Improving road safety and health:

Understanding kava's impact on driver fitness

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Executive Summary

Kava (*Piper methysticum*) is a traditional and culturally significant Pacific Island beverage that produces a soporific relaxant effect. Kava's psychopharmacological action is similar to the anti-anxiety drug Benzodiazepine. Traditional users often consume the kava drink at volumes as much as 30 times greater than pharmacologically recommended doses. Prompted by concerns regarding driver impairment post kava drinking, a study was undertaken replicating traditional kava sessions in terms of duration and kava consumption, to investigate the effects of kava on driving capability.

Kava consumers (n=20) attended two six-hour kava sessions, each participant drinking an average 3.52 litres of kava. A non-kava consuming control group (n=20) was included in the study. At baseline all the participants completed computerised industry standard driver safety assessment tests (Vienna Test System: Traffic WAFA Alertness and WAFG Divided Attention) to measure reaction time, perception and attention. Re-testing was conducted at hourly intervals over the six-hour period. Pre/post analysis compared person to person and between groups change. Statistical modeling was based on ANOVA, independent t-tests and Bayesian analysis.

Data analysis indicated no statistically significant ($p<0.05$) difference between reaction time and divided attention, both within individuals and between groups, at any measurement point over the six-hour testing period. The mean reaction time and divided attention time at the baseline measurement was 249.95msec (milliseconds) and 583.58msec respectively. The control and active group mean reaction time at the final test was 256.70msec and 271.8msec respectively. The mean divided attention times for the control and active groups at the final test were 499.75msec and 568.32msec.

Kava at traditional consumption volumes was not correlated to response latency or impairment on perception and attention tasks, when measured using authoritative driver assessment tests. From this it could be inferred that kava use at traditionally influenced consumption volumes does not compromise driver safety. However, it can be argued that

current industry standard accepted cognitive measures of driver safety, assess impairment from the use of euphoric and hallucinogenic drug substances (such as alcohol and marijuana), and fail to account for kava's unique form of 'intoxication'. Further research beyond the assessment of these two cognitive functions is, it seems, desirable to better understand if kava has any effect on driver ability.

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1 Introduction

Kava drinking has been central to the traditional practices of Pacific Island peoples for more than two thousand years. More recently, kava consumption has moved from the Pacific Islands to New Zealand (NZ), Australia, Canada, the USA and Europe as the Pacific diaspora has spread across the world. This has in turn influenced kava use by non-Pacific Islanders who, in some cases, reflect traditional use, consuming volumes as high as 30 times the pharmacologically recommended daily dose. Increased kava consumption has also led to an increase in driving to and from kava venues, with the police in NZ and the Pacific reporting rising numbers of suspected kava 'intoxicated' drivers. This report documents a study that measured impairment during and following the consumption of kava with the aim of understanding driver ability to improve road safety.

2 Background

Piper methysticum, or the kava plant (Figure 1), is believed to have originated in Northern Vanuatu (Lebot et al., 1992) and was subsequently transported throughout Eastern Pacific Oceania and areas of Western Pacific Oceania by early voyagers and traders from approximately 3000 years ago (Crowley, 1994; Kirsh & Green, 2001). The crushed root and basal stump of the kava plant are steeped in water to make a traditional drink (Figure 2). It should be noted that the kava plant and drink are culturally significant to many Pacific people, being consumed at almost every major event from birth to death (Aporosa, 2014b). In Fiji and Eastern Pacific Oceania (Polynesia), kava drink is frequently used to facilitate *talanoa* (culturally guided discussion) at *kalapu*'s (traditionally influenced kava drinking venues[1]), where drinkers sit cross-legged on woven mats with the kava served from a centrally located *kumete* (wooden kava bowl) (Figure 3). The average duration for a Fijian, or Polynesian kava session, is approximately six hours, in which drinkers can consume up to 3.6 litres (6.33 pints) (Aporosa, 2014b).

[1] *Kalapu* is a Tongan word literally meaning 'club'. *Kalapu* is increasingly being adopted (particularly in NZ) by ethnicities (other than Tongans) to indicate or refer to traditionally influenced kava drinking venues and spaces.

Figure 1: *Piper methysticum* (kava), approximately 3 years of age.

(Aporosa, 2008, p.21).

(Singh, 2004b, p.61).

Figure 2: Contemporary mixing of kava: The ground kava roots (and/or basal stump) in a silk bag being mixed through water in a *kumete*. Cups made from coconut shell are visible.

(Payson, 2008, p.127)

Figure 3: Kava drinking in rural Fiji and mixed ethnicity kava drinking at a New Zealand *kalapu*.

(Author, 2009)

(Author, 2017)

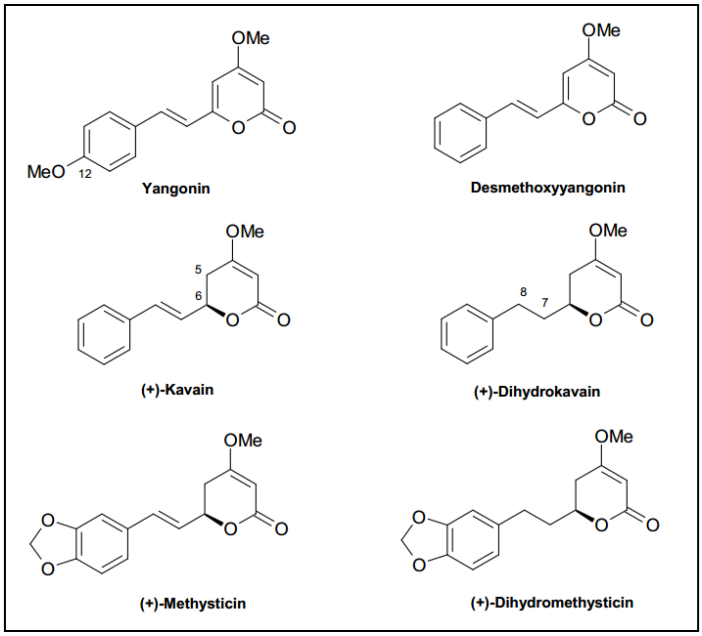
Kava as a drink was relatively unknown outside of the Pacific Islands and diasporic Pacific communities until the early 2000s, when a report in Europe claimed 83 patients taking pharmaceutically manufactured kava tablets had died from hepatotoxicity or liver damage (Schmidt et al., 2005; Showman et al., 2015). This led to what is commonly known as the 'European Kava Ban', and the withdrawal of kava from European markets. This had the effect of raising the profile of kava to the point where it was mentioned in medical and media reports, linking aqueous kava with hepatotoxicity.

In 2014, following a 12-year court battle, the kava 'ban' was overturned by the Federal Court of Germany. The Court ruled that it was unlikely kava had caused the reported deaths, and that liver damage from kava was so rare it was negligible. The Court added that claims of kava being responsible for liver related deaths was a misrepresentation of the possible effects (Schmidt, 2014). The 2016 World Health Organisation's (WHO) kava risk assessment report states: "On balance, the weight-of-evidence from both a long history of use of kava beverage and from the more recent research findings indicates that it is possible for kava beverage to be consumed with an acceptably low level of health risk." (Abbott, p.26; also see Teschke, 2010) Although the reputation of kava is slowly recovering, the belief that kava damages the liver remains, a myth that continues to be perpetuated in some authoritative publications (for instance, see Graziano et al., 2017, p.754; Procyshyn et al., 2017, p.390). Other kava myths include hallucinogenic effects, and that it is addictive and causes a damaging skin condition. *(For more on these misunderstanding and other kava myths, please see Aporosa, 2016).*

Kava contains a number of active compounds called kavalactones, of which six are reported as being responsible for its psychoactive affect (Lebot & Levesque, 1996; Dragull et al., 2006). Kavalactones have each been allocated a number to aid chemotype classification. The following kavalactones are prefaced with their chemotype number and followed with their abbreviation: 1. *demethoxy-yangonin* (DMY); 2. *dihydro kavain* (DHK); 3. *yangonin* (YAN); 4. *kavain* (KAV); 5. *dihydromethysticin* (DHM); and, 6. *methysticin* (METH) (see Figure 4). Pharmacologists explain that kava's blocking of the calcium ion channels leads to a reduction of

neurotransmitter release excitation, and the potentiation of $GABA_A$ through enhanced ligand binding to the GABA (*gamma-Aminobutyric acid*) receptors. This creates a reduction in the neuronal re-uptake of noradrenaline responders and possibly dopamine, leading to a reversal of monoamine oxidase (MAO) B inhibition (Ligresti et al., 2012; Lim, 2016). Although this explanation implies a high level of knowledge regarding kavalactone psychopharmacology, Bwarenaba and colleagues (2017) warn that kavalactone "modes of action are not fully understood" (p.1), with even less known about "the neurophysiological mechanisms associated with kavalactone metabolism" (p.5). Additionally, the WHO also admit to large knowledge gaps concerning kava ethnobotany and psychopharmacology, and have requested research on these factors related to "human health effects" (Abbott, 2016, p.viii).

Figure 4: Chemical structure of the six major kavalactones present in kava beverage made from the roots and basal stump. Me = methyl group.



(Dragull et al., 2006, p.21).

Kava users describe kava as producing a subtle sense of relaxation and sociability without impairing judgment or causing disinhibition, as experienced with alcohol (Aporosa & Tomlinson, 2014). Kava is neither alcoholic nor hallucinogenic, and its effects are different to those of alcohol or most other euphoria inducing drugs (Aporosa, 2011). Therefore, kava 'intoxication' is

a unique experience, one which is frequently criticized by new users who report, “they don't feel a thing” (Thomson, 1999, p.72).

Kavalactones within the plant and drink are responsible for kava’s use in traditional medicine (Lebot & Cabalion, 1988). This includes anti-stress inhibitors recognized by Western pharmacology. This has led to kava being prescribed (ME, 2017) as a non-addictive anxiolytic alternative to Benzodiazepine, in the treatment of Generalised Anxiety Disorder (GAD) (Sarris et al., 2013; Lim, 2016). Kava prescribed for GAD is typically administered in tablet (and occasionally syrup extract) form, and restricted to no more than 300mgs of kavalactones per day. This complies with pharmacological guidelines (MediHerb, 1994), providing therapeutic value without causing cognitive impairment (Mills & Bone, 2005).

Kava’s anti-anxiety affect, together with its attraction as an alcohol alternative and ‘social lubricant’, has led to its use by an increasing number of non-Pacific ethnicities, some of whom also engage in lengthy traditional-style kava sessions consuming quantities similar to Pacific peoples (Aporosa, 2015; Showman et al., 2015; Wolinski, 2018) (see Figure 5). However, unlike prescribed kava tablets, kavalactone quantities ingested during ‘average’ drinking sessions in Fiji, often equate to more than 8,000mg of kavalactones, or 30 times the pharmacologically recommended daily dose (Aporosa, 2008; Aporosa & Tomlinson, 2014; Showman et al., 2015).

Figure 5: Non-Pacific kava users in Auckland, New Zealand.

(Henry, 2018).

LaPorte and his team (2011) were the first to complete a systematic review to assess the effect of kava on cognition. In most of the studies they reviewed, participants received between 200 – 300mg (tablet doses) of kavalactones per day. LaPorte et al. (2011) state that due to inconsistent "study designs and use of control" (p.108), it was difficult to establish the psychopharmacological effect of kava. For instance, in one of the studies they reviewed, it was suggested that acute kava use increased body sway and significantly impaired visual attention, whereas in another study it was reported that: "kava significantly enhanced visual attention and working memory" (p.110). They concluded: "kava has non-deleterious effects on cognition during acute administration or produces reduced visual attention at higher doses during cognitive demand." (p.110) However, while LaPorte and colleagues cautioned that until "a more detailed account of the risk benefit ratio of kava on cognition" has been established, "caution is advised when driving and operating heavy machinery" (p.110).

In a recent study from New Zealand (NZ), 20% of drivers reported taking drugs known to interfere with their driver safety within three hours of driving (Starkey et al., 2016). Drug and alcohol-driving prevalence in NZ is a significant health and safety issue and is estimated to have an annual 'social cost' of $564m (MoT, 2017, p.4). Injury resulting from road traffic accidents is the leading cause of hospitalisation for Pacific men and women (Slack et al., 2009). Hospitalisation as a result of drug use among Pacific peoples is also higher than the general population, and increases significantly with age (Slack et al., 2009). Research focused on Tongan kava drinkers, report that it is common for 70% to drive home from kava drinking sites (Maneze et al., 2008), travel that can include inter-city driving (Aporosa, 2018).

Over the past five years the NZ police have reported increased numbers of kava 'intoxicated' drivers (Morgan, 2014, 2017; Welsh, 2016). Further, the New Zealand Institute of Environmental Science and Research (NZESR) reports the increased presence of kavalactones in the blood of deceased motor vehicle accident victims (Poulsen et al., 2012). By combining statistics on kava use from the NZ Alcohol and Drug Use Survey with two ethnographic studies, it is argued that there are more than 15,000 kava users driving motor vehicles following lengthy

kava drinking sessions on an average Friday or Saturday night in NZ (Aporosa, 2015). It is presumed that the high Pacific hospitalisation rates resulting from road traffic accidents and drug use, have an as-yet unrecognised link with kava use and driving. This potentially puts a large number of Pacific peoples and other road users at high risk of injury.

To date, there has only been one successful kava-driving prosecution in NZ (DCNZ, 2000), and a small number overseas (Swenson, 1996; Jolly, 2009). While this subject is of concern to road safety commentators and law enforcement agencies (Morgan, 2014, 2017; Wainiqolo et al., 2015; Welsh, 2016), the reality is that very little is known about the effects of kava at traditionally consumed volumes, on cognitive faculties related to driver safety. Additionally, when LaPorte et al.'s (2011) limited understanding regarding kava cognition is added to Bwarenaba and colleagues (2017) concerns about kava psychopharmacology and neurophysiology, this points to the urgency to undertake research in this field.

2.1 Research Aim

This study, the first of its kind, aims to measure reaction time and divided attention during and immediately following traditionally influenced kava consumption, and apply the results to safe driver functionality, to better understand kava related cognition, driver safety and coordination. It is anticipated this information will improve road safety, reduce injury and hospitalisation, and the related economic and social costs, to positively impact on the health of Pacific peoples and other road users both in NZ and internationally.

2.2 Hypothesis

Kava consumers (active group) show changes in (1) reaction time and (2) divided attention compared with the control group.

3 Methodology

This study was based at the University of Waikato's Anthropology Programme and School of Psychology Traffic and Road Safety Research Group (TARS). Ethics approval for this

study was granted by the University of Waikato's Human Research Ethics Committee (HREC [Health] #34).

This study is aimed at measuring the cognitive effects during and following the ingestion of high volumes of kava as consumed in traditional settings. Underpinning the methodology is the Post-Development Pasifika methodological framework, developed by Aporosa (2014a). The framework is guided by Fijian cultural respect based values and appropriate practice which can be applied across Polynesia. Additionally, the framework encourages and endorses naturalized research settings as sites of participant engagement.

The number of participants necessary to achieve statistical significance was influenced by McCready (1996). McCready suggests that in experimental projects that involve psychometric testing, studies with a similar research focus should be used to guide sample sizes. Nine studies that measured cognition in kava users following minute pharmacologically recommended doses were consulted (Mathews, et al., 1988; Russell et al., 1987; Saletu et al., 1989; Münte et al., 1993; Prescott et al., 1993; Heinze et al., 1994; Schelosky et al., 1995; Foo & Lemon, 1997; Spillane et al., 1997). Heinze et al.'s 1994 study, was identified as the most influential, as it measured effects similar to the aims of this study i.e. driver ability. The Heinze research utilised twelve participants and provided statistically robust effect measured data. This present study chose to more than double this number.

3.1 Participants

Thirty kava using participants, who will be referred to as the 'active' group, were recruited from *kalapu*'s in Hamilton and Auckland. An additional thirty non-kava drinking participants, referred to as the 'control' group, were recruited by word of mouth and through online advertisements and notice boards at the University of Waikato.

3.1.1 Participant eligibility

The initial recruitment criteria were for participants to be over 18 years of age, and who held a full (unrestricted) driver's licence. This was followed by completion of an eligibility screening form. This was aimed at ensuring that all participants were in good health and an absence of neurological or psychological conditions such as previous head injury, concussion or psychotic disorders. On the eligibly screening form, participants were asked a range of simple questions related to the health matters above, and asked to tick either 'yes' or 'no'. Participants were also screened for anxiolytic or sleep medication use.

Active participants were also questioned about their kava use. This allowed novice kava users to be identified and weeded out, as the study required those who could demonstrate regular use of kava at high consumption volumes over multiple hours. Following selection for participation, the active participants were requested not to consume any kava over the four days prior to testing. Drawing on *in vivo* experiments with mice and rats, Singh (2004c) states that kava has a drug half-life of nine hours. This suggests it takes as many as 90 hours, or almost four days, for kava to be eliminated from the body (Aporosa, 2008). The use of 'suggests' is deliberate due to Bwarenaba et al.'s (2017) observations regarding limited kava psychopharmacology. Due to the increasing use of kava by non-Pacific peoples, 'other' ethnicities in addition to Pacific Islanders were also recruited, and included, in both the active and control groups.

Eligibility screening of control participants also sought information on whether they had ever consumed kava, and if so, how recently. In a few cases, where control participants had consumed kava within six months of testing, they were made ineligible for inclusion in the study to ensure all control participants were kava free. Nine of the 20 control participants acknowledged previous kava use, most commonly as part of either a cultural experience at the University of Waikato or whilst on holiday in Fiji. However, none of them had consumed kava within the past 12 months.

Individuals who met test eligibility following screening were given an information sheet explaining the research aims, procedures, expected time commitment and requirement not to consume alcohol within 24 hours prior to testing. The control participants were also reminded that they must remain kava free to be eligible for testing, whereas the active participants were advised to cease kava use four days out from testing. One week prior to testing, the computer and test software were given dry-run, in the presence of an Information Technology specialist.

3.1.2 Participant demographics

Sixty-seven participants were initially screened for recruitment with seven subsequently not meeting the criteria. This provided three groups of twenty participants with each group comprising ten active kava drinkers and ten control non-drinkers. Shortly after the start of the first test session there was a mass computer failure, which led to the elimination of group one from the study. Of the remaining 40 participants who progressed to full testing, their average age was 35.2 years old (SD = 9.8). Average age for those in the 'active' groups was 35.3 years (SD = 9.5) and control 35.1 years (SD = 10.4). Participants were mostly male with the exception of two females in both the 'active' and 'control' groups.

The ethnic make-up of the 40 participants were: twelve Māori (indigenous New Zealanders), who accounted for the largest ethnic representation of which four were active participants; six each identified as Tongan and Fijian, of which all were active participants; five New Zealand Europeans (one of whom was active); four Samoan's (one being active); two Papua New Guineans (both control); one each of Rarotongan, Indian, Hawaiian and Afrikan descent, all of whom were control participants; and, one Amer-Indian who was an active participant.

Thirty-six of the participants were male. There were two Tongan females who were active participants and two Māori females who were both control participants.

3.1.3 Research participant test preparation

On each of the three testing days, research assistants transported the participants from their homes in vans to a lecture room (see Figure 6) adjacent to a psychology computer lab at the University of Waikato. The participants arrived at 6pm allowing time to be briefed on the study procedures and sign informed consents. It was made clear to all the participants that should they wish to withdraw from the study, they were free to do so at any time and a taxi would be ordered to transport them home. No participants withdrew from the study.

Active participants were also reminded that they would be served kava at precise intervals six times per hour for six hours, and that at each serving they were free to choose their serve quantity which ranged from decline to 25mls (milliliters), 50mls, 57mls and 100mls (equivalent to 0.2 pints) (see measuring tubes on top rim of *kumete* in Figure 6). The participants were advised that their kava intake would be recorded by one of the two research assistants aiding the chief investigator. In addition to this, research assistants were also asked to make notes of any unusual behavior amongst the participants, as the test event proceeded. The participants were also reminded that they would undergo computer-based psychometric testing in the adjacent computer lab on the hour over the test period.

Figure 6: Kava consumption venue in lecture room adjacent to computer lab.

(Author, 2009)

All research participants were invited to partake of snack foods and non-alcoholic drinks at their leisure during the test period. These consisted of typical kava *chasers*, or food items consumed during a regular kava session. These included salted potatoes chips and peanuts, apples and pears, and lollipops (a boiled sweet on the end of a stick). Drinks consisted of water, sports rehydration drinks, lemonade and Coco-Cola. Participants were asked not to consume energy drinks or caffeine. Additionally, the participants were informed that they were free to move about and leave the lecture room to use the toilet, although the active group members were to be present in the lecture room at kava serving times, and all participants were to be available on the hour for testing in the computer lab. Although the study adhered to some strict conditions (such as driver's licence requirements, a health screening check, kava serving times and psychometric testing procedures), flexibility around kava serving portions, the consumption of *chasers*, and the freedom to move about was allowed so as to create a 'naturalised' setting of kava consumption.

Two short videos explaining the psychometric tests were shown to the participants with time given for questions and answers. Once all the participants were familiar with the test procedures, they were taken a few metres away to an adjacent computer lab where they completed the first psychometric test session to provide baseline data.

Following baseline testing, all of the participants were returned to the 'kava room' and invited to sit on woven mats where *isevusevu* was presented. This is a Fijian influenced cultural acknowledgement in which the participants were thanked for their time and participation. All Pacific peoples have cultural practices similar to *isevusevu,* including Māori who call their equivalent *pōwhiri* or *whakatau* (Aporosa, 2017). Additionally, this aided a 'naturalised' kava consumption setting, and complied with Pacific cultural expectations and obligations guided by Aporosa's (2014a) Post-Development Pasifika methodological framework. The control participants did not consume kava during the *isevusevu* or at any point during the testing phase. Over the following six hours, the participants engaged in *talanoa*, consuming *chasers* and drank

kava, as supplied relevant to their participation in the study, and occasionally left the room to use the toilet.

3.2 Psychometric tests

Two driver safety psychometric assessments were drawn from the Vienna Test System (VTS) test battery. VTS have been supporting traffic psychology research through the provision of industry standard assessment measures for more than 50 years. All VTS assessments have been validated with more than 1,350 assessments being used at test centres worldwide (VTS, 2016). These tests are designed for multiple-use on single participants without compromising test-retest validity. Additionally, the tests are ethno-friendly and designed so as not to marginalize participants in the study, who may have English as their second language.

Two specific VTS tests were chosen as these measured cognitive faculties critical to driver safety and could be completed within nine minutes; a time period that did not disrupt the kava serving time rate/pace. A total of seven test sessions were held per group (10 active and 10 control), over the six hour test period, delivered in a University of Waikato School of Psychology computer lab (adjacent to the 'kava room') on a suite of Dell Optiplex (9020) desktop computers. These tests were:

3.2.1 The WAFA Alertness (intrinsic visual) test

The WAFA Alertness (intrinsic visual) test takes three minutes to complete and requires the participant to press a 'green' button (on the computer keyboard) as quickly as possible every time a large black circle is presented on the white computer monitor screen. The WAFA test measured reaction and attention intensity (Sturm, 2011a). In total, 50 stimuli were presented of which 25 were randomly selected for "mean reaction time" assessment (Aschenbrenner et al., 2012, p.9,15).

3.2.2 The WAFG Divided Attention (uni-modal visual) test

The WAFG Divided Attention (uni-modal visual) test also took three minutes to complete, with participants required to monitor two visual stimuli (a black square and a circle

on a white computer screen). If one of the stimuli becomes lighter [in colour], twice in succession, the participant registers this change by pressing a 'green' button (on the computer keyboard) as quickly as possible (Sturm, 2011b). The lights in the laboratory were dimmed to aid participants in recognizing stimuli colour-change. The WAFG test specifically measures parallel processing and selectivity of attention (Sturm, 2011b). In total, 85 sets of stimuli were presented of which 21 were randomly selected for "Mean reaction time" assessment (Aschenbrenner et al., 2012, p.9,15).

3.3 Kava preparation, cultural compliance and serving

Dried powered kava root/basal stump, originating in Tonga, was purchased from a reputable and well-patronised retailer in Hamilton, New Zealand. A sufficient quantity of kava was purchased for all tests to aid standardization. Kava retained for later testing was compressed into heavy-duty plastic bags, and stored in an airtight container in a dry dark cupboard at approximately 18^{o}C to maintain freshness (AECOM-Kalang, 2017). Prior to each test session, 36 litres (9.51 gallons) of kava was mixed for consumption approximately one hour before the session. This used a standardized recipe with the kava concentration estimated to be similar to the average kava strengths in *kalapu*'s in New Zealand. Additionally, the 'recipe' enabled easy duplication.

A 200gm (7.05 ounce) sample of the dried kava powder, together with the recipe, was couriered to T.K Group Laboratories in Iowa, USA for kavalactone strength analysis. Kava residue from multiple samples that had been evaporated at 40ºC, were extracted for analysis. The kava was found to contain no adulterants, with a strength rating of 9.26% total kavalactones by dry weight, a chemotype of 423651, and a mean kavalactone content of 145mg per 100ml of kava beverage. Certificates of analysis are attached at Appendix A.

The kava was served from a *kumete* in *bilo*/*ipu* (cups made from coconut shells) using measured quantities (decline to 25mls (milliliters), 50mls, 57mls and 100mls) as directed by the active participants. A time chart ensured that the kava was served at the appointed times (see Figure 6).

3.4 Test conclusion

The final testing was undertaken following the sixth hour of kava consumption. The participants were acknowledged with *tatau*, similar to *isevusevu*, a Fijian influenced protocol thanking the participants for their time and participation. This also complies with Pacific cultural expectations, obligations, and the Post-Development methodological framework (Aporosa, 2014a). This provided an opportunity for some of the control participants to consume kava for the first time. The *tatau* was followed by a substantial meal, and presentation of a $100 gift voucher to thank each of the participants for their time. All were provided with a ride home. The test session took approximately 7.5 hours to complete.

3.5 Data analysis

VTS test results were presented in numerical format and were easily imported to a variety of software options allowing data analysis. Pre/post data analysis was conducted comparing person to person and between groups change. Statistical modeling was based on ANOVA, independent t-tests and Bayesian inference. Bayesian analysis produces a 'Bayes factor' which is used for comparative analysis. A Bayes factor is a ratio of the likelihood of a specified hypothesis (e.g. effect of a treatment) to a second hypothesis (e.g. no effect).

4 Results and discussion

4.1 Kava consumption volumes

Only eight of the 20 active participants consumed the maximum average of 3.6 litres of kava over the six hour test period, drinking the full 100mls of kava at each serving. At least half of the eight participants who consumed the maximum average – which included one of the female participants – complained that 100mls was less than they usually drank, and requested larger portions of kava. This was declined. In most cases the remaining 12 participants commenced the testing with smaller portions although by midway through the test session they increased their servings to consume the full 100mls at each serve. When questioned at the conclusion of testing about this, most reported feeling a little under pressure at the start, concerned that they may not be able to complete the testing. However, as the testing

progressed they felt more confident that they could finish the full test session and therefore changed to consume the standard average. Over the full six hours of testing, the active participants consumed on average 3.0585lts (litres, or 6.117 pints) each (SD = 0.713 litres), of kava drink which can be calculated as up to 4,426.145mgs of kavalactones. This is approximately 15 times greater than pharmacologically recommended dose.

4.2 Psychometric tests

The mean reaction time and divided attention at baseline was 249.95msec (SD=37.57) and 583.58msec (SD=226k .62) respectively.

4.2.1 The WAFA Alertness (intrinsic visual) test

Conflicting with the hypothesis, the data analysis of the WAFA Alertness test results indicated no statistically significant (p<0.05) difference in reaction time and attention intensity [F=(13,264), 0.582, p=0.868], both within person and between groups at any measurement point over the six-hour testing period (Figure's 7). Figure 7A shows the mean reaction and attention intensity time for the control participant groups, at hourly test intervals. After six hours, the mean reaction and attention intensity time was 256.70msec (SD=36.86); this being 7msec (milliseconds) slower than baseline. Similarly, Figure 7B shows the mean reaction and attention intensity time for the kava consuming active participant groups at hourly test intervals. After six hours, the mean reaction and attention intensity time was 271.8msec (SD=46.32); this being 22.10msec slower than baseline. The insignificance of this difference is demonstrated when compared to the effects of alcohol consumption on driver reaction time. Grant et al. (2000), showed that consuming 50μg (milligrams) of alcohol (equivalent to the current 0.05 NZ driver blood alcohol limit), slowed driver reaction time by 70msec, and this increased to 120msec at 0.08 (the previous limit). Finally, Figure 7C shows the change in an individual's mean reaction time from baseline correlated with volume of kava consumed. Positive values indicate slower reaction time and negative values faster reaction time.

Bayesian inference analysis corroborated the ANOVA and independent t-tests analysis results. This showed that at baseline the p-value (probability value) for all participants was 0.280 and at the end of testing at hour six when the active and control participants were compared, 0.585. Bayesian comparative analysis between baseline and final test at the sixth hour for the control participants was p=0.627 and for the active participants p=0.983. Bayesian comparative analysis between the control participants and the active participants at the sixth hour of testing yielded a Bayes factor of 0.472. This Bayes factor suggests no meaningful difference between the active and control.

4.2.2 The WAFG Divided Attention (uni-modal visual) test

Again conflicting with the hypothesis, data analysis of the WAFG Divided Attention results indicated no statistically significant (p<0.05) difference in parallel processing and selectivity of attention [F=(13,264), 0.834, p=0.624], either within person or between groups at any measurement point over the six-hour testing period (see Figure's 8). Figure 8A shows the mean divided attention time for the control participant groups, at hourly test intervals. After six hours, the mean divided attention time was 499.75msec (SD=167.62), which is a small decrease (ie. improvement) of 83.83msec when compared with baseline. Similarly, Figure 8B shows the mean divided attention time for the kava consuming active participant groups at hourly test intervals. After six hours, the mean divided attention time for the active participant groups was 568.32msec (SD=217.71), a, average decrease (ie. improvement), although only slightly by 15.26mls. Figure 8C shows the change in an individual's mean reaction time from baseline correlated with volume of kava consumed. The positive values indicate slower reaction time and negative values faster.

Bayesian inference analysis corroborated the ANOVA and independent t-tests analysis results. This showed that at baseline the p-value for all participants was 0.385 and at the end of testing at hour six when the active and control participants were compared, 0.607. Bayesian comparative analysis between baseline and final test at the sixth hour for the control

participants was p=0.965 and for the active participants p=0.783. Bayesian comparative analysis between the control participants and the active participants at the sixth hour of testing yielded

Figure 7: WAFA Alertness (intrinsic visual) test results

Figure 7A: **Control** participant hourly reaction time

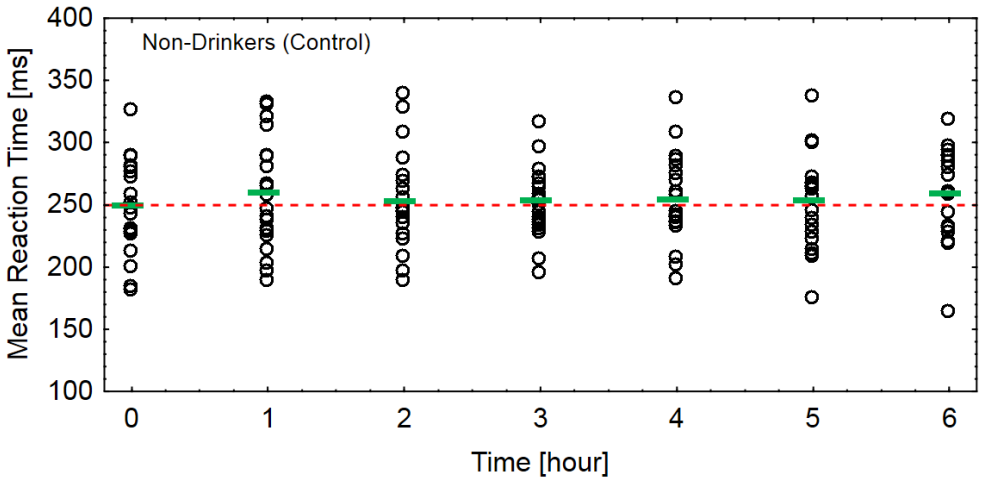


Figure 7B: **Active** participant hourly reaction time

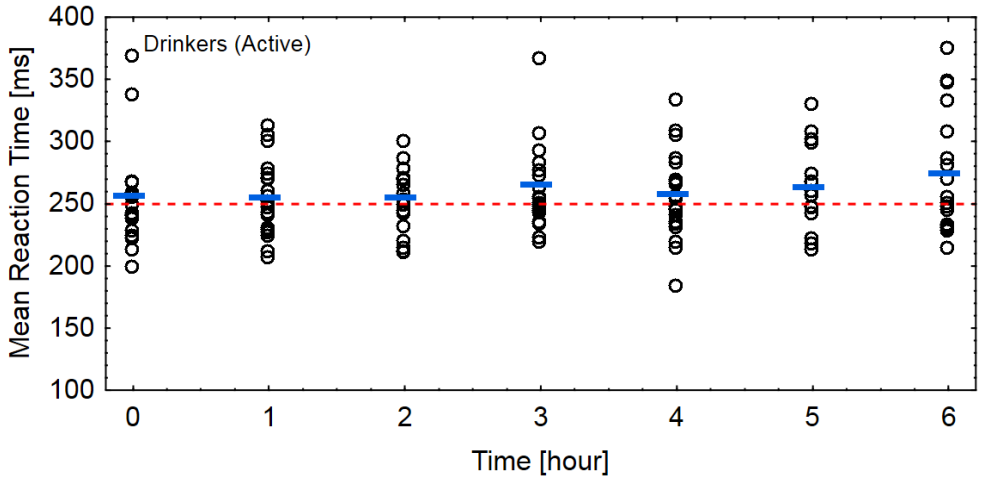


Figure 7C: Change in individual mean reaction time from baseline with kava consumption

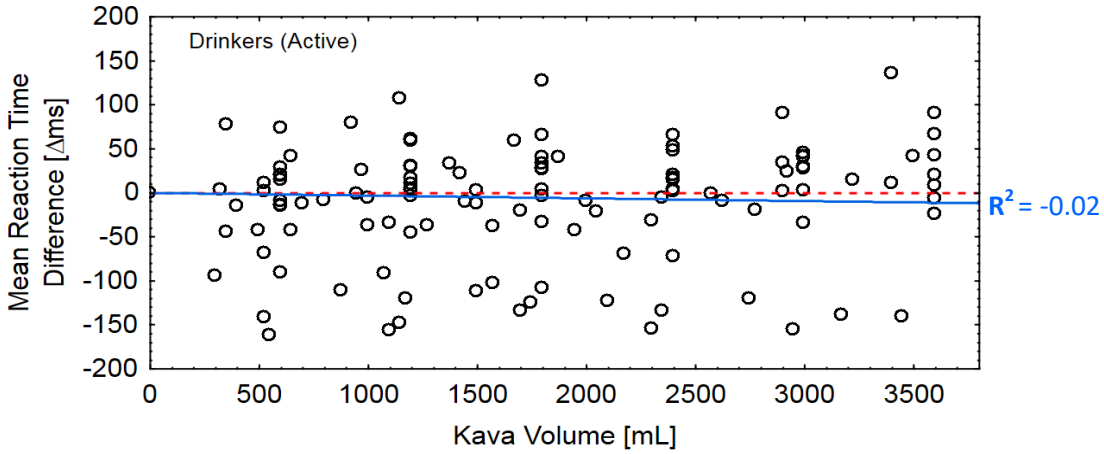


Figure 8: The WAFG Divided Attention (uni-modal visual) test results

Figure 8A: **Control** participant hourly divided attention time

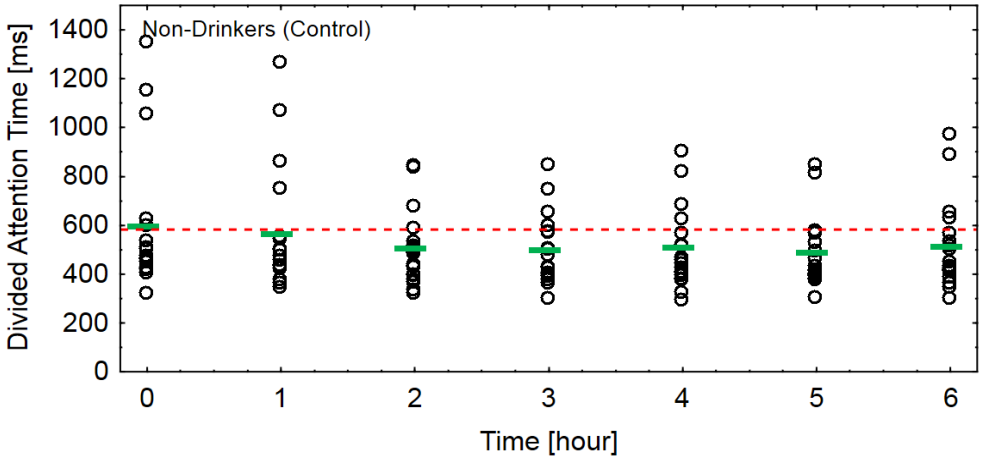


Figure 8B: **Active** participant hourly divided attention time

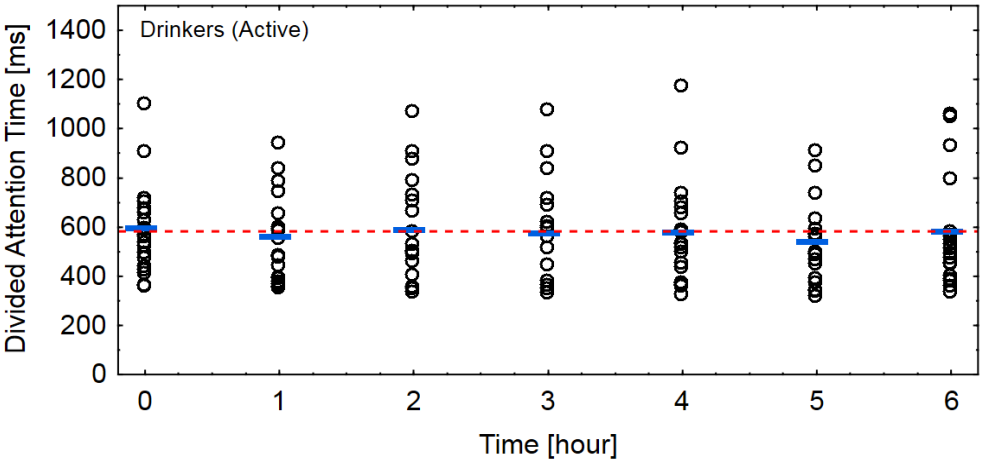
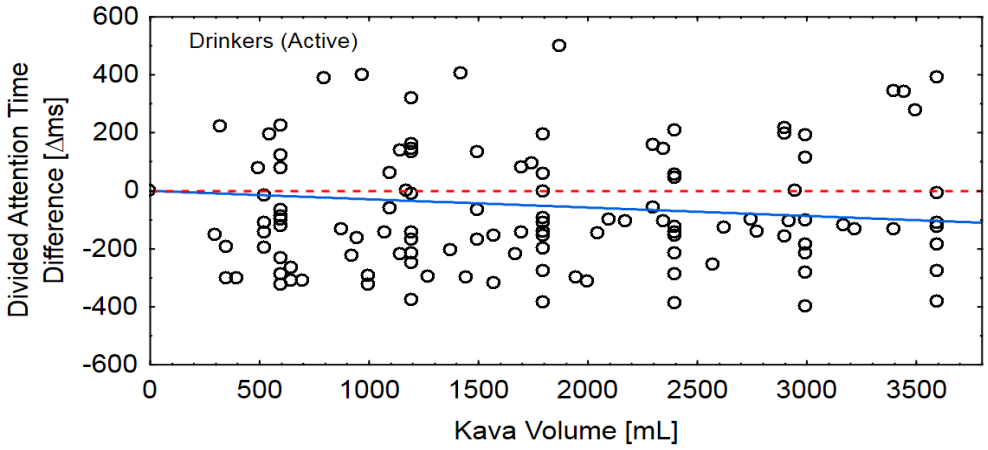


Figure 8C: Change in individual mean divided attention time from baseline with kava consumption

R^2 = -0.03



4.3

a Bayes factor of 0.492. Again, this Bayes factor suggests no meaningful difference between the active and control and show no correlation between consuming kava at traditional volumes and response latency or impairment on divided attention tasks.

4.3 Observations

At the conclusion of each test event, the chief investigator and research assistants discussed observations they had made regarding participant changes over the duration of the test period. The research team unanimously agreed that as the six hours progressed, subtle changes were observed in many of the kava drinkers, namely slowing psychomotor response, a somnolent-like state, altered word pronunciation and a slowing of speech rate. These observations align with reports from both NZ and Pacific based Police, who state that kava drink-drivers observed during road-side stops, typically exhibit decelerated body movement and slurred or slowed speech (Morgan, 2014/2017; Welsh, 2017; Galuvoa, 2018; Kalura, 2018; Mishra, 2018).

4.4 Explanations

Anomalies between police observations of kava drink-drivers at road stops and those of the research participants as observed during this study, led to lengthy discussions with experts such as; Professor Jerome Sarris, a specialist in psychopharmacology and cognitive function from the University of Melbourne who led the kava GAD studies; Professor David Nutt, a

neuropsychopharmacologist specialising in drug-brain interaction at the Imperial College London; Associate Professor Brett Langley, a neuroscientist from the Harvard Medical School (now at the University of Waikato); and, Professor Nicola Starkey, a specialist in cognition, driver fitness and psychological assessment. These conversations were to consider possible explanations for the effects of kava including an analysis of the study structure, approach and methodology.

One aspect that was discussed was the possibility of participant interruption/disruption over the six-hour testing period. During a typical kava session, drinkers sit for lengthy periods and engage *talanoa*. Discussions raised the possibility that excessive cognitive assessment may have influenced test results, as participants were required to leave the kava consumption environment each hour and move to the adjacent computer lab for testing. It was suggested that in future, fewer tests would still provide adequate data points while having a lesser impact on the kava environment and participants.

The effects of kava reverse tolerance was also discussed with the experts. Unlike many substances, kava users do not build tolerance (Singh, 2004a). Rather, users experience 'reverse tolerance', in which repeated use leads to greater effects. Although reverse tolerance has been suggested as a potential reason why kava does not cause physical addiction (Mindel, 1998; Steiner, 2001), the reality is that little is known about kava and this phenomenon. It was agreed this unknown was beyond the current study.

The team also discussed studies that had investigated the effects of Benzodiazepine on driving. Of interest to those deliberations was the work of Saletu and colleagues (1989). They discovered that although there are similarities in anxiolytic action between Clobazam (a type of benzodiazepine) and kava, EEG-brain mapping of participants taking kava reported a decrease in beta activity, whereas Clobazam greatly increased beta activity. Increased beta activity is associated with attention, alertness, problem solving, decision-making and judgment. These results indicated active differences beyond anxiolytic action between kava and benzodiazepine

medication. Moreover, that difference potentially provides an explanation for the lack of impact by kava on attention and divided attention in the present study; an effect which stands in opposition to the actions of benzodiazepine on these same cognitive faculties. Additionally, Verster et al. (2004) state that although benzodiazepine medication reduces anxiety, there is variability in effect characteristics, drug half-life and dosages between benzodiazepine varieties, with resulting differences in cognitive impact on driving ability.

In addition to Saletu et al. and Verster et al's. work, the literature explaining kava's action on $GABA_A$, particularly the role kava plays in decreasing neurotransmitter function in the central nervous system (CNS), was considered (Ligresti et al., 2012; Lim, 2016). It was suggested that potentially the psychometric tests used in the current study lacked utility in kava cognition assessment, as they relied on visual-sensory as opposed to the CNS to measure impairment. It was further suggested that an alternative approach was required, one that could measure kava impairment via the CNS. Moreover, the ability to measure subtle/slight change would be critical in light of Saletu and colleagues findings regarding kava's action in increasing beta activity.

A new testing system has subsequently been identified. This is a recently developed highly sensitive neuroscientific assessment tool (name deliberately withheld due to ongoing research), which measures slight changes in strategic, tactical and operational cognitive aspects including fine-motor-skills and fatigue. This assessment measure was recently pilot tested during a kava session of six hours (in which two participants consumed 3.6 litres of kava each and were tested at baseline, at the mid-point and again at conclusion). The pilot study results demonstrated the viability of undertaking a full-length study, which is planned for 2019.

The findings from the present study were discussed with staff at the Counties-Manukau and Otahuhu Police Prosecutions Section, Auckland (Morgan, 2017; Fletcher, 2017) and Road Policing at NZ Police National Headquarters (Welsh, 2017), as well as in the Pacific region – Vanuatu, Fiji, Tongan and Samoa (Galuvoa, 2018; Grewe, 2018; Kalura, 2018; Mishra, 2018). They reported ongoing concerns by some patrol officers regarding drivers who appeared

intoxicated although showing no evidence of this in breath screening tests. Some of these drivers were believed to have recently consumed kava, however due to limited knowledge regarding kava's effects on driving, together with no current measurement of impact, officers were often prevented from taking decisive action unless other offences had occurred. The findings were also discussed with ESR scientists responsible for testing the presence of drugs in blood samples (21 April 2017, Polson & McCarthy). They reported an increased detection of kava in the blood of motor vehicle accident victims – both survivors and deceased. However, they acknowledged their lack of understanding and applicability regarding these findings.

5 Conclusions and recommendations

The use of kava by Pacific peoples as part of cultural practice and *talanoa*, and by non-Pacific people as a social lubricant and alcohol alternative, is increasing. It is common for kava drinkers to consume up to 3.6 litres of kava over prolonged periods of up to six hours (Aporosa, 2014b). The effects of kava are described as slightly euphoric although non-hallucinogenic, with relaxant anti-anxiety effects similar to Benzodiazepine (Sarris et al., 2013). High consumption of kava is observed to produce slower motor response and slurred speech (Morgan, 2014/2017; Welsh, 2017). While there are potential benefits from kava drinking and factors making kava a better choice than other forms of recreational drugs such as alcohol, (Aporosa & Tomlinson, 2014), the effects of kava drink on reaction time and divided attention presents some important questions that need to be addressed.

This report on an experimental study with a group of practiced kava drinkers, as well as non-kava drinkers, while showing some effects, did not reveal a significant reading on reaction time and divided attention. Experts suggested frequent interruptions to test participants was a possible reason for a lack of test results, implying fewer tests may have produced an alternative result. Other factors may have led to the inconclusive results, including 'reverse tolerance' (Singh, 2004a) and the use of visual-sensory tests. As Bwarenaba et al. (2017) have pointed out, kavalactone "modes of action are not fully understood" (p.1) with even less known about "the

neurophysiological mechanisms associated with kavalactone metabolism" (p.5), hence this calls for a re-think in how to measure kava consumption and its effects, especially in the case of kava drinking and driving.

Although the experimental study did not reveal significant data, the anecdotal evidence from police, who continue to apprehend drivers who appear intoxicated while showing negative results from breath tests, shows that more work in this field is urgently required. With new testing looking in more detail at the central nervous system, to measure impairment as opposed to visual response, this may reveal more credible and reliable data. A neuroscientific assessment tool, which measure kava affect via the CNS, has shown promise in preliminary tests and has been endorsed by a panel of experts as a viable means to continue this important research.

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Finally, this study would not have been possible without those who joined the study as research participants. Due to confidentiality, they cannot be named.

7 References

Abbott, P. (2016). *Kava: A review of the safety of traditional and recreational beverage consumption (Technical Report)*. Rome: Food and Agriculture Organization of the United Nations and World Health Organization.

AECOM-Kalang. (2017). *Fiji kava quality manual*: Pacific Horticultural and Agricultural Market Access (PHAMA) program, Pacific Community's Pacific Agriculture Policy Project (SPC PAPP) and the University of the South Pacific (USP).

Aporosa, S. (2008). *Yaqona and education in Fiji: A clash of cultures?* Germany: VDM Verlag.

Aporosa, S. (2011). Is kava alcohol?: The myths and the facts. *Journal of Community Health and Clinical Medicine for the Pacific*, *17*(1), 157-164.

Aporosa, S. (2014a). *Yaqona (kava) and education in Fiji: Investigating 'cultural complexities' from a post-development perspective*. Albany: Massey University, Directorate Pasifika@Massey.

Aporosa, S. (2014b). Yaqona (kava) as a symbol of cultural identity. *Locale: The Australasian-Pacific Journal of Regional Food Studies, 4*, 79-101.

Aporosa, S. (2015). The new kava user: Diasporic identity formation in reverse. *New Zealand Sociology, 30*(4), 58-77.

Aporosa, S. (2016). Kava: Weighing the negatives against the positives. Paper presented at the *Pasifika Medical Association 20th Anniversary Conference; Plenary panel: Kava - killer or cure?* Aug. 24, Auckland. Retrieved from https://docs.wixstatic.com/ugd/bd81a7_4556c71320b94e 18a40da10e47949a10.pdf.

Aporosa, S. (2017). *Kava and Māori: New drug fashion or re-engagement with 'kawa'?* Te Kōtahi Institute, University of Waikato (in post).

Aporosa, S. (2018). Personal observation (based on more than 20 years of kava use in Fiji, New Zealand and internationally with Pacific and non-Pacific kava use groups).

Aporosa, S., & Tomlinson, M. (2014). Kava hangover and gold-standard science. *Anthropologica (Journal of the Canadian Anthropology Society), 56*(1), 163-175.

Aschenbrenner, S., Kaiser, S., Pfüller, D., Roesch-Ely, D., & Weisbrod, M. (2012). *Basic cognitive testing: COGBAT manual*. Austria: Schuhfried.

Bwarenaba, B. K., Juliana, P., Kellie, S., Mengarelli, M. S., & Eric, A. N. (2017). A behavioral survey of the effects of kavalactones on caenorhabditis elegans neuromuscular transmission. *Journal of Experimental Neuroscience, 11*, 1-7.

Crowley, T. (1994). Proto who drank kava? In A. Pawley & M. Ross (Eds.), *Austronesian terminologies: Continuity and change (Pacific linguistics. Series C-127)* (pp. 87-100). Canberra: Dept. of Linguistics, Research School of Pacific and Asian Studies, The Australian National University.

DCNZ: District Court of New Zealand. (2000). Police v Tupou, December 7. *District Court Register, 2001*, 503-512.

Dragull, K., Lin, G. D., & Tang, C. S. (2006). Chemistry, pharmacology, and safe aspects of kava. In E. Johnston & H. Rogers (Eds.), *Hawaiian 'awa: Views of an ethnobotanical treasure* (pp. 19-29). Hilo, HI: Association for Hawaiian 'Awa.

Fletcher, S. (2017). Personal communication with Regulation 24 (Police) Officer, Manurewa Police Prosecutions Office, Auckland, May 4.

Foo, H., & Lemon, J. (1997). Acute effects of kava, alone or in combination with alcohol, on subjective measures of impairment and intoxication and on cognitive performance. *Drug and Alcohol Review, 16*, 147–155.

Galuvao, L.V. (2018). Personal communication: meeting with Executive Office, Land Transport Samoa, Oct. 2.

Grant, S. A., Millar, K., & Kenny, G. N. C. (2000). Blood alcohol concentration and psychomotor effects. *British Journal of Anaesthesia, 85*(3), 401-406.

Graziano, S., Orsolini, L., Rotolo, M. C., Tittarelli, R., Schifano, F., & Pichini, S. (2017). Herbal highs: Review on psychoactive effects and neuropharmacology. *Current Neuropharmacology, 15*(5), 750-761.

Grewe, S. (2018). Personal communication: meeting with Deputy Commissioner of Police, Tonga and five senior members of Police, Oct. 11.

Heinze, H. J., Munthe, T. F., Steitz, J., & Matzke, M. (1994). Pharmacopsychological effects of oxazepam and kava special-extract in a visual search paradigm assessed with event-related potentials. *Pharmacopsychiatry, 27*, 224-230.

Henry, T. (2018). *Club kava underground*. (digital image used with permission). June.

Jolly, D. N. (2009). *DUI / DWI: The history of driving under the influence*. Colorado: Outskirts Press.

Kalura, E. (2018). Personal communication: meeting with Sergeant of Police, Road Safety Division, Vanuatu Central Police Station, Nov. 8.

Kirch, P. V., & Green, R. C. (2001). *Hawaiki: Ancestral Polynesia*. Cambridge: Cambridge University Press.

LaPorte, E., Sarris, J., Stough, C., & Scholey, A. (2011). Neurocognitive effects of kava (Piper methysticum): A systematic review. *Human Psychopharmacology: Clinical and Experimental (Hum Psychopharmacol Clin Exp), 26*(2), 102–111.

Lebot, V., & Cabalion, P. (1988). *Kavas of Vanuatu: Cultivars of Piper methysticum Frost* (Vol. Technical Paper No.195). Noumea: South Pacific Commission.

Lebot, V., & Levesque, J. (1996). Genetic control of kavalactone chemotypes in Piper methysticum cultivars. *Phytochemistry, 43*(2), 397-403.

Lebot, V., Merlin, M., & Lindstrom, L. (1992). *Kava: The Pacific drug. (Psychoactive Plants of the World Series).* New Haven: Yale University Press.

Lim, T. K. (2016). Edible medicinal and non-medicinal plants: Volume 11, modified stems, roots, bulbs. New York: Springer.

Ligresti, A., Villano, R., Allarà, M., Ujváry, I., & Di Marzo, V. (2012). Kavalactones and the endocannabinoid system: The plant-derived yangonin is a novel CB1 receptor ligand. *Pharmacological Research, 66*(2), 163-169.

Maneze, D., Speizer, A., Dalton, N., & Dennis, S. (2008). A descriptive study of kava use among Tongan men in Macarthur, Sydney South West. *Australian & New Zealand Journal of Public Health, 32*(4), 314-316.

Mathews, J. D., Riley, M. D., Fejo, L., Munoz, E., Milns, N. R., Gardner, I. D., . . . Gununywawuy, J. (1988). Effects of the heavy usage of kava on physical health: Summary of a pilot survey in an Aboriginal community. *Medical Journal of Australia, 148*, 545-555.

McCready, W. C. (1996). Applying sampling proceedures. In F. T. L. Leong & A. Austin (Eds.), *The psychology research handbook* (pp. 98-112). London: SAGE.

ME: Medical Editor. (2017). Kava legal in relaxation of WA's drug laws. The West Australian (online), Feb. 14. Retrieved from https://thewest.com.au/news/health/kava-legal-in-relaxation-of-was-drug-laws-ng-b88384684z.

MediHerb. (1994). Kava - A safe herbal treatment for anxiety. *MediHerb Professional Newsletter, No.39 May (part 2)*.

Mills, S., & Bone, K. (2005). *The essential guide to herbal safety*. Missouri: Elsevier.

Mindell, E. (1998). *All about kava*. New York: Avery.

Mishra, M. (2018). Personal communication: phone call with Senior Superintendent, Police Traffic Division, Fiji, Sept. 25.

Morgan, M. (2014/2017). Personal communication with Police Sergeant, Counties-Manaukau Prosecutions Section, Nov. 4/May 4.

MoT: Ministry of Transport. (2017). *Drugs and alcohol 2017*. Wellington: NZ Government. Retrieved from www.transport.govt.nz/assets/Uploads/Research/Documents/Alcohol-drugs-2017.pdf

Münte, T. F., Heinze, H. J., Matzke, M., & Steitz, J. (1993). Effects of oxazepam and an extract of kava roots (Piper methysticum) on event-related potentials in a word-recognition task. *Pharmacoelctroencephalography, 27*, 46–53.

Payson, R. (2008). *The culture of kava: A visual journey of rituals and ceremonies among rural Fijians.* (Master of Social Science), Brooks Institute of Photography.

Poulsen, H., Moar, R., & Troncoso, C. (2012). The incidence of alcohol and other drugs in drivers killed in New Zealand road crashes 2004-2009. *Forensic Science International*, *223*(1-3), 364-370.

Prescott, J., Jamieson, D., Emdur, N., & Duffield, P. (1993). Acute effects of kava on measures of cognitive performance, physiological function and mood. *Drug and Alcohol Review, 12*, 49-58.

Procyshyn, R. M., Bezchlibnyk-Butler, K. Z., & Jeffries, J. J. (2017). *Clinical handbook of psychotropic drugs* (Vol. 22nd revised edition). Toronto: Hogrefe Publishing.

Russell, P. N., Bakker, D., & Singh, N. N. (1987). The effects of kava on alerting and speed of access of information from long-term memory. *Bulletin of the Psychonolis Society, 25*(4), 236-237.

Saletu, B., Grünberger, J., Linzmayer, L., & Anderer, D. P. (1989). EEG-brain mapping, psychometric and psychophysiological studies on central effects of kavain-a kava plant extract derivative. *Human psychopharmacology, 4*, 169-190.

Sarris, J., Stough, C., Bousman, C. A., Wahid, Z. T., Murray, G., Teschke, R., Schweitzer, I. (2013). Kava in the treatment of generalized anxiety disorder: A double-blind, randomized, placebo-controlled study. *Journal of Clinical Psychopharmacology, 33*(5), 643-8.

Schelosky, L., Raffauf, C., Jendroska, K., & Poewe, W. (1995). Kava and dopamine antagonism. *Journal of Neurology, Neurosurgery & Psychiatry, 58*, 639-640.

Schmidt, M. (2014). German Court ruling reverses kava ban; German Regulatory Authority appeals decision. *HerbalEGram, 11*(7). Retrieved from http://cms.herbalgram.org/heg/volume11/07July/GermanKavaBanReversal.html?ts=1443080085&signature=4a90e6b17036bdef1912cdf1d6eb19c3

Schmidt, M., Morgan, M., Bone, K., & McMillan, J. (2005). Kava: A risk-benefit assessment. In S. Mills & K. Bone (Eds.), *The essential guide to herbal safety* (pp. 155-221). Missouri: Elsevier, Churchill, Livingstone.

Singh, Y. N. (2004a). An introduction to kava Piper methysticum. In Y. N. Singh (Ed.), *Kava: From Ethnology to Pharmacology (Medicinal and Aromatic Plants - Industrial Profiles Volume 37)* (pp. 1-9). Boca Raton: CRC Press.

Singh, Y. N. (2004b). Botany and ethnobotany of kava. In Y. N. Singh (Ed.), *Kava: From Ethnology to Pharmacology (Medicinal and Aromatic Plants - Industrial Profiles Volume 37)* (pp. 50-75). Boca Raton: CRC Press.

Singh, Y. N. (2004c). Pharmacology and toxicology of kava and kavalactones. In Y. N. Singh (Ed.), *Kava: From Ethnology to Pharmacology (Medicinal and Aromatic Plants - Industrial Profiles Volume 37)* (pp. 104-139). Boca Raton: CRC Press.

Slack, A., Nana, G., Webster, M., Stokes, F., & Wu, J. (2009). *Costs of harmful alcohol and other drug use: Report for the NZ Ministry of Health and ACC*. Wellington: Business and Economic Research Ltd.

Showman, A. F., Baker, J. D., Linares, C., Naeole, C. K., Borris, R., Johnston, E., . . . Turner, H. (2015). Contemporary Pacific and Western perspectives on `awa (Piper methysticum) toxicology. *Fitoterapia, 100*, 56–67.

Spillane, P. K., Fisher, D. A., & Currie, B. J. (1997). Neurological manifestations of kava intoxication. *Medical Journal of Australia, 167*(3), 172-173.

Starkey, N., Charlton, S. G., Malthotra, N., & Ameratunga, S. (2016). Prevalence of psychotropic drug use prior to driving. *Journal of Transport & Health*. *4*, 108-117.

Steiner, G. G. (2001). Kava as an anti craving agent: Preliminary data. *Pacific Health Dialog, 8*(2), 335-339.

Sturm, W. (2011a). *Perception and attention function: Alertness (Test label WAFA)*. Austria: Schuhfrid GmbH.

Sturm, W. (2011b). *Perception and attention function: Divided attention (Test label WAFG)*. Austria: Schuhfrid GmbH.

Swenson, J. N. (1996). Man convicted of driving under the influence of kava. *Deseret Morning News: UT,* p. B3.

Teschke, R. (2010). Kava hepatotoxicity: A clinical review. *Annals of Hepatotoxicity, 9*(3), 251-265.

Thomson, P. (1999). *Kava in the blood: A personal and political memoir from the heart of Fiji*. Auckland: Publishing Press.

Verster, J., Veldhuijzen, D. S., & Volkerts, E. R. (2004). Residual effects of sleep medication on driving ability. *Sleep Medicine Reviews, 8*(4), 309-325.

VTS: Vienna Test Systems. (2016). Traffic – fitness to drive. Online. https://www.schuhfried. com/ tests/traffic/

Wainiqolo, I., Kool, B., Nosa, V., & Ameratunga, S. (2015). Is driving under the influence of kava associated with motor vehicle crashes? A systematic review of the epidemiological literature. *Australian and New Zealand Journal of Public Health, 39*(5), 495-499.

Welsh, G. (2017). Personal communication: email, from the Inspector in Charge: Road Safety, New Zealand Police National Headquarters, 26 June.

Wolinski, C. (2018). This ancient South Pacific sipper is officially trending. VinePair (online), March 21. Retrieved from https://vinepair.com/articles/what-kava-kava-drink/.

Yardley, C. (Photgrapher). (2016, April 2). *Kava is prepared in a traditional kava bowl* [digital image]. Retrieved from https://www.stuff.co.nz/national/health/78418804/Waikato-study-examines-kavas-effects-on-driving

Appendix A: Certificate of Analysis

T.K. Group Labs
Ankeny, IA 50023

TrueKava.com
gs@truekava.com

Certificate of Analysis

Date: October 24, 2016
Client: Apo Aporosa
Description: Dehyrated residue of Piper methysticum root powder drink
Lab sample: B0296

Sample Preparation

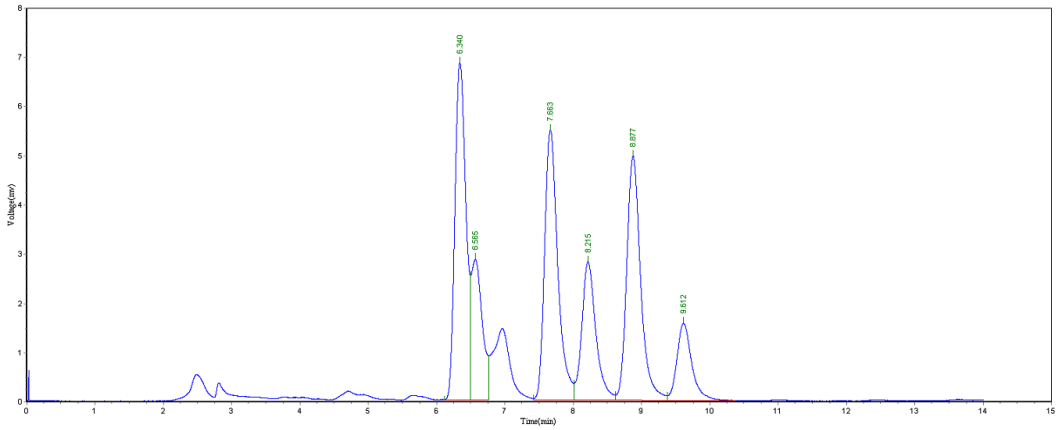
A traditional cold water extracted kava drink was prepared with 100g Piper methysticum root powder and 2L water using extraction method and filtration as prescribed by Client. Multiple 100ml samples of this preparation were evaporated at 40°C and the resultant residue was tested by the method described in this Certificate.

Chemical Analysis

Identity: Piper methysticum
Purity: No adulteration detected
Strength: 9.26% total kavalactones by dry weight
Chemotype: 423651
Mean residue per 100ml sample: 1.573g
Mean KL% of residue: 9.24%
Mean kavalactone content of prepared drink: 1.45mg/ml

Data

code	description		chemotype
B0296	Apo D1		423651
qual:	pass		
K/DHM:	1.40	rel. %	total %
1	Desmethoxyyangonin	6.52	0.60
2	Dihydrokawain	20.73	1.92
3	Yangonin	18.90	1.75
4	Kawain	22.38	2.07
5	Dihydromethysticin	16.01	1.48
6	Methysticin	15.47	1.43
		100.00	9.26



	code	desc		std K	K/DHM		chemotype			
	B0296	Apo D1		47415	1.40		423651			
peak	KL	KL	RT	height	area	factor	corrected	rel. %	KL	total %
1	M	Methysticin	6.34	6840.103	77155	0.66	50923	15.47	6	1.43
2	DHM	Dihydromethysticin	6.565	2851.213	31944	1.65	52708	16.01	5	1.48
3	K	Kawain	7.663	5485.617	73670	1.00	73670	22.38	4	2.07
4	DHK	Dihydrokawain	8.215	2811.333	40144	1.70	68245	20.73	2	1.92
5	Y	Yangonin	8.877	4961.59	70697	0.88	62213	18.90	3	1.75
6	DMY	Desmethoxyyangonin	9.612	1571.208	23838	0.90	21455	6.52	1	0.60
							329213	100.00		9.26