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De-mythologizing and re-branding of kava as the new 'world drug' of choice

S Apo Aporosa

Abstract

What seemed impossible 50 years ago is today becoming a reality as 'soft drugs' such as cannabis are being decriminalized and accepted for their calming effects as well as their legitimate medicinal properties. Several countries have now made the possession of cannabis legal, with others considering this, while the coffee shops in the Netherlands have been supplying cannabis in different forms for many years. It is now the turn of kava to be re-evaluated, to see whether there are properties in this plant that might be readily substituted for more conventional and harmful drugs, for instance tobacco and alcohol. However, as highlighted by Norton and Ruze (1994), kava like cannabis, has an enduring reputation that still makes it difficult for many to accept. Kava has been mythologized as an illicit alcohol, highly addictive, and causing physical harm. When examining the history of kava use in traditional contexts and considering the evidence now available, it is possible to demythologize this characterization. Looking at the potential benefits, it is time to re-brand kava, not only on the grounds as a relaxant, but in possessing life enhancing medicinal properties and as an alternative to alcohol, understanding that will be beneficial to policy makers, doctors and pharmacists.

Keywords

kava, addiction, alcohol, harm, liver, misunderstanding, myth

Introduction

This article aims to challenge the historical denigration and denial of the benefits of kava. To achieve this, the article will look at the origin of the kava drink and its usage in a traditional context, followed by an analysis of the negative portrayal of kava, from the time of Captain James Cook (1770s) to the present. This will include an analysis of the myth that kava can cause liver damage, induce drunkenness and incapacitate kava users, and lead to addiction. This article will also consider death and harm statistics related to tobacco and alcohol use which are compared with the effects of kava drinking on consumers and those impacted by kava use. The final section will report on the international uptake of kava and new forms of kava use as part of the re-branding that continues to face opposition.

Critical to this discussion is a definition of kava, particularly with kava's recent 'modification' into tablet/capsule form sold from pharmacies and health-food outlets, or the mixing of kava with other substances to create pop-culture foods and beverages. Kava expert Dr Vincent Lebot assists in establishing that kava definition for this article: In a recent interview he stated, 'Kava is kava; it is the traditional beverage prepared by cold water extraction of the ground organs of the plant *Piper Methysticum* [which will be explained shortly], and nothing else.' (Blades, 2018). This also aligns with the Government of Vanuatu's definition of kava within their *Kava Act* (GoV, 2002) and New Zealand where kava is regulated as a 'food' under the *Food Standards Code* (New Zealand Government, 2015).

This article will be useful to policy makers and also doctors and pharmacists who are often a first point of information for those seeking knowledge on the safe use of new drugs and natural medicines.

A brief outline of kava's psychoactive and medicinal properties

Kava, yaqona, sakau, 'ava, 'awa, is a drink made by steeping the wet crushed or dry pounded roots and

The University of Waikato, New Zealand

Corresponding author:
S Apo Aporosa, Te Huataki Waiora School of Health, The University of Waikato, Private Bag 3105, Hamilton 3240, New Zealand.
Email apo.aporosa@waikato.ac.nz

basal stump of the *Piper methysticum* plant in water (Figure 1). The resultant slightly peppery earthy flavoured drink produces a mild relaxant soporific effect (Aporosa and Tomlinson, 2014). Six key psychoactive compounds called kavalactones have been identified as responsible for kava's pharmacological effect. These act on receptors in the central nervous system, causing a slight numbing and slowing in the response time in muscles, limbs and the brain (Singh, 2009).

Kava also plays a significant role in traditional medicine. This is best summarized by Lebot and Cabalion (1988) who present a valuable table informed from across the Pacific in which they list symptoms and the appropriate kava preparation method for each condition (23–29). These include mild local anaesthetic and analgesic effects, and antifungal and antibacterial action (Singh, 2009). Kava's efficacy as an anxiolytic, used in the treatment of generalized anxiety disorder, has been recognized by contemporary pharmacology (Blades, 2016; Sarris et al., 2013), as has its use as an alternative to hormone replacement therapy (HRT) for women (Braun and Cohen, 2010; Cagnacci et al., 2003). More recently kava has been used in cancer research, specifically ovarian, bladder, colon, lung cancer and leukaemia (Lim, 2016). While the medicinal benefits of kava have been widely reported, there is also increased interest in kava as a recreational drink and alternative to alcohol, to the point that it is now being consumed in franchized bar settings in the USA (Showman et al., 2015; Wolinski, 2018). In some bars, kava is mixed with other substances to potentiate effects, a practice several medical professionals have voiced concern over due to risks associated with 'drug interaction' (AddictionResource, 2019). However, kava's increased popularity has been matched by kava suspicion, myth and misreporting, to the point that the Pasifika Medical Association (PMA) was obliged to make this a theme within their 2016 annual conference.[1]

The kava drink today and its origins

Archaeologists, linguists and botanists believe kava originated in northern Vanuatu approximately 3000 years ago (Lebot et al., 1992). It is thought that the spread of kava followed early migrational trade routes as far west as Papua New Guinea, to Hawaii in the east, and Aotearoa New Zealand in the south where it failed to grow (Crowley, 1994). As Lebot and Levesque (1989) explain: 'for kava, dispersal of vegetative propagules by wind or bird is impossible, [and] the plant therefore owes its survival entirely to human distribution of stem cuttings' (234). Together with kava's use in indigenous medicine (Lebot and Cabalion, 1988), the plant in both its raw and drinkable form play significant roles in traditional practice, being widely used to mark life events from birth to death (Aporosa, 2019b). In a number of the Island nations such as Pohnpei (Micronesia), Vanuatu, Fiji, Tonga and Samoa, much of that traditional use remains, whereas in other areas such as Te Au Maohi (the greater Rarotongan island group), French Polynesia and Hawaii, colonial contact and missionization saw kava use reduced and in some cases eliminated altogether (Aporosa, 2014).

Today kava has shifted to the Pacific diaspora. As Pasifikans[2] have continued to use kava in their new home environments, they have introduced new user groups to this iconic drink. In most cases, these new non-Pasifika kava users employ kava purely for recreation, for the purpose of relaxation and/or as a medicine, namely to reduce anxiety and facilitate sleep. However, there are others who have drawn on kava's traditional meanings and sociocultural function to both enhance and expand their sense of identity through the practice of kava drinking (Aporosa, 2015).

Change has also occurred among indigenous users. For instance, extremes between traditional and urban contemporary styles of kava consumption can be found (Corcuran and Brynjolffssen, 2008). Whereas urban contemporary niVanuatu kava drinkers tend to stand and drink, urban Fijian and Tongan kava users, including those in diaspora, tend to mix and serve the beverage from a designated bowl to drinkers sitting on woven mats on the floor (Figure 2). The latter is argued to have greater adherence with Pasifika cultural values, in which some believe it is disrespectful to stand



Figure 1. Dry pounded kava root being mix for drinking (photographer: Todd Henry, 2019).

and drink kava. It can be seen that kava's ongoing use stays within its traditional environments for some, while for others there is a shift in the diaspora with new user groups, who position the drink and its associated practices as identifiably Pasifikan (Aporosa, 2015; Lebot et al., 1997).

Kava myth and misreporting

Myth: Kava is alcohol

The 'kava is alcohol' myth is believed to have started when Johann Forster (1754-1794), a naturalist aboard Captain James Cook's Endeavour, gave kava its botanical name *Piper methysticum*. *Methysticum* is a Greek word meaning 'intoxicating', or according to Forster, 'intoxicating pepper' – *Piper methysticum* (Singh and Blumenthal, 1997; Steinmetz, 1960). Added to this myth, was a report that masticating kava during preparation, 'transformed the starch of the root into sugar, and that this by fermentation turned into alcohol' (Lewin, 1998: 185). Further, after drawing on an early 1800s report from Fiji, Thurn and Warton (1925) were adamant kava was 'liquor . . . [with] its effects being similar to that of laudanum' (102). Norton and Ruze (1994: 93) suggest that early European accounts of kava drinking not only associated kava with alcohol, but also opium, which further maligned kava's reputation. Churchill (2010) suggests that from the outset, kava's botanical name suggested kava had an 'intoxicating quality', and that this made 'it more difficult to correct the error' (57).

The effects of kava 'soporifism'. Kava is not alcoholic; it is 'nonfermented, non-alcoholic, nonopioid, [and] nonhallucinogenic' (Norton and Ruze, 1994: 93), producing 'a pleasant, warm, and cheerful, but lazy feeling, [making people] sociable, though not hilarious or loquacious; the reason is not obscured' (Hocart, 1929: 59). Aronson (2008) removes ambiguity when he stated, 'Reason and consciousness remain unaffected' (183). Professor Peter D'Abbs (1995), from the Darwin School of Medicine, verifies that kava use will 'not lead to violent behavior' often associated with alcohol use, does not 'befuddle the mind and can be used to stimulate clear-headed discussion' (169). Where alcohol



Figure 2. The serving of kava from a designated bowl to drinkers sitting on woven mats on the floor (photographer: Todd Henry, 2017, 2018).

has the ability for some drinkers to: 'release aggressive impulses; if anything kava inhibits or disassociates them. You cannot hate with kava in you', argues Lemert (1967: 333).

Kava versus alcohol. Comments such as kava drinkers remaining clear-headed, and not interfering with reasoning, demonstrate that kava's effects on the brain are quite different to that of alcohol. If a comparison was to be made between alcohol and kava, it would be described as a slightly drunk feeling in the body after consuming high volumes (Aronson, 2008). Cairney et al. (2003) who studied kava and mental clarity, found: 'no impairment in cognitive or saccade function in individuals who were currently heavy kava users, nor was there any impairment in individuals who had been heavy kava users in the past but had abstained for longer than six months' (389). Thomson (2008) adds that 'most people who drink kava for the first time- . . . expend too much effort analysing its effects on them and can be heard muttering that they don't feel a thing' (72).

Kava and addiction

In Lewin's (1998) study of late 19th and early 20th century colonial impressions of kava, he reported that users allegedly craved kava: 'like all other passions of a similar nature, morphinism, alcoholism, etc. The kava drinker is incessantly tormented with the craving for his favourite beverage' (187). Lewin described this before making it clear that: 'I do not, however, consider it probable that kava is the original cause of these afflictions' (187). This though did not prevent Aronson (2008) from quoting Lewin's '. . . incessantly tormented . . .' sentence, for which he did not acknowledge the original author, and therefore linked kava with addiction (183).

Is kava addictive? Various addiction 'specialists' have reported that psychological dependence on kava includes cravings and dysphoria when the drug is withheld (Golan et al., 2008: 268). Adams (1998) reports

> A consultant psychiatrist at the St Giles psychiatric hospital in Suva, in eastern Fiji, Jude Ohaere, sees kava abuse casualties every day . . . Dr Ohaere has no doubt that kava users become dependent on the drug, and warns that the long-term effect of its active ingredients – kava lactones – are unknown. (19)

In another attack on kava following the rise in popularity of kava bars in the USA, Rodriguez (2016) suggests that: 'when someone walks into a Kava bar, the last thing they are thinking of is going away to rehab in the next few months.' He adds that the 'regular use of Kava can lead to a chemical withdrawal syndrome, with some developing a physical dependence very quickly.' This however has been denied by long-term heavy kava users who report that periods of kava abstinence, as part of cultural observance, are common, with users reporting or showing no addictive symptoms during these times. Such commentary aligns with a gathering body of research and ethnographic comment, showing that kava use, even at high volumes and regular use, is not generally addictive (Bilia et al., 2001; Connor et al., 2001; Geier and Konstantinowicz, 2004; Keltner and Folkes, 2005; MediHerb, 2004; Scherer, 1998; Thompson et al., 2004). Drawing on earlier research, Aporosa (2014) summarizes by suggesting that 'if the label ''addiction'' is to be applied to yaqona [kava], I would hesitantly use ''socially addictive'' in the sense that it has been habituated to most aspects of Fijian socialization' (147).

Despite kava being a daily practice in many Pasifika communities (Aporosa, 2019b), assertions that kava is non-addictive is well documented (as shown in the lengthy reference list above). Additionally, Sarris et al. (2013) undertook a double-blind, placebo comparison aimed at kava withdrawal and addiction, reporting 'no addictive qualities or withdrawal issues' (1727). Admittedly, the doses administered (120 mg titrated to 240 mg of kavalactones per day per participant over six weeks) are considerably less than those consumed by traditional and recreational kava drinkers (Sarris et al., 2013: 1727) – amounts that can be more than 30 times those used in the Sarris et al.'s study (Aporosa, 2017b).

Kava addiction – Medical evidence. Of interest is kava's use in several drug-addiction therapy programmes, encapsulated in the title of Steiner's (2001) article, 'Kava as an anti-craving agent', which reports the preliminary results of kava to mitigate alcohol, tobacco and/or cocaine craving. Braun and Cohen (2010) also discuss the value of kava to benzodiazepine withdrawal. They report that kava 'may have an anxiolytic effect beyond the benzodiazepines' (281), and that 'withdrawal symptoms following discontinuation of benzodiazepines occurred somewhat less frequently under treatment with WS®1490 [kava extract], and even if they did occur, the anxiolytic effect remained' (282). Further, kava has been used as part of two District Health Board (New Zealand (NZ)) addiction rehabilitation programmes; one in the Bay of Plenty aimed at alcohol which is now in its seventh year (Crowley, 2015, personal communication) and the other in Marlborough, a NZ smoking cessation programme entitled 'Kavacation' which boasts a 90% success rate (Daunauda, 2016). Moreover, Marotta (2018, personal

communication) reports the use of kava and talk therapy, modelled on traditional Pacific kava use systems as extremely valuable in his work with heroin addicts in Thailand and Massachusetts, USA. Leading kava expert, Dr Vincent Lebot (1991) adds weight to this discussion when stating: 'by pharmacological standards, kava is not classified as a drug, as its consumption never leads to addiction or dependence. It has psychoactive properties but is neither an hallucinogenic nor a stupefacient' (169).

Kava and liver damage

The suggestion that kava damages the liver, first surfaced in early 2000 following reports in Western Europe that 83 patients taking kava tablets died (Schmidt et al., 2005). This led to what is commonly known as the *European Kava* Ban. At the time of the ban, European doctors were estimated to have been prescribing 70 million (tablet) doses of kava daily, with most of this supplied for alleviating anxiety symptoms (Schmidt et al., 2005: 186). The withdrawal of kava from the European markets led to a 12-year court battle which was not resolved until 2014 by the Federal Court of Germany. The final ruling by the Court was that it was unlikely kava had caused the reported deaths, and that liver damage from kava was so rare it was negligible. The Court rejected claims of liver damage caused by kava, and specified that these assertions were a gross misrepresentation of the possible effects (Kuchta et al., 2015; Schmidt, 2014).

Kava and liver damage – The medical evidence. Showman et al. (2015) provide a valuable review of the kava hepatotoxicity claim and counter claim literature (60–61) which includes potential 'Mechanisms of toxicity' (61–63). They summarize that although there is evidence of a link between 'kava and liver toxicity demonstrated *in vivo* and *in vitro*, in the history of Western kava use, toxicity is still considered relatively rare. Only a fraction of the handful of cases reviewed for liver toxicity could be, with any certainty, linked to kava consumption and most of those involved the co-ingestion of other medications/supplements. That means that the incident rate of liver toxicity due to kava is one in 60–125 million patients' (65).

Singh (2014) discusses additional potential mechanisms of toxicity; adulterants added to kava to artificially boost weight to increase sale profit. This can include 'sawdust, flour, and the dregs from the extraction of sugarcane' (42). As Aporosa warned in a recent radio interview, exporters who engage in this type of unethical practice are playing a risky game, one that could have widespread implications should the adulterant contain bacteria, 'salmonella for instance, and if someone gets sick . . . this could threaten kava importation' as it will be 'kava' that will be cited as the health threat and not the adulterant (Kumar et al., 2018, interview: 45 seconds).[3] To assist in safeguarding kava quality, the World Health Organization (WHO) and UN have developed a Kava Codex Alimentarius Quality Standard which should be in place by 2020. Commenting on this Codex, Vanuatu kava expert Dr Vincent Lebot stated Tonga, Fiji, Samoa and Vanuatu were also seeking to register the word 'kava' as a traditional beverage associated with

> healthy and safe raw materials used to prepare the beverage . . . kava is banned in the EU and banned in Australia and we believe this is due to a major misunderstanding regarding what kava is . . . We want to promote kava for what it is, a very healthy traditional beverage . . . If some companies elsewhere want to extract the active ingredients and prepare some capsules or whatever, this is not called kava any more. Like if you put caffeine in a capsule, you cannot call it coffee; if you put in dry raisin peel, you cannot call it wine, and same for tea. Kava is kava; it is the traditional beverage prepared by cold water extraction of the ground organs of the plant *Piper Methysticum*, and nothing else. We want to protect the geographical origins and the healthy quality kava plants we use here on an original basis. (Blades, 2018; also see Procyk and Lebot, 2013)

Linked to concerns of kava hepatotoxicity is an increase in gamma-glutamyl transpeptidase (GGT) levels in the blood following kava use. In their article, Moulds and Malani (2003) first addressed this matter in 2003 when they asked rhetorically: 'How relevant is the finding that some . . . heavy kava drinkers have raised serum GGT levels?', responding: 'raised GGT levels do not necessarily imply ''subclinical'' liver toxicity' (452). When questioned by the author in 2009 about their subclinical liver toxicity comment, former Dean of Fiji School of Medicine, Professor Robert Moulds, commented that the abnormalities can be a concern among doctors who may not be conversant with liver function tests of kava drinkers, pointing to his colleagues article: 'while elevated GGT and white blood cells [lymphocytes] were abnormal [to those unfamiliar with kava's effects on the liver], this does not mean that this abnormality is of concern' (Malani, 2002: 7). Mantesso (2016) also confirms that, kava 'may throw out the liver function a little bit, altering liver enzymes. Now that's not necessarily saying it's causing liver damage.' Moreover, Evans (2009) explains that 'non-steroidal anti-inflammatory drugs, lipid-lowering drugs, antibiotics, histamine blockers (used to treat

excess stomach acid production), antifungal agents, seizure medication, antidepressants, and hormones such as testosterone', can elevate GGT levels, although these continue to be routinely prescribed (24).

Kava versus other drugs

In making the case for kava, it is worth extrapolating the rarity of kava hepatotoxicity (liver damage as mentioned above) against the risk level posed by other commonly prescribed drugs. A comparison with Diazepam, a widely prescribed benzodiazepine that has similar effects to kava, is useful at this point. Schmidt et al. (2005), who investigated 83 kava toxicity reports that had been influential to initiating the *Kava Ban* in Europe, pointed out that: 'only three cases could be attributed to kava with high probability'. Of these cases it was suspected that other factors were responsible for the negative reaction (Schmidt et al., 2005: 182). The study reported 12 'probable' cases of liver failure would account for a kava toxicity rate 'of 0.23 cases per 1 million daily doses' (187). Schmidt and colleagues note that at the time of the *European Kava Ban*, diazepam toxicity rates accounted for 2.12 cases of per million daily doses (187). In another study, kava hepatotoxicity rates were compared with that of Paracetamol/Panadol. In that study, Rasmussen (2005) reported that these commonly prescribed over-the-counter pain medications accounted for 'an estimated 458 deaths due to acute liver failure in the U.S. each year', and summarized that kava was 'dramatically' safer than the popular readily available analgesic's (7).

In an article which considers rates of kava hepatotoxicity, Baker (2011) argues that attempting to calculate risk between kava and commonly prescribed pharmaceutical drugs is ambiguous. For instance, he explains that simply trying to estimate 'the number of people taking a specific medication' is a challenge in and of itself, whereas 'counting cases of adverse reaction' is even more difficult (374). Regardless that the risk determination of well-controlled pharmaceuticals is ambiguous and problematic, these continue to be widely prescribed. Conversely, while 'the frequency of toxicity from any kava-containing substance is exceedingly low; low enough that it can be difficult to ever observe it in the relatively small populations in which kava is traditionally consumed', kava tends to draw a higher level of criticism than a number of well controlled and regularly prescribed pharmaceuticals known to be associated with hepatotoxicity (Baker, 2011: 379–380) such as Diazepam and Paracetamol/Panadol as explained above.

Kava versus tobacco and alcohol. Of even greater relevance in the re-branding of kava, is the risk comparison between kava, alcohol and tobacco. The WHO reports that annually there are approximately 3 million deaths worldwide from alcohol (Poznyak and Rekve, 2018), with this socially accepted substance reported as the leading cause of harm worldwide for 15 to 49 year olds (Griswold et al., 2018), and alcohol addiction the most prevalent form of addiction globally (Degenhardt et al., 2013). Australia, one of the few countries worldwide with heavy kava use restrictions,[4] reported more than 5500 deaths as a direct result of alcohol use in 2014, and over 15,500 from smoking tobacco (Cancer Council Australia, 2016; Gao et al., 2014: vii). Over the last 10 years there has not been a single death worldwide attributed directly to kava.[5] The 2016 WHO kava risk assessment reported that 'on balance, the weight-of-evidence from both a long history of use of kava beverage and from the more recent research findings indicate that it is possible for kava beverage to be consumed with an acceptably low level of health risk' (Abbott, 2016: 26). The use of the terms 'on balance' and 'weight-of-evidence' by the WHO is in contrast to the risks posed by tobacco and alcohol. In a recent drug-harm ranking exercise undertaken in Australia, experts assessed the harm levels of 22 drug substances to both the user and others using the Multi-criteria Decision Analysis (MCDA) methodology. When the scores for both the harm to the user (36) and harm to others (41) were combined (77), 'alcohol was the drug ranked as causing the greatest overall harm', scoring higher than crystal methamphetamine (42/24: harm to user/others respectively), heroine (45/13), tobacco (18/14), cocaine (22/3) and ecstasy (5/2) (Bonomo et al., 2019: 763). Conversely, kava was ranked as the least-most harmful of the 22 assessed substances, with the harm to the user scored at 2, and harm to others 1 (Bonomo et al., 2019: 764). Concerning alcohol and harm to others, an estimated 53 million people in the USA – or 1 in 5 – are reported to annually experience 'secondhand harm' from alcohol use (Nayak et al., 2019) whereas the 'wealth of new evidence on the health effects of exposure to secondhand tobacco smoke' led the WHO (2007) to implement policy recommendations aimed at protecting others from second-hand tobacco smoke (3). Due to the disproportional risk levels of (legal) alcohol and tobacco when compared with kava, weight and balance comparisons as reported by the WHO present kava in a very favourable light.

No drug is harm-free, the case for and against kava

So far we have seen how myths around kava evolved over time. This was the result of the first explorers Eurocentric views of the South Pacific, and mis-information and prejudice that has survived into the 21st

century. There is another aspect to this regarding kava, and that is the cultural disdain for what is seen as an organic as opposed to a synthetic product. Rates (2001) argues that it was 'the Industrial Revolution [starting 200 years ago] and the development of organic chemistry [that] resulted in a preference for synthetic products' (603). He argues the agenda behind this 'preference' is 'the economic power of the pharmaceutical companies . . . [and] industrialised western societies, in which drugs from natural resources were considered either an option for poorly educated or low income people or simply as religious superstition of no pharmacological value' (603).

The assumption in Rates' study is that unless a substance is produced in a lab, it is/was likely to be inferior or even dangerous, regardless of a lengthy history of safe traditional use. This opinion was echoed in the following comment recently published in *Current Neuropharmacology*:

> This review [which included kava] demonstrates that even if psychoactive plants have been known and used from ancient times, there is still limited information regarding subjective and neuro-pharmacological effects and consequent eventual toxicity when plants are used alone or in combination with "classical" drugs of abuse. For this reason, significant safety concerns should be raised on recreational use of these substances. (Graziano et al., 2017: 757)

This is reported despite the 2016 WHO's kava risk assessment which notes a lengthy history of kava use and an 'acceptably low level of health risk' (Abbott, 2016: 26).

Coomber and South (2004) add that regardless of the value that a number of traditional medications may have to medical advancement, contemporary Western discourse continues to link these traditional substances with 'backwardness or underdevelopment' (18). Escobar (1988) sees that the oppositional binaries of 'us and them', 'primitive/modern', 'backward versus developed' becomes a 'fictitious construct, an omnipresent . . . discourse . . . of power' propagated by the Eurocentric view that positions anything deemed to counter modernity and economic development as 'primitive' (429). Therefore, cultural practices and traditional medicines like kava in its natural form become threats that must be regulated or eliminated.

Kovaleva (2016) discusses the perceived 'threat' that alternative safer substances such as kava pose to the alcohol industry, an industry that has worked hard to 'normalise' alcohol in contrast to alternative substances. 'In spite of the statistics that exemplify the financial and social ramifications of alcohol consumption, alcohol remains readily accessible and available' comments Hardwick (2019), who adds that drinking is frequently considered a 'rite of passage . . . and excessive consumption often normalised' by sectors of society. Moreover, Petticrew et al. (2018) argue the alcohol industry actively uses deception and deliberately 'misrepresents scientific evidence' related to the health implications of alcohol use in a similar manner to the tobacco industry (300).

The case against tobacco and alcohol. While unconscious Eurocentrism and the power of the alcohol and tobacco industry is argued as contributing to the negative framing of 'natural' drugs such as kava as a threat and vicariously unsafe, it would be naïve to assume that kava is completely harmless. No drug is harm-free and neither is this article suggesting kava provides the ultimate, idyllic alternative to all substances. As discussed earlier, the recent Australian MCDA drug-ranking exercise acknowledged that kava has some level of harm, albeit extremely small, to both the user and those associated with the user (Bonomo et al., 2019). Instead, what is being argued is that kava's negative reports be considered alongside the scientific evidence, and that the risk levels be weighed and reported accurately. Although side effects from kava are rarely reported and considered to be minimal, when compared against the health and socio-cultural implications of even moderate alcohol consumption for instance, kava rates extremely well.

As an illustration of the potential harm caused by alcohol misuse in the United Kingdom, leading neuropsychopharmacologist and former drug advisor to the UK Government, Professor David Nutt, stated, 'when the harm to the user and the harm to others are weighed up, alcohol is more harmful than heroin, crack, and methamphetamine' (Whelan, 2014; also see Nutt et al., 2010). Addiction specialist Jeremy McMinn continues: 'alcohol is in essence a Class B drug [meaning it has a very high risk of harm], but it is so pervasive and traditional that no one sees it like that' (Whelan, 2014). Castles (2015) reported that on New Year's Eve (2014/15), alcohol accounted for a tripling of personal injuries and poisoning incidents attended by St. John Ambulance (New Zealand).[6]

The observations of Nutt, McMinn and Castles together with the earlier discussion on Australian alcohol and tobacco death rates, when set against the claim that kava is the cause of liver damage in which kava hepatotoxicity rates are compared with Diazapam and Paracetomol/Panadol, need to be heeded. Since kava cannot be attributed directly to any deaths worldwide in the past 10 years, and the WHO reports kava risk to be at 'an acceptably low level', this suggests that kava potentially fits more with the side-effects of coffee, having an insignificant risk. Kava expert Dr Vincent

Lebot concurs, stating, 'drinking kava is no different than drinking coffee', although warns, 'If you abuse coffee, you might have side effects [too]' (Scaccia, 2018). Having said this though, the WHO kava risk assessment asks that 'further studies are needed to define the parameters necessary to ensure safe use of kava beverage' (Abbott, 2016: 26).

Re-branding kava – 21st century myth making

Almost 20 years ago, Lebot et al. (1997) made a bold prediction that kava would one day become a 'world drug' (202), taking its place as a possible 'ethnic Valium or alcohol' (210). That prediction appears to now be a reality, with online magazine *VinePair* recently announcing that kava is 'officially trending' owing to widespread availability in bars in the USA (Wolinski, 2018). Kava is franchized under names such as *Kavasutra* and *Root of Happiness Kava Bar*, where it is promoted as a natural relaxant and an alternative to alcohol (Basit, 2016; Rechenberg, 2016; Scaccia, 2018). That trending is seen also in New Zealand, Japan (see the 'Fiji Bar' in Osaka, Nand, 2018), Hong Kong, France and Thailand (Aporosa, 2015; Bolatagici, 2011; Wihongi, 2018, personal communication). Increased use has however spawned a plethora of new myths, including sensational news headlines that kava can 'kill' (Island Business, 1989: 51; Rodriguez, 2016; Stanton, 2017; Yunupingu, 1987: 15). This included a recent exaggerated report followed the drinking of kava by the United Kingdoms' Prince Harry during the Royal Visit to Fiji in which a Professor of pharmacology suggested the Monarchs actions were 'dangerous . . . [and] foolish' as he was 'risk[ing] . . . liver damage from kava' (Dymond, 2018).

As if also foreseeing this rise of kava misinformation, Lebot et al. (1997: 202) have attempted to address the matter of kava's misrepresentation, suggesting claims of harm and reports of 'kava as killer' were 'dubiously simplistic' and 'comprised erroneous statement[s]'.[7] Braun and Cohen (2010), referring to inaccurate reporting on kava, state: 'As in the popular press, the medical press is prone to creating sensational headlines to attract interest, but this can lead to inaccurate assumptions' (12; also see Note 1). Misreporting and discounting of the facts regarding kava have made it extremely difficult for academics, let alone the public, to distinguish fact from fiction, harm from health, positive from negative. Moreover, the misreporting of kava fact in peer review publications (e.g. Barguil et al., 2013; Procyshyn et al., 2017; Stolerman, 2010) has been unhelpful for doctors and pharmacists who are often a first point of information for those seeking knowledge on new medicine safety and use (Maclennan et al., 2016).

Conclusion

This article has looked at the history of kava use in the Pacific, the variation in use across the diaspora and how today kava may have many medicinal applications. While traditionally kava beverage is part of traditional rites of passage, today kava is also seen as a reminder of home when consumed away from the Islands within the Pasifika diaspora. Taking kava is however still mythologized by many, on grounds of inciting drunkenness, addiction and physical harm. However, medical evidence is plain, that kava is non-alcoholic, non-addictive, does not cause liver failure and according to the WHO, has not been the direct cause of any fatalities for the past 10 years worldwide. This differs from statistics for the tobacco and alcohol industry, which according to studies in Australia alone, has been the cause of many thousands of mortalities each year.

While it is interesting to see the emerging markets for different kava products appearing in diverse locations such as the US, Asia and Europe there is still an antagonism towards kava. With those making the claims unable to counter with scientific evidence of kava's properties, and a growing interest in natural products less harmful than traditional recreational drugs, it is only a matter of time before we see kava in even more countries in the years ahead, valued for not only for its calming effects as the new 'world drug' of choice but its array of medicinal benefits.

Declaration of conflicting interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: With the exception that the author is of Fijian ancestry, frequently uses kava as part of his culture, and has previously farmed kava in rural Fiji, no substantial conflicts of interest are reported by the author.

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ORCID iD

S Apo Aporosa https://orcid.org/0000-0002-4490-9545

Notes

1. On 25 August 2016, the PMA held a panel within their three-day conference entitled, 'Kava—killer or cure', aimed at addressing some of the myths of kava related to health. At this panel, Dr Ifiraimi Waqainabete, then a surgeon at the Colonial War Memorial Hospital in Suva,

Fiji, and currently the Minister of Health, described concerns regarding the high number of young male Fijian kava drinkers who were presenting at the hospital with liver abscesses. Although Dr Waqainabete made it clear that it was *not* kava that caused these abscesses, but poor water quality and hygiene, approximately 3 hours after his presentation, Television New Zealand's 6 p.m. news reported a direct link between kava and liver abscesses, quoting Dr Waqainabete (Dreaver, 2016). That news report was watched at the conference venue by more than 100 attendees, some of whom were medical professionals sitting drinking kava. This caused a great deal of discussion and criticism, mainly because the article had suggested kava, and not poor water quality and hygiene, was the cause of these liver abscess. The author contacted the reporter and challenge her regarding this and was advised that no correction or apology would be made.

2. Pasifika/Pasifikan is a term often used in Aotearoa New Zealand and Australia to denote those of Pacific Island ancestry as a collective and/or those who live in a 'foreign' country, whether as visitors, recent migrants, or even those born in that 'foreign' country, who identify first and foremost with their ancestral homeland in the Pacific (Aporosa, 2015).
3. As if to confirm Aporosa's warning, a few months after his comment, Duff (2018) reported the 'mandatory recall' of Kratom (*Mitragyna speciosa*) – a natural pain reliever and recreation drug from Asia – after '200 individuals . . . developed *Salmonella* infections' following Kratom use.
4. Until recently, The United Arab Emirates (UAE), Poland and Australia were the last remaining countries to maintain bans and/or restricts on kava. The UAE has 'a very strict, zero-tolerance anti-drugs policy' which includes kava (Emerites.com, 2018). This is unlikely to change. Until late 2017, kava in Poland was 'listed in the same category as heroin, cocaine, amphetamine, LSD and so on' (Garae, 2017), although following a social media driven campaign, Poland legalized kava mid 2018 (Garae, 2018). The kava situation in Australia is vastly more interesting and complex, particularly as it was introduced to the indigenous peoples of the Northern Territories in the 1980 as part of a '"harm reduction" measure' aimed at curbing alcohol use (Hunter and D'Abbs, 2003: 333). As Professor Peter D'Abbs (1995) from the Darwin School of Medicine commented, that initiative proved valuable as kava 'did not befuddle the mind and could therefore be used to stimulate "clear-headed" discussions' (169), interaction that also melded well with 'traditional drinking practices, . . . [and] their [Aboriginal] attendant social control mechanisms' (168). Additionally, the unregulated availability of kava in Australia provided the Pacific diasporic community with their indigenous substance, which also reduced alcohol use and the socio-cultural problems associated with it (Pinomi, 2008). Following regulatory change in 2007, unrestricted access to kava altered. Kava in Australia now falls under one of two regulatory systems: an amendment to the *Therapeutic Goods Administration* (2007) which 'effectively terminat[ed] the supply and availability of kava to Indigenous people in Arnhem Land' making kava possession in Australia's Northern Territories illegal (Urquhart and Thomson, 2008); and a 2007 amendment to the *Customs (Prohibited Imports) Regulations 1956*. This essentially prohibited the advertisement and sale of kava whereas importation was strictly limited to scientific and medical purposes. The only concession was that passengers arriving into the country would be permitted to bring with them 2 kilograms (kg) of powdered kava without a permit provided they were over 18 years of age and the kava was packed in their personal baggage (Australian Customs and Border Protection Service, 2011). According to the Australian Department of Health and Aging (2011), this 2 kg allowance was to be used to recognize the cultural importance of kava to the Pacific Island community residing in Australia. This concession though did not extend to the Northern Territories where a complete ban remained in place. For more on this topic, including why change occurred and the consequences, see Aporosa (2014: 161–162). The Australian Government is currently considering increasing the 2 kg kava allowance to 4 kg. Some commentators are questioning why a kava restriction exists at all, particularly when similar restrictions do not apply to alcohol in Australia (Aporosa, 2019a). This argument appears to have great merit considering the recent Australian drug-harm assessment which ranked alcohol and kava at opposing ends of the risk scale (Bonomo et al., 2019: 764).
5. Indirectly, kava has been reported as 'contributing' to death. For instance, Ketola et al. (2015) report the use of an intravenous kavalactone ethanol cocktail mix to commit suicide by a victim with a history of 'depression and . . . suicidal[ity]' (e8). They add, 'the oral administration of kavalactones has generally been considered to be fairly safe, but our case implies that an intravenous injection of these compounds can be fatal even though the concentrations in the femoral blood were about the same as in non-fatal cases' (e11). Additionally, Tarbah et al. (2003) report the death of a man following 'head injuries caused by a falling wall after the person had consumed kava together with cannabis'. This raises several questions regarding the action of kava in these two fatalities including: was it the intravenous use of kava specifically that led to death, or other conditions related to the cocktail mix; and, why was kava identified as responsible for death by a 'falling wall'? Death as a result of falling from, or into, a wall as a result of kava use has greater merit. In a more direct but confusing report, Barguil et al. (2013) discuss what they call 'post-kava session sudden death syndrome' (165). This 'syndrome', or the possibility of an association between kava consumption and ischaemic heart disease (IHD), was first investigated by Clough et al. (2004). In that case–control study, they reported, 'There is no clear evidence for an association between kava use and IHD' (140). Moreover, they doubted such a causal relationship 'would develop in time. . . [as] kava has been used for centuries by Pacific peoples with no evidence for an association with heart disease.' (140) Nine years later, Barguil et al. (2013) made a brief but sensational claim, stating that

kava caused 'post-kava session sudden death syndrome' and cited Clough and colleagues to support that claim. Barguil et al. explained that nine people had died as a result of 'post-kava session sudden death syndrome' over a 13-year period in New Caledonia. However, they noted that 'No autopsies were carried out' and six of the nine deceased had been 'heavy smokers, [had] severe hypertension, sleep apnoea, cardiac arrhythmia, asthma, [and/or a] family history of sudden death' (167). This claim was published as fact in a peer reviewed journal.

6. The online link provided as part of the Castles (2015) reference does not include the One News (TVNZ) article in which Castles stated, 'St Johns workload triples on New Year's Eve with the majority of that work being alcohol related', a comment substantiated in an interview of St. John Ambulance Operations Manager.
7. This article has focused on three of the most common kava myths. For commentary on additional myths, misinformation and uninformed opinion such as, 'kava tastes like mud', 'kava causes skin problems', 'kava takes men away from their families', and 'kava is un-Godly, anti-Christian and linked to pagan spiritualism', this is discussed in Aporosa (2017a).

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