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# The impact of traditional *kava* (*Piper methysticum*) use on cognition: Implications for driver fitness

S. 'Apo' Aporosa<sup>a,\*</sup>, Hakau Ballard<sup>b</sup>, Rishi Pandey<sup>c</sup>, Mary Jane McCarthy<sup>c</sup>

<sup>a</sup> Te Huataki Waiora School of Health and Te Kura Whatu Oho Mauri School of Psychology, University of Waikato, Aotearoa New Zealand

<sup>b</sup> School of Computing & Mathematical Sciences, University of Waikato, Aotearoa New Zealand

<sup>c</sup> Forensic Specialised Analytical Services, Institute of Environmental Science and Research Limited (ESR), Aotearoa New Zealand

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#### ABSTRACT

*Ethnopharmacological relevance*: Few studies have examined the impact of *kava* (*Piper methysticum G. Forst. f.*) on cognition when consumed at traditionally influenced volumes; most have used modified tablet-form *kava*, with the results erroneously overlaid on naturalistic *kava* consumption. *Kava* is a culturally significant Pacific drink with similar effects to Benzodiazepine. Traditionally influenced *kava* use sessions last, on average, 6 h in which attendees consume 3.6 L (7.6 pints) each of beverage *kava*, with some then driving home.

Aim of the study: This study evaluated the impact of traditionally influenced kava consumption on participants' neurological functioning. Testing occurred before, throughout and immediately following a typical *faikava* (kava-drinking) session, with the data then used to assess kava's potential impacts on driver functionality and safety. *Methods: Kava* using participants (n = 20) were assessed with the Brain Gauge following and during a traditionally influenced kava session and compared against control (n = 19). Brain Gauge measures slight changes to six cognitive faculties: Speed, Accuracy, Temporal Order Judgement (TOJ), Timing Perception, Plasticity, and Focus.

*Results and conclusions:* Comparisons of the within-cohort data showed a positive change in the Focus for the active group at the final testing point following 6-h of *kava* consumption. Between-cohort data showed a significant level of regression in the active participants' TOJ at the final testing point. No statistically significant level of impairment for the other five cognitive domains was detected. Although the results suggest that *kava* when consumed at traditional levels may have a slight positive effect on Focus, this result needs to be treated with caution, given the significant level of impairment noted at the final testing point for participants' TOJ. Temporal Order Judgement is associated with executive function, including decision making, behavioral control and information processing, all crucial aspects of driver safety. This is a new finding and suggests *kava* effects following traditional use differ from those caused by other substances, and may impair driver safety, although again, in a different way to other commonly consumed recreational substances. The findings also add quantitative understanding to ethnographic data on *kava* effects, suggesting the often-used term '*kava* intoxication' is misleading and incorrect.

# 1. Introduction

The study described in this article aimed to measure the impact that traditionally influenced *kava* consumption had on aspects of participants' neurological functioning. Testing was conducted during and immediately following a typical *faikava* (*kava*-drinking) session, as well as before the session to establish a baseline. The results were then applied to driver functionality.

The study was based at the University of Waikato's Te Huataki Waiora School of Health, with input from the School of Psychology's *Traffic and Road Safety Research Group*. Its purpose was to improve understanding of *kava*-related cognition, driver safety and coordination issues, following mounting national and international recognition of the need for more research and understanding of *kava* psychopharmacology. This recognition itself derives from increasing use of *kava* in diasporic Pacific and other non-traditional communities and settings around the world.

\* Corresponding author. E-mail address: apo.aporosa@waikato.ac.nz (S.'. Aporosa).

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Abbrevia	ations
ANZ	Aotearoa New Zealand
ESR	New Zealand Institute of Environmental Science and
	Research
HPLC	high-performance liquid chromatography
HRC	Health Research Council of New Zealand
(PPdMF)	Pacific Post-development Methodological Framework
T1	test 1, the collection of baseline data
T2	test 2, the mid-point collection of test data
T3	test 3, the final test data collection
TOJ	Temporal Order Judgement

The research intention was to use the results to improve the health of Pacific people, both in Aotearoa New Zealand (ANZ) and internationally, as well as that of other road users, by fostering safe *kava*-related driving practices, and thereby reducing injury and hospitalisation rates. If successful, such measures could also be expected to reduce the economic and social costs associated with road trauma, and would assist the efficacy of road policing, particularly from an evidentiary perspective.

The study was informed by two earlier investigations also aimed at understanding driver safety following naturalistic (or traditionally influenced) *kava* use (Aporosa, 2017, 2018; Aporosa et al., 2020, 2021).

Testing involved the use of a somato-sensory tool – the Brain Gauge – to assess the cognitive function of 20 active *kava* drinkers compared with 19 control participants (non-*kava* drinkers). Testing occurred during *faikava*; a typical *kava*-drinking session held in a naturalistic setting underpinned by Pacific respect-based values, with the aim of understanding how *kava* consumption might affect driving safety.

The study hypothesised that the participants consuming *kava* in the active group would show changes in their neurological functioning (namely speed, focus, fatigue, accuracy, sequencing, timing perception, plasticity and connectivity), when compared with the participants not consuming *kava* in the control group.

In the event, this hypothesis was partially confirmed, but not to the extent or in the cognitive areas initially anticipated. Apparent anomalies in the data were unable to be explained by psychopharmacologists. This is likely to be due, in part, to the fact that research into the relationship between traditionally influenced *kava* consumption and cognition is a new area, with an attendant lack of data and understanding from which to draw conclusions.

Despite and because of this, the study makes an important contribution in this area; adding significantly to what is known about *kava*'s impacts on cognition, when consumed at traditional use volumes. It also reinforces the already apparent knowledge gaps around *kava* psychopharmacology, strengthening the case for more research in this area.

#### 1.1. Kava, kavalactones and their effects on the central nervous system

*Kava* (*Piper methysticum*) is a shrub grown widely across the Pacific. The term '*kava*' is also used to refer to the drink made from the *kava* plant's roots and basal stump. The plant and the drink are both integral to Pacific traditions and cultural practices (Aporosa, 2014, 2019b).

*Kava* does not contain alcohol. Drinking *kava* makes users feel relaxed, rather than euphoric, and fosters clear-headed discussion (Aporosa, 2019a; Carlini, 2003). *Kava* is generally reported to be safe, non-hallucinogenic and non-addictive, and does not trigger any major health concerns (Aporosa, 2019a; Bian et al., 2020).

These qualities have led to *kava* being considered as a useful alternative to benzodiazapam when it comes to treating generalised anxiety disorder (Sarris et al., 2013b).

Kava's safe status is also reflected in how it is regulated in ANZ, where the government classifies it as a 'food' (New Zealand

Government, 2015); in the World Health Organization's assessment of *kava* beverage as 'low risk" (Abbott, 2016, p. 26); and in the Australian drug harm study where *kava* was ranked as the least-most dangerious drug substance (out of a total of 22 substances) at three (3) harm points (Bonomo et al., 2019). Interestingly, the study ranked alcohol as "the most harmful substance overall" (p. 759) at 77 harm points, and cannabis at 17 harm points (p. 764).

*Kava*'s relaxant effect can be attributed to the lipid-soluble kavalactones it contains (Sarris et al., 2011). Current understanding is that *kava* contains 20 different kavalactones (Bian et al., 2020), which have a range of psychoactive effects: antithrombotic, hypnotic, sedative (Cairney et al., 2002), anxiolytic (Pittler and Ernst, 2003; Singh and Singh, 2002), muscle relaxant (Duffield and Jamieson, 1988) and analgesic (Singh, 1992). These effects are understood to stem from *kava* acting to decrease neurotransmitter function in the central nervous system (Carlini, 2003; Ligresti et al., 2012; Lim, 2016).

Of the 20 kavalactones found in kava, six are considered to be key, of which one is kavain (4, KAV). Each of the kavalactones has been ascribed a chemotype number and followed with their abbreviation: 1. demethoxy-yangonin (DMY); 2. dihvdro kavain (DHK); 3. yangonin (YAN); 4. kavain (KAV); 5. dihvdromethysticin (DHM); and, 6. methysticin (METH) (see Fig. 1). Reporting on the elimination half-life of kava (the time it takes for the concentration of kava, once ingested, to reduce by half in the body), Saletu et al. (1989) calculated that a 200 mg tablet dose of kavain would take 9 h to reach this concentration (p. 188). From this, Aporosa (2008) calculated that full elimination of kava from the body could take upwards of 90 h (p. 46). However, subsequent commentators have suggested kava's half-life at more than 9 h, with an attendant increase in the time required until full elimination is achieved. This was considered to be particularly the case when kava is consumed at traditional high volumes and contains all 20 kavalactones (Bian et al., 2020).

#### 1.2. Traditionally-influenced, or naturalistic, kava consumption

Pacific Island people have drunk *kava* for more than 2000 years, with *kava* drinking continuing as a dominant traditional practice. As Pacific people have migrated, they have taken their *kava*-culture with them, with *kava* use and its related cultural practices continuing within Pacific diasporic communities. This has also influenced *kava* use among non-Pacific people who are often using *kava* as an alternative to alcohol (Aporosa, 2015, 2019b; Aporosa and Forde, 2019; Sumampow and Henry, 2021; Tecun, 2021).

Traditionally, *kava* is made in a *kumete* (traditional wooden *kava* bowl). The *kava* rhizome and lateral roots, which is often dried and pounded following harvesting, is steeped in water in the *kumete*. The *kumete* also acts as the serving vessel, with *kava* served in *bilo* or *ipu* (cups made from half coconut shells) to drinkers (see Fig. 2).

*Kava* preparation and consumption follows established cultural protocols, with drinkers typically sitting cross-legged in a circle on mats on the floor. Cultural etiquette observed during a *kava* session includes designated times of formal speech and announcements, a serving order based on hierarchy, and drinkers taking part in *talanoa* or culturally guided discussion throughout the session. The whole process is underpinned by Pacific respect-based values (Aporosa, 2014) (see Fig. 3).

On average, a traditional *kava* session will last 6 h or more (Aporosa and Tomlinson, 2014). Over this time, drinkers typically consume around 3.6 L (or 7.6 pints) of the *kava* drink.

Depending on the age of the *kava* plant when harvested and the concentration of the *kava* when mixed into the beverage, this means consumers often ingest more than 8,000 mg of kavalactones per sitting, which represents 30 times the pharmacologically recommended daily dose (Aporosa and Tomlinson, 2014). Pharmacologists typically recommend no more than 200mgs of kavalactones per day (MediHerb, 1994, p. 2), a dose level claimed to prevent cognitive impairment (Mills and Bone, 2005, p. 484, 488).

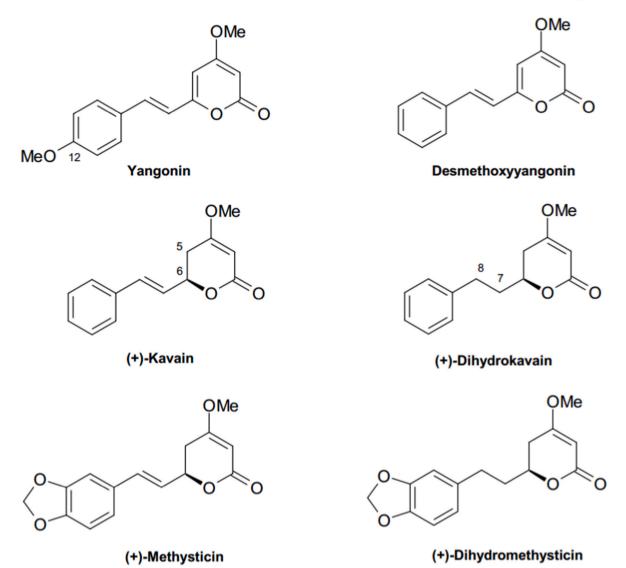


Fig. 1. Chemical structure of the six major kavalactones present in *kava* beverage made from the roots and basal stump. Me = methyl group. (Dragull et al., 2006, p. 21).



Fig. 2. Mixing kava in a kumete (kava bowl) in Auckland, Aotearoa New Zealand (photographer: Todd M. Henry, 2019).

# 1.3. Concerns about kava's impact on driver safety

The increasing international popularity of *kava*, and the mounting evidence relating to its psychoactive properties, has raised issues around how *kava* consumption affects people's cognitive functions. These concerns have particularly revolved around driver safety (Wainiqolo et al., 2015), and correspond to increased reports by the police in several Pacific nations, ANZ, Australia and the United States of suspected *kava* 

'intoxicated' drivers.

In ANZ, police report they are stopping increasing numbers of drivers who appear to be mildly intoxicated, with the suspected reason for the intoxication being *kava* (Morgan, 2014; 2017; Tokalau, 2020; Welsh, 2017). Police have reached this conclusion when, despite an observable impairment, breath-screening tests are returning negative results. Similarly, Australian police are drawing links between increased *kava* use and a corresponding increase in serious motor vehicle accidents (Fu



Fig. 3. Kava drinking by participants with mixed ethnicity in a private home (Māori owners) in Waikato, Aotearoa New Zealand. (photographer: Author, 2009).

et al., 2019); while researchers in Iowa in the United States report "kava impairment was demonstrated through four cases of self-reported kava use ... [suggesting *kava* use] may hinder one's ability to operate a vehicle safely" (Berry et al., 2019, p. 1948).

Seemingly supporting the New Zealand Police's observations, the New Zealand Institute of Environmental Science and Research (ESR) report it is becoming more common to detect kavalactones in the blood of people who have died as a result of a motor vehicle accident (Poulsen et al., 2012). However, it is acknowledged that current large knowledge gaps around the interpretation of kavalactones in the blood means it is not possible to determine when exactly these kavalactones were consumed (Poulsen and McCarthy, 2020).

With *kava* not metabolising on the breath in a similar manner to alcohol, and therefore not allowing breath screening, and with blood testing of suspected *kava* drink-drivers being ineffectual for evidential purposes, the monitoring and policing of *kava*-impaired drivers is extremely difficult.

This current lack of a suitable evidentiary standard measure, against which to assess driver competency following *kava* use, not only impedes road policing efficiency and road safety, but also hampers judicial process and the protection of innocent third-parties. So far, ANZ police have only bought two successful prosecutions against drivers whose driving was impaired by *kava* use (DCNZ, 2000; Hart, 2021; Tokalau, 2020), and there has been a similarly low level of prosecution success overseas (Swenson, 1996; Jolly, 2009).

Yet despite this, the numbers of people who are potentially driving after consuming *kava* are large. In Australia, it has been reported that, among Tongan *kava* drinkers interviewed, around 70 per cent typically drove home after attending a *kava* drinking session (Maneze et al., 2008). When this estimate is applied to the reported 20,000 people in ANZ who usually drink *kava* on Friday or Saturday night (Aporosa, 2015), this would suggest there could be as many as 14,000 *kava* users controlling motor vehicles over this time. Given the dispersed nature of *kava* drinking venues, this is travel that can include long-distance and inter-city driving (Aporosa, 2018). Although the numbers driving following *kava* use is large, what is yet to be assertained is the percentage of those numbers who are cognitively impacted at a level that compromises safe driving, together with that cognitive disruption comprises.

When account is taken of the fact that people driving while under the

influence of drugs or alcohol is a significant health and safety issue in ANZ, with an equally significant 'social cost' (Ministry of Transport, 2017, p. 4), and that -a dominant reason Pacific adults in ANZ are hospitalised is due to injuries from road traffic accidents (McCormack et al., 2012, p. 2), the need for more research to build better understanding of how *kava* affects driver safety is bought to the fore.

# 1.4. Previous studies of kava's impact on cognitive function

Prior to 2017, studies looking at *kava*'s effect on cognitive function had returned inconsistent results.

In 2017, a report into 12 clinical trials aimed at assessing *kava*'s effect on "mental function", demonstrated this inconsistency when it found that while some of the trials (four) showed that *kava* "improved accuracy and performance on visual attention and working memory", others (five) showed that "kava to have little or no negative effect on cognitive processes", while yet another reported that *kava* impaired reaction time (Sarris and McIntyre, p. 16).

Several studies, mostly led by the German researcher Klaus Herberg, have assessed kava's impacts on selected cognitive faculties in comparison with other drug substances. For instance, following a 300 mg daily dose of kava extract (decocted with ethanol), and administered over 15 days, no disruption to concentration was noted when compared with placebo-control (Herberg, 1991, 1993b). In a shorter seven-day study, and again using the same kava extract dose compared against a placebo protocol, Herberg (1992, 1993a, 1997) added 0.5% alcohol. This study showed no effect beyond the alcohol effect on concentration in control subjects given placebo. In another study, Herberg (1996; also see Herberg and Winter 1996) assessed the effects of a combination of kava extract (240 mg per day, ethanol decoction) and Benzodiazepine Bromazepam (10 mg per day) when compared with a kava extract only dose. This 14-day double-blind, randomized cross-over trial showed performance and vigilance parameters remained on the level of baseline for the kava only dose. However, impairment was identified with the kava Bromazepam combination. In one of the very few post-kava use driving focused studies, Sarris et al. (2013a) used a driving simulator to assess participant impairment following the ingestion of a kava extract (180 mg, water extraction) compared with Benzodiazepine Oxazepam (30 mg) and placebo. Results showed the kava extract dose did not impair driving ability, whereas the dose of Oxazepam reduced

concentration and alertness and caused significantly slower braking reaction time when compared against placebo. These studies suggest extracted *kava* (at the designated administration doses) does not impact selected cognitive faculties, unlike *kava* combination with, or compared to, alcohol or benzodiazepine.

Of significance is that most of the participants in the 12 clinical trials discussed earlier (which showed inconsistent results), together with the Herberg and Sarris et al. studies, consumed pharmaceutically modified *kava*, in tablet (or capsule) form. Administered at a pharmaceutically recommended dose, such tablets contain selected extracted kavalactones (Bian et al., 2020, p. 13). This is a vastly different substance to traditionally prepared *kava* consumed in aqueous form, with attendant differences in effect.

This difference between pill-style and naturalistic *kava* raises questions whether the former substance should be considered *kava* at all, or something else quite different (Aporosa, 2019a, p. 1). Equally dubious, is the common practice of extrapolating the findings of research that used modified pill-style *kava* onto naturalistic *kava* use psychopharmacology: a practice that incorrectly assumes affect correlation between the two substances.

Garner and Klinger (1985) are one of a very few researchers who have undertaken quantitative research using traditionally prepared *kava*. Their participants were given two 150 ml *bilo/ipu* of Fijian *kava* beverage – albeit an extremely small quantity compared with typical traditional drinking volumes – and then assessed for visual function. Although participants were observed to have increased pupil diameter and disturbance to oculomotor balance together with a reduction in near point of accommodation and convergence, no change was identified in visual, stereoacuity, or ocular refractive error.

The first study that aimed to understand *kava*'s impacts on cognition, and in turn driver safety, both during and following its consumption at traditional volumes, started in ANZ in 2017 (Aporosa, 2018). This was funded by the Pacific division of Health Research Council of New Zealand (HRC).

No statistically significant differences were detected in the study, which used two visual-sensory psychometric measures from the Vienna Test System battery to compare the cognitive function (particularly reaction and divided attention) of a control (non-*kava* drinking) and active (*kava* drinking) group of participants following a traditional influenced *kava* consumption session (Aporosa, 2017, 2018; Aporosa et al., 2020).

Despite this, the researchers noted mildly slurred speech and slightly deminished motor responses among the active participants, with this impact beginning to be apparent after 3 h, at the mid-point of the *kava* consumption and testing (Aporosa, 2017).

A later feasibility study sought to resolve this apparent disconnect; between what the researchers observed to be happening to active participants' cognitive abilities and the lack of any statistically significant impairment detected via testing. The feasibility study used the Brain Gauge, as an alternative testing method (Aporosa et al., 2021).

The approach and learnings from the feasibility study were then used to inform a full research study, again funded by the HRC: Pacific. The results of that full study are described in this article and reported in greater detail in a technical report to the HRC (Aporosa, 2021).

#### 2. Material and methods

The study was based within Te Huataki Waiora School of Health and supported by Te Kura Whatu Oho Mauri School of Psychology at the University of Waikato, ANZ. The study was guided by the Pacific Postdevelopment Methodological Framework (PPdMF) and the *faikava methodology*.

The PPdMF itself combines post-development theory (Peet and Hartwick, 2009, pp. 226-7) and the Fijian *vanua* research framework (Nabobo-Baba, 2016, pp. 24-36). It was specifically developed to ensure that Western-developed, -standardised and -normed psychometric measures are applied ethically and equitably when used with Pacific

peoples (Aporosa, 2014a, p. 102; Aporosa et al., 2021).

The *faikava methodology* uses a naturalistic *kava*-use environment to collect both quantitative and qualitative research data. This approach to data collection has been used for research purposes for more than 20 years, and is endorsed by the HRC through their funding of eight major research projects using this methodology (Aporosa et al., 2021, p. 83).

#### 2.1. Participants

Thirty-nine participants completed the testing: 20 in the 'active' *kava*-consuming group, and 19 in the 'control', non-*kava*-consuming group. Power calculations were utilised to inform minimum participant numbers to ensure statistically significant results (Aporosa, 2021, p. 16). All participants were males (recruitment specifically targeted males as they historically dominate as the *kava*-consuming gender<sup>1</sup>) and included people of Pacific Island and other ethnicities, with an average age of 34.12 years (SD = 9.61).

The total participant group was split in two for the testing; with testing carried out on two separate occasions, and each occasion hosting 10 active and 10 (or 9) control participants. Participants were given instructions on how to prepare for the tests, including abstaining from drinking certain beverages (alcohol, coffee, energy drinks, Coca-Cola, *kava*) for set periods before the test.

## 2.2. Faikava venue

On the day of the test, participants were transported to a *faikava* venue, created for this purpose on the university campus. As in natural *faikava*, participants sat cross-legged on woven mats on the floor while consuming *kava*. The *kava* was served from a *kumete*, in *bilo/ipu*, with the whole process guided by Pacific respect-based values.

*iSevusevu* – a Fijian-influenced cultural practice in which attendees are acknowledged and the purpose of the gathering explained (Aporosa, 2014) – was presented at the beginning of the test session; as was *tatau* (similar to *isevusevu*) at the end. These practices upheld Pacific cultural expectations and obligations and complied with the PPdMF and *faikava methodology* used in the study (Aporosa, 2014; Aporosa et al., 2021).

Over the following 6 h, the active participants drank *kava* (if they were members of the active group), engaged in *talanoa* (a Pacific form of discussion) and consumed 'chasers' (snacks typically eaten with *kava*). Chasers included fruit, salted peanuts and potato chips, and sugar-free sweets. Participants were allowed to move around to stretch their legs and to leave the *faikava* venue to use the toilet.

Participants in the control group did not drink *kava* at any point during the 6 h spanned by the testing, although did engage in *talanoa* and consume chasers.

The lead author has extensive experience in *kava* preparation and consumption protocols and ensured these were followed throughout the session (Aporosa, 2014). This included eating chasers and moving about, both of which helped recreate a naturalistic *kava* consumption setting, which is a crucial part of the *faikava* methodology (Aporosa et al., 2021).

# 2.3. Kava preparation and consumption

The *kava* used during the *faikava* session was made using dried powdered kava root and basal stump from Fiji (Aporosa, 2021, pp. 20-21). Approximately 36 L (9.51 gallons) of *kava* was pre-prepared 1 h before each test session.

During the session, this prepared kava was served to the participants

<sup>&</sup>lt;sup>1</sup> Although Fijian woman have been drinking kava for generations, contemporary kava drinking by woman from other Pacific nations is increasing, with this done in mixed gender and female-only groups. There is also a marked increase in non-Pacific woman attending *faikava* venues (Lolohea, 2021; Henry and Aporosa, 2021, pp. 188-189; Tecun, Reeves and Wolfgramm, 2020, p. 184).



Fig. 4. Kava being poured into the kumete for serving to study participants. (Photographer: Todd M. Henry, 2019).



Fig. 5. Kava being served in 100ml (0.2 pint) portions in bilo/ipu, from a kumete, during the test session (Photographer: Todd M. Henry, 2019).

in the active group in *bilo or ipu* from a traditional *kumete*, at 100 ml (equivalent to 0.2 pints) portions (see Fig. 4 and Fig. 5). Portions were served at the rate of six serves per hour, throughout the 6-h test session. All 20 of the active participants consumed their full share (3.6 L or 7.6 pints) of the prepared *kava* during the test period.

High-performance liquid chromatography (HPLC) analysis of the *kava* used in the study showed its:

- strength rating was 5 per cent total kavalactones by dry weight
- chemotype was 462531
- mean kavalactone content was 115 mg per 100 ml of kava beverage.

This equates to each active participant having consumed 1,840 mg of kavalactones at the time of the second (T2) test, 3 h after *kava* drinking started, and 3,680 mg after 6 h of *kava* consumption, just prior to the final (T3) test. This means participants consumed almost 15 times more kavalactones than the pharmacologically recommended dose.

#### 2.4. Chemical fingerprinting of kavalactones by UPLC-MS/MS

Kava was mixed by the lead author at the ESR in Wellington, replicating the aqueous beverage used during data collection. Finger printing of kavalactones in the beverage was completed by co-author Dr Rishi Pandey using targeted Ultra-performance liquid chromatography in tandem mass spectrometry (UPLC-MS). Individual fingerprints for each of the kavalactone prepared in ethanol were obtained by direct infusion of each of the six pure kavalactone standards sourced from Sigma-Aldrich, New Zealand. UPLC-MS/MS analysis and MS/MS Quantitation was performed on an AB SCIEX triple quad 5500 MS coupled to a Sciex Exion liquid chromatography system.

Briefly, 10 mL of each of the samples was sub-sampled, centrifuged at 4500 rpm for 10 min to pellet down the debris or undissolved matter. 50 microL of the clear aqueous layer was further diluted 1 in 20 with SQ water and 50 microL of internal standard diazepam-d5 was added to the mix. Briefly, Chromatographic separation of 1 µL of extracted sample injected was obtained on a Phenomenex Luna C18 column (dimension 150  $\times$  2.1 mm, 3  $\mu$ m particle size, 100 Å) maintained at 40 °C throughout the gradient run of 10 min with mobile phase A; 0.1% formic acid in water and mobile phase B; 0.1% formic acid in methanol at a flow of 0.5 mL/min. The gradient time course followed was: 0.00-5.00 min 40% mobile phase B; 5.00–6.0 min a linear increase in a gradient to 95% mobile phase B at a flow rate of 0.6 mL/min; 6.00-8.00 min a hold with 95% mobile phase B; 8.00-10.00 min a linear decrease in gradient to 40% mobile phase B and hold from 10.00 to 10.05 min at 40% mobile phase B, to equilibrate the column. The mass analyzer was an AB SCIEX 5500 Triple-Quad operated in positive ion mode, using electrospray ionisation (ESI) operated at the following conditions: gas 1, nitrogen (60 psi); gas 2, nitrogen (50 psi); ion-spray voltage, 5000 V in positive mode; ion-source temperature, 450 °C; curtain gas, nitrogen (30 psi). Nitrogen collision gas was set at medium for all experiments. The dwell times were optimised using the scheduled MRM algorithm incorporated in the AB Sciex Analyst® software and flexible window widths functionality

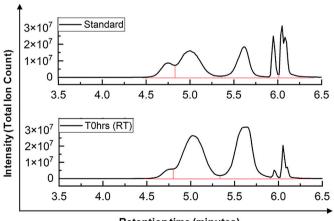
was applied. All data processing is performed using Sciex MultiQuant® 3.0.2 software with the SignalFinder1 algorithm.

The reconstructed ion chromatograms and product ion spectra were found to be consistent with the spectra acquired by direct infusion of individual pure kavalactone standard (Fig. 6 and Fig. A1.). The observed mass spectra and MRMs (quantitative and qualifier ions) for each of the six kavalactones in the aqueous kava extract agreed with those identified in the unextracted kavalactone standard mix (Fig. 7.). The quantitation of the kavalactones was performed using a six-point standard calibration curve constructed with diazepam-d5 (IS) in 40% methanol (0.12, 0.26, 0.55, 1.1, 2.2 mg/L) developed using a set of six-point calibration at different dilutions. The standard curve was fitted to a Hill's regression curve. The fresh aqueous kava extract exhibited clear fingerprint for each of the six kavalactones, with kavain (34.7 mg/L) and dihydrokavain (30.6 mg/L) being the most abundant kavalactones, followed by dihyromethysticin (15.4 mg/L), methysticin (8.4 mg/L), desmethoxyyangonin (6.2 mg/L), and yangonin (2.0 mg/L). Overall, the results demonstrated clear fingerprint of the six kavalactones in traditionally prepared fresh kava extract (Tang and Fields, 2019).

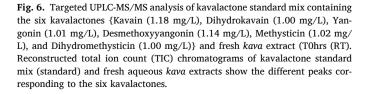
# 2.5. Brain Gauge psychometric testing

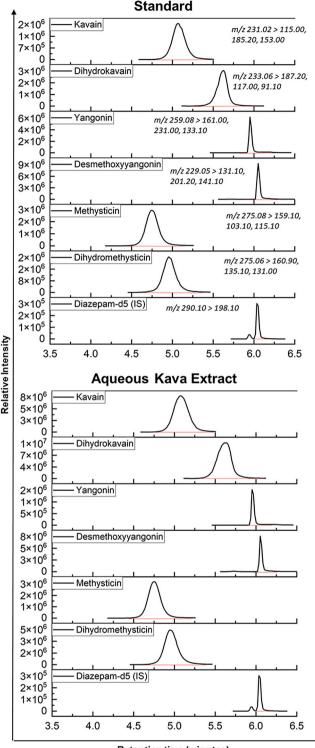
The study used the Brain Gauge (www.corticalmetrics.com) testing tool to measure subtle changes in participants' cognitive faculties, including fine motor skills and fatigue, and thereby assessing their neurological functioning (King et al., 2018). The Brain Gauge tool links to an associated Brain Gauge application, which sets tasks for participants to complete on a computer screen. The tasks are designed to measure the six attributes or domains.

- Speed including reaction time and how variable it is. Fatigue affects speed, and can be defined as how the "brain tires during a mentally demanding task", which has an impact on reaction time (Pawluk, 2018b).
- Accuracy or how well the brain can differentiate between similar sensations or stimuli. Accuracy is "responsible for integrating sensations that are detected by different parts of the body" (Pawluk, 2018a).
- Temporal order judgement (TOJ) and connectivity or how well the brain keeps track of the order of events, including sequencing (Pawluk, 2018e).



Retention time (minutes)





Retention time (minutes)

**Fig. 7.** Mass spectra for each of the six kavalactones and Internal standard (IS) diazepam-d5 as extracted and identified by targeted multiple reaction monitoring (MRM), for the unextracted kavalactone standard mix solution (Standard), and aqueous *kava* extract. MRM transitions monitored for each of the kavalactones is shown as the m/z in the of standard stacked plot. The MRM detection windows were set at 12 s and 30 s as determined by the density of MRM concurrency in chromatography, target scan time was 0.4 s, resulting in at least 15 points across the peak baseline. At least 3 MRMs per analyte were monitored, with one quantitative MRM (based on the maximum intensity of the analyte) and two qualifier ions.

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- Timing Perception or how well the brain keeps track of time, which is itself linked to "motor learning, balance and coordination, and timing accuracy" (Pawluk, 2018f).
- Plasticity or how well the "brain is able to react and adapt to changes in [the test subjects] surroundings", allowing response adjustments to new situations and environmental change (Pawluk, 2018d).
- Focus or how well the "brain is able to concentrate on the task at hand" which is "associated with motivation, attention span, determining similarities and differences between objects or events, and the ability to predict future consequences" (Pawluk, 2018c).

The Brain Gauge tool and app calculate an overall composite score (cortical metric) for participants. It also reports on scores for individual tasks and normative scores, enabling the two to be compared (Cortical Metrics, 2017). These scores can be used to assess the strategic, tactical and operational cognitive faculties that affect drivers' performance (Barkley and Cox, 2007).

Participants completed the Brain Gauge test battery:

- at the start of the study before they had consumed any kava T1
- after 3 h, which was halfway through the kava session T2
- after 6 h, at the end of the kava session T3.

Numerical data gathered during testing was exported and analysed using Student's *t*-test (Normal) and Bayesian inference techniques.

- Student's *t*-test analysis compares and measures the significance of differences between two groups: in this study, the active and control groups. The independent samples *t*-test was used for the analysis. Additionally, analysis within active and control groups across different time points used the paired *t*-test.
- Bayesian analysis produces a 'Bayes factor', which is a ratio, representing the likelihood of a specific hypothesis being correct or occurring, compared to the likelihood of an alternative hypothesis.

#### 3. Results

# 3.1. Psychometric test data

Comparisons of the within cohort data (comparing control group data with control group data, and active *kava* using group data with active *kava* using group data) at the three test points (T1, T2 and T3) showed some statistically significant ( $\leq 0.05$ , underlined) variations, and is shown in Table 1.

However, only one data set (for the Focus domain of the active group) will be discussed here. This data shows a statistically significant level of impairment to the Focus of the active participants at T2 (or following 3 h of *kava* use) (p = 0.02225). However, at T3, and following 6 h of *kava* drinking, the Focus of the active participants shows a (non-statistically significant) level of improvement (p = 0.0599). This positive change in Focus for the active group will be examined further in the Discussion section of this article, in relation to driving.

Of greater interest, is the between-cohort data (comparing data from the control group and the active *kava*-using group) at the three test points (T1, T2 and T3) and shown in Table 2. Contrary to the study's initial hypothesis, this data shows no statistically significant level of impairment for five of the cognitive domains measured by the Brain Gauge for the active participants (when compared with the control participants) over the course of the 6-h *kava*-use. These domains were Focus, Accuracy, Timing Perception, Plasticity and Fatigue.

However, this was not the case for the TOJ test scores. This data showed a significant level of regression in participants' TOJ at the T3 testing point (p = 0.007301 [underlined in the Table 2], BF = 6.193058). Fig. 8 presents a simple box plot of the results of this data. Fig. 9 shows the same data presented as a violin-plot, to provide alternative illustration.

As has been stated above, TOJ includes sequencing and is associated with "how well [the] brain is able to keep track of the order of events" (Pawluk, 2018e). King, Hume and Tommerdahl (2018) add that TOJ, as assessed by the Brain Gauge, is a "metric associated with the 'when' pathway (frontal-striatal)" (p. 4). Pawluk (2018e) elucidates that "frontal-striatal pathways control many of the brain's <u>executive func-tions</u>, including decision making, behavioral control, and information processing" [emphasis is the author's].

This finding of regression in participants' TOJ supports, to some extent, the study hypothesis – that participants in the active *kava*-using group would show changes in their neurological functioning, when compared with the participants in the control group – although, with the acknowledged limitation that *kava* appears to impact only one of the six domains measured by the Brain Gauge.

The apparent impact of traditionally influenced levels of consumption on *kava* users' TOJ, and hence executive functions, is argued to have implications for safe driving, a theme that will be explored in the Discussion section of this article.

#### 3.2. Observations

In addition to the Brain Gauge testing, the author and research assistants made observations throughout the test period regarding changes in the participants' behavior. Subtle changes were noted in many of the active participants, with these changes often becoming more noticeable after 4 h of *kava* use (Aporosa et al., 2020). Changes noted included active participants appearing somnolent, with slowed speech and slightly altered pronunciation, and a slower psychomotor response (Aporosa, 2021, pp. 32-36).

The observed changes would appear to be consistent with those from police working at roadside checkpoints in ANZ and the Pacific islands, who state that the drivers who have been drinking *kava* before being stopped typically move and speak a little slower and may slightly mispronounce their words (Berry et al., 2019; Galuvao, 2018; Kalura, 2018; Mishra, 2018; Morgan, 2014; 2017; Tokalau, 2020; Welsh, 2017).

Another observation of interest was the apparent change in Focus of the participants, at each of the three testing points, and how this differed between the active and control groups.

Observationally, at T1 baseline testing, the control participants generally appeared much more focused on the computer screen than the average active participant. For instance, the control participants appeared to lean further forward in their chair, allowing closer proximity to the computer screen and suggesting an increase in concentration when undertaking the Brain Gauge tasks. At T2, the control participants appeared slightly less intent and focused, adopting a more relaxed posture, when compared with T1; then at T3, appeared vastly more relaxed.

#### Table 1

Within cohort data at T1, T2 and T3 with significantly statistical data underlined.

Test	Focus	Accuracy	Temporal Order Judgement	Timing Perception	Plasticity	Fatigue
Control-T1 v Control-T2 Control-T1 v Control-T3	0.03943	0.003769	0.2057	0.7241	0.001382	0.0267
	0.04786	0.6725	0.08645	0.2126	0.1324	0.05641
Active-T1 v Active-T2 Active-T1 v Active-T3	0.02225	0.6374	0.8907	0.09863	0.9972	0.3301
	0.0599	0.07084	0.2512	0.1242	0.2446	0.8949

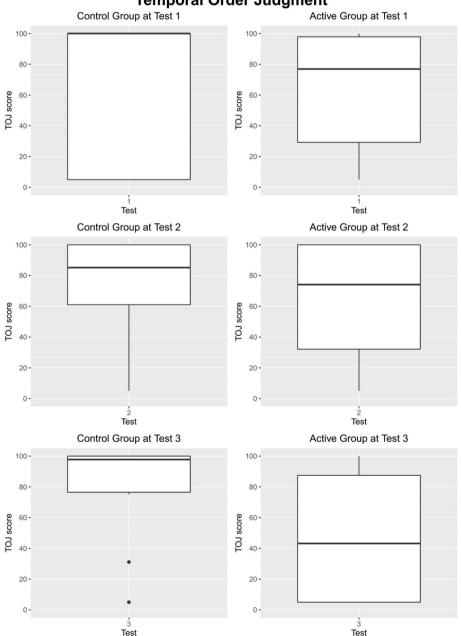
#### Table 2

Between cohort data at T1, T2 and T3 with significantly statistical data underlined.

Test	Focus	Accuracy	Temporal Order Judgement	Timing Perception	Plasticity	Fatigue
Control-T1 v Active-T1	0.1056	0.353	0.7854	0.3471	0.4133	0.7914
Control-T2 v Active-T2	0.2257	0.2664	0.5427	0.07599	0.3003	0.3568
Control-T3 v Active-T3	0.1243	0.6883	<u>0.007301</u>	0.4068	0.3207	0.3074

Conversely, the active participants appeared more relaxed than the control group at T1, although at T2 and T3 there appeared to be an incremental increase in how they sat in their chairs and focused and interacted with the Brain Gauge task. For instance, at T1, the active participants appeared relaxed both in their seated position and in how they engaged with the Brain Gauge task. However, at T2, most of the active participants appeared to exhibit a greater level of concentration on the task when compared with T1; then at T3, appeared even more focused on the Brain Gauge tasks than at T2.

Admittedly these comments are based on speculation linked to observation. However, this change in demeanor by the active participants from T1 to T3, also appears to be reflected in the active participant Focus scores and the shift from a statistically significant level of regressed Focus at T2 to an improved level of Focus at T3. The change is also reflected (to a lesser degree) in the Fatigue data, which showed a non-statistically significant level of Fatigue improvement from T2 to T3 (as shown in Table 1).



# **Temporal Order Judgment**

Fig. 8. Simple box plot showing the results of the Temporal Order Judgement data.

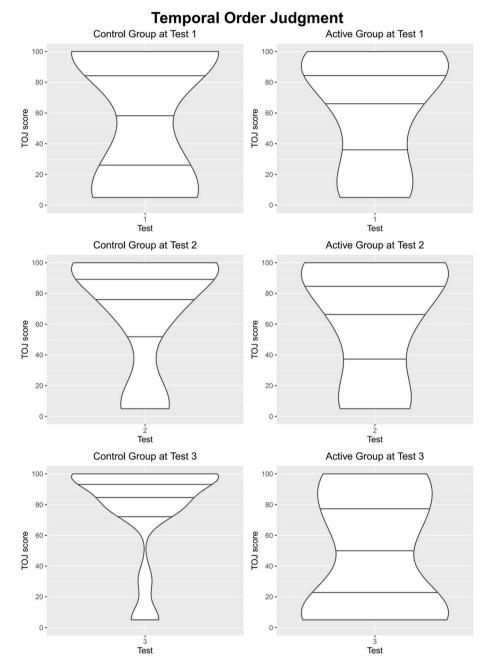


Fig. 9. Violin-plot showing the results of the Temporal Order Judgement data.

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#### 4. Discussion

Taken at face-value, what the results currently suggest is that *kava* may have a small positive effect on Focus. In other words, that following 6 h of traditionally influenced *kava* use, the *kava* potentially improves how well the "brain is able to concentrate on the task at hand" (King et al., 2018; Tommerdahl, 2018), as well as enhancing "motivation, attention span ... and the ability to predict future consequences" (Pawluk, 2018c). This in turn would tend to slightly enhance a driver's alertness and therefore improve driving safety.

However, it is argued that any suggestion that *kava* may positively effect Focus needs to be balanced against the TOJ results. The data shows that the active participants TOJ as significantly impaired at the final T3 testing point, when each of the kava users had consumed 3.6 L of *kava*, containing 3,680 mg of kavalactones.

Pawluk states that the Brain Gauge measure of TOJ is associated with the "integrity of the frontal-striatal cortex ... [with] the frontal-striatal pathways control[ing] many of the brain's executive functions, including decision making, behavioral control, and information processing" (2018e). Elsewhere, it is reported that executive function plays a vital role in safe driving, with the operational components of executive function listed as decision-making/judgment, impulse control/inhibition, self-awareness/insight, cognitive flexibility, planning and working memory (Asimakopulos et al., 2012). Anstey et al. (2005) add that "executive function is necessary for integrating information and planning a response" (p. 46).

This apparent confusion in the data – which on the one hand shows an improvement in Focus scores for the active *kava* drinking participants from the T2 to T3 tests, while at the same time showing a significant decline in TOJ, and therefore a negative impact on executive function, over the same period – was discussed with several psychopharmacology experts. Although unable to explain the anomaly, these experts recognised that understanding of the effects of *kava* on cognition, when consumed at traditionally influenced volumes (as opposed to modified pill-style *kava*), is still new and evolving. What also needs to be recognised is that executive function "is a complex construct to both understand and assess" (Asimakopulos et al., 2012, p. 423), suggesting that the confound cannot be solely explained by reference to the *kava*-science knowledge gap.

It is valuable at this point to further consider *kava*'s effects in comparison to other drug substances, particularly as the effect of *kava* has been (incorrectly) compared with alcohol intoxication, as well as laudanum, cannabis, opiates (narcotics) and hallucinogens (Aporosa, 2019a).

Earlier, it was stated that unlike alcohol and most other recreational drugs, *kava* drink induces a relaxed state and clear-headedness, rather than euphoria or hallucinations (Aporosa, 2011, 2019a; Carlini, 2003). Much of that understanding is based on ethnographic commentary. This study's findings add quantitative support to that qualitative data, demonstrating how vastly different *kava*'s effects are to those of the drug substances it is commonly, and incorrectly, compared with.

As the findings show, while *kava* has a significant impact on TOJ, no interference occurs to Focus, Accuracy, Timing Perception, Plasticity or Fatigue, cognitive faculties typically disrupted by alcohol and cannabis use. They also show that *kava*'s effect cannot be described as hallucinogenic; drugs that do fall within this category typically cause hallucinations and anomalies in perception, together with considerable change in subjective thought, consciousness and emotion (Goldberg and Dillon, 2005). Additionally, this study shows that *kava*'s effects cannot be described as narcotic; another effect descriptor commonly applied to *kava*. The U.S. Drug Enforcement Administration (2020) states that "the

term 'narcotic' comes from the Greek word for 'stupor'" (p. 1); such an effect is clearly lacking in high-volume *kava* use. Finally, the present discussion demonstrates that the common term used to capture *kava*'s effects – 'kava intoxication' – is both misleading and incorrect.

Instead, this study clearly shows that *kava*, when consumed in naturalistic settings over many hours, has unique but subtle effects. These are vastly less impactful on cognitive faculties, and very different to the effects of alcohol, cannabis, hallucinogens and narcotics. However, it must be noted that although this present discussion explores differences in effects between differing substances, it is not suggesting that *kava* has no impact on driver safety.

Earlier in this article, it was explained that traditionally influenced *kava* consumption typically occurs in communal environments, over many hours, with users often drinking more than 20 times the pharmaceutically recommended daily dose; and an estimated 70 per cent of those users then driving home, some long distance and inter-city. This study indicates that such high traditional volumes of *kava* consumption can significantly impair TOJ, including decision making, behavioral control and information processing, all crucial aspects of driver safety.

This raises the issue and challenge of how to modify driver behavior when it comes to combining *kava* consumption and driving. This challenge was taken up by the lead author, as part of the current study, via a trial for a culturally appropriate, Pacific language friendly behavior modification programme. This took the format of a multi-lingual brochure, developed and made available at *faikava* venues. This programme and its results are explained in the research technical report to the HRC (Aporosa, 2021, pp. 39-46).

#### 5. Conclusion

The study and understanding of *kava* psychopharmacology can be confusing. Much of the terminology used in ethnographic studies and the media to explain *kava*'s effects at high dose is misleading (Aporosa, 2019a). Additionally, most research aimed at understanding how *kava* effects cognition and behavior has used tablets or capsules containing selected extracted kavalactones; a vastly different substance to natural *kava* consumed in *faikava* settings (Aporosa, 2019a; Aporosa et al., 2021).

Despite this, very little *kava* psychopharmacology research has been completed using *kava* as it is typically consumed in naturalistic settings (Aporosa et al., 2021).

Also of significance is that an often-overlooked aspect of any substance use is the associated influences and impacts of 'set and setting', or a person's mindset related to the social and physical environment of the substance being used (Zinberg, 1986; McElrath and McEvoy, 2002; Aporosa et al., 2021).

Further contributing to *kava* psychopharmacology confusion is the inconsistency and subjectivity across studies reporting *kava*'s impacts on cognition, with some research suggesting that *kava* improves mental function, whereas others state "kava to have little or no negative effect on cognitive processes" (Sarris and McIntyre, 2017, p. 16). Again, most of these studies used tablet-form *kava*, although the findings have often (erroneously) been interpreted as applying to naturalistic traditionally influenced *kava* users.

Add to this confusion the increasing reports from police of stopping drivers, mostly of Pacific ethnicity, who they believe are impaired by *kava* use, and their limited ability to measure and deal with this; and the also increasing anecdotal reports linking *kava* use with Pacific people's over-representation in motor vehicle accident statistics, and it becomes apparent why both the police and the ESR have called for research to understand the effects of naturalistic high-volume *kava* use on driver

#### safety.

In this second study of its kind, a somatosensory psychometric tool (Brain Gauge) was used to measure slight changes in six specific neurological functions – namely Focus, Accuracy, Temporal Order Judgement, Timing Perception, Plasticity and Fatigue – during and following *faikava*. Of these functions, only participants' Temporal Order Judgement (TOJ), which is associated with a person's executive function, was shown to be (strongly) statistically impaired when compared with the control group (Aporosa, 2021).

This is both a unique and new finding. It suggests that when consumed at traditional use volumes, *kava* compromises driver safety via a disruption to TOJ only. However, the nature of this impairment is not the same as that caused by alcohol, cannabis, and other recreationally consumed substances that are known to impact drivers' cognitive abilities. This research also adds to discussion in an earlier section comparing the effects of pharmaceutically recommended extracted doses of *kava*, which had no impact on cognitive faculties, with small doses of Benzodiazepine and alcohol, which did. It must be pointed out though that high doses of extract *kava* may nevertheless have a negative impact on driver or machine use safety (Hänsel, 1993). Further, the current findings add quantitative understanding to ethnographic data on *kava* effects, suggesting the often-used term '*kava* intoxication' is misleading and incorrect.

Although this study generated new neurophysiological understanding concerning *kava* use, it also highlighted gaps in current *kava* psychopharmacology knowledge, particularly around how and why *kava* has an impact on selected cognitive facilities, but not others. This anomaly requires further investigation. The understanding is not likely to advance until, at a minimum, "the neurophysiological mechanisms associated with kavalactone metabolism" (Bwarenaba et al., 2017, p. 5) when consumed at traditionally influenced use volumes, are understood. This lack of understanding essentially hampers further *kava* drink-driving research, except for learnings likely to derive from testing *kava* users in a driving simulator.

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# Availability of data and materials

The datasets used and analysed during the current study are available from the author on reasonable request.

#### Declaration of conflicting interests

The lead author declares he has been drinking *kava* for more than 20 years and that this is an important part of his Fijian ancestry and cultural practice. Second-author, Mr Hakau Ballard, also uses *kava* as part of his Tongan ancestry.

# Ethical approval and informed consent

Ethics approval for this study was granted by the Health and Disability Ethics Committee within the New Zealand Ministry of Health (reference number 19/NTB/44).

#### CRediT authorship contribution statement

**S. Apo Aporosa:** Conceptualization, Funding acquisition, Investigation, Methodology, Project administration, Resources, Data curation, Writing – original draft, Writing – review & editing, (approx. 70%). **Hakau Ballard:** Formal analysis, roles included formal analysis (statistical analysed and interpretation of the research data) and, Writing – review & editing, (approx. 10%). **Rishi Pandey:** Formal analysis, roles included formal analysis (chemical fingerprint) and, Writing – review & editing, (approx. 10% each), and. **Mary Jane McCarthy:** Formal analysis, roles included formal analysis (chemical fingerprint) and, Writing – review & editing, (approx. 10% each), and. **Mary Jane McCarthy:** Formal analysis, roles included formal analysis (chemical fingerprint) and, Writing – review & editing, (approx. 10% each).

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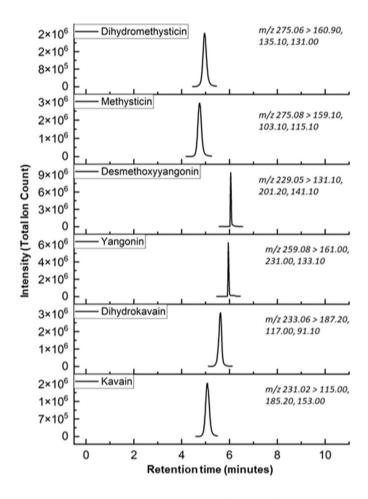
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Finally, this study would not have been possible without those who joined as research participants. Due to confidentiality, they cannot be named.

# Appendix A



**Fig. A1.** Mass spectra finger print for kavalactones obtained by direct infusion of each of the six pure kavalactone standards prepared in methanol on the AB SCIEX 5500 Triple-Quad mass analyzer. MRM transitions identified for each of the kavalactones is shown as the m/z in the of standard stacked plot. The MRM detection windows were set at 12 s and 30 s as determined by the density of MRM concurrency in chromatography, target scan time was 0.4 s, resulting in at least 15 points across the peak baseline. At least 3 MRMs per analyte were monitored, with one quantitative MRM (based on the maximum intensity of the analyte) and two qualifier ions.

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