤1.1 mmol/l-F) in 46% of current users and 85% of nonusers.

Table 2 Dietary markers stratified by kava use

	Kava use				
	Never	*Past*	*Current*	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/dl)	884 (819–955)	964 (888–1045)	980 (899–1068)	0.202	0.113
γ-Tocopherol (μg/dl)	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/dl)	1.7 (1.4–2.1)	1.6 (1.2–2.0)	1.4 (1.1–1.7)	0.467	0.225
Lutein + 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 mean (95% confidence interval); antioxidant data are adjusted for gender and plasma cholesterol.
*Significantly different from never users.

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 a significantly lower body fat, low levels of biomarkers of dietary quality that were similar to those already reported for other Aboriginal Australian populations (Lee *et al*, 1995; Rowley *et al*, 2001), 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*, 1988, p 545). In Pacific island kava drinkers evidence for such weight loss, by contrast, is equivocal (Frater, 1952; Ruze, 1990), which along with kava's association with feasting in Pacific traditions (Holmes, 1967; Singh, 1992), implies the likely influence of social factors. Psychosocial factors are plausible causal agents for loss of body fat and are further indicated in Aboriginal Australian kava drinkers. For example, some Arnhem Land aboriginal people have used kava continuously for up to 18 y (Clough *et al*, 2003) and unpublished participants' 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 a severe lack of cardioprotection associated with diets high in vegetables and fruits. We previously reported the tendency for elevated plasma cholesterol, including HDL, associated with kava use, but not triglycerides (Clough *et al*, 2003). However, the cholesterol levels in kava users were unremarkable relative to cholesterol levels in Caucasian populations. While a low HDL in kava users was less likely, the ratio of LDL–HDL cholesterol (data not shown) did not differ across kava-using groups ($P_{\mathrm{ANOVA}} = 0.591$, $P_{\mathrm{linear\ trend}} = 0.872$).

Tobacco, cannabis, and alcohol users generally had cholesterol levels within a reference range. Alcohol was used by 48% of participants and cannabis by 40% and both were associated with kava use (Fisher's exact $P < 0.001$, data not shown). Tobacco was used by 83% of participants but was not associated with kava use (Clough *et al*, 2003) (Fisher's exact $P = 0.110$, data not shown) and generally low antioxidant levels did not vary across kava-using groups (Table 2). 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 (Stone, 1994). Strong confounding effects seem unlikely, however, since triglycerides were not elevated overall (Table 1). Not all alcohol users in the sample used it excessively. Health workers described 44% of alcohol users as 'heavy' users, that is, using > 318 g/month of pure alcohol (data not shown). While these 'heavy' alcohol users also tended to be less likely to have a low HDL (OR = 0.3, 0.1–1.1, $P = 0.064$, age-adjusted), when they were excluded from the analysis, continuing kava users remained less likely to have a low HDL (OR = 0.5, 0.3–0.9, $P = 0.015$). There were no associations between cannabis use and cholesterol levels. Drug-induced intrahepatic cholestasis is known to disrupt lipid profiles (Stone, 1994). But it is not known whether the obstructive hepatic response already reported in these same kava users (Clough *et al*, 2003) could lead to atherogenesis with increased CHD risk (Naschitz *et al*, 2000). Moreover, liver function changes are not necessarily accompanied by weight loss, unlike the dyslipidaemia of *anorexia nervosa* (Stone, 1994). Further research is warranted to consider the inter-relationships of these factors with other measures of CHD risk.

In conclusion, although causal mechanisms are not known, 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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