Kava and Balance

The Effects of Traditionally Influenced Kava Consumption on Human Physiology

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Abstract

In recent years there has been increasing interest in the effects of kava as a medicinal substance. However, there are unknown facets of kava effects that continue to be the issue of debate within the literature. One such area is the discussion about whether kava causes drinkers to lose their balance, which has been largely ignored by researchers. There is a single comprehensive study which investigates the issue (Prescott, Jamieson, Emdur, & Duffield, 1993). The thesis opens with an introduction which outlines the basis of kava research and the justification for this research project; it is the first study to investigate the association between consuming kava at naturalistic volumes and increased body sway. The need to understand the issue is elevated by evidence which suggests that kava drinkers routinely drink kava for many hours and then drive home.

As part of the requirements for this project, a journal article was submitted as a result of a data collection session involving experienced kava drinkers (N=6; low numbers were in part caused by time constraints and the COVID-19 lockdown period). The methodology was based on past naturalistic kava experiments and influenced by Post-Development Pacific Framework. Methods: Data collection surrounding a culturally influenced kava session (6 hours), involving experienced kava drinkers, 10-minute drink intervals (100ml kava), and three test batteries; pre-test (baseline), mid-test (3 hours) and post-test. Tests: Force Plate Centre of Pressure (COP) – 3x30s closed eyes, closed stance condition, and y-balance test – anterior condition, 3x right leg then 3x left leg.

The current study neither confirmed nor disputed the evidence that kava may induce increased body sway when consumed at high volumes. As a result, further research is needed as there is still a wealth of anecdotal evidence which suggests the effect may be moderate or significant. However, the current study did not replicate the results of Prescott et al., (1993), a study which involved a similarly low number of participants, lower volumes of kava consumption, and similar data collection duration. As a result, the results of this thesis project suggest further research is needed; to fill the gaps in kava pharmacodynamics and addressed issues apparent in replicability of body sway studies. The study provides a protocol and methodological framework for future, more controlled studies investigating kava's effects on human body sway.

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I would like to thank Dr Kim Herbert-Losier for being willing to give expert guidance and insight concerning biomechanical research instruments and techniques, which were an essential part of the project, as well as her encouragement and advice.

I would also like to acknowledge the participants who volunteered to be part of my research project. Their time is valuable, and each of them was willing to give up a whole day for the data collection kava session. Without their participation and enthusiasm for the project, the research conducted here would not have been possible.

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List of Abbreviations

COP – Centre of Pressure

BZD – Benzodiazepine

GABA – Gamma-Aminobutyric Acid

ACH – Acetylcholine

ACHE – Acetylcholinesterase

MAO-B - Monoamine oxidase B

NMJ – Neuro-muscular Junction

KAV – Kavain

DHK – Dihydrokavain

YAN – Yangonin

METH – Methysticin

DYN – Desmethoxyyangonin

DHM – Dihydromethysticin

ml- millilitre

mg – milligram

L – litre

M – mean

Std Dev – Standard Deviation

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Preface

I first encountered kava when I was 11 years old. My family and I were on holiday, staying on a resort island in Fiji. Typically, at the end of dinner each night, a band played for the benefit of tourists with sunstroke. The group of musicians were situated at the end of a small pier, in a semi-circle, surrounding what I now know was a tanoa or kumete (kava bowl). One night, I approached them. They were polite and let me sit with them. One of the men offered me a coconut shell filled with kava, a brown-grey substance in the night, which I consumed without thinking. It was all the kava I had tasted and would continue to the only kava I had tasted for almost a decade.

Later, I was finishing up an undergraduate degree. A research fellow sitting in a well-lit corner office of a drab university building invited me to join a kava session. The research fellow happens to be the supervisor of this current thesis, and he. I did not think too much of the drink to begin with. I liked the taste, and the environment of relaxed, non-judgmental conversation, but I could not appreciate the point of drinking it. The fact that the social aspect of kava was the purpose of consumption only became apparent to me when I was invited to a casual fono (meeting), which occurred twice a month, where students and researchers could engage with each other around the kava bowl. The space becomes a welcoming place to discuss research and study issues, in a setting where enjoying kava is central but does not interfere with conversation.

As members of this group, we passed each other kava bowls (bilo) and relaxed against the backdrop of conversations. These first experiences have been mimicked in future kava sessions which I have attended and formed my image of a faikava or kalapu (Tecun, 2017). I understand that kava ceremonies are more formal and ritualised elsewhere in Oceania and that cultures drink kava in different ways. However, for a university student with little knowledge of kava culture, this was how naturalistic kava consumption can be defined, at least in the casual setting in which I have drunk kava.

Chapter 1

Introduction

In recent years there has been a generally accepted perception that kava causes instability in consumers, which is described in terms such as induced ataxia, increased instability, intoxication, and increased body sway (LaPorte, Sarris, Stough, & Scholey, 2011; Perez & Holmes, 2005; Prescott et al., 1993; Turner, 1986). This thesis attempts to assess how traditionally influenced kava beverage effects human balance. Kava is both the plant (Figure 1) and a beverage (Figure 2) made from the kava plant (*Piper methysticum*) which is consumed throughout Oceania (Lebot, Merlin, & Lindstrom, 1997). The kava plant is harvested, and its roots are ground and mixed with water to make the kava beverage (Aporosa, S., 2019; Balick & Lee, 2002; Sarris, LaPorte, & Schweitzer, 2011). Current kava practices generally involve using dried kava rootstock (Teschke, Qiu, & Lebot, 2011).

Figure 1 The kava plant (Piper methysticum) in Tonga (Photographer: Todd Henry, 2019)

Historically, Oceanic people have understood that kava has medicinal properties; specific cultivars were chosen for separate ailments (Lebot et al., 1997 pp.112-16; Whistler 2000, p.112), including; headaches, aches and pains, migraines, sleeping problems, respiratory tract irritation, though a more general use was to induce physical and mental relaxation – reduce anxiety and worries (Sarris et al., 2013; Lebot et al., 1997, pp.112-16). The cultivars have been divided into four categories; noble – acceptable for commercial export and general consumption, medicinal and tu dei varieties – the export of which is prohibited, and wichmanni – the wild variety of kava.

An important distinction made in this thesis is between studies involving traditionally influenced kava consumption (>3000mg kavalactone/day; Figure 2) and clinical trials where research administer kava pills in pharmacologically approved doses (<400mg kavalactone/day); very few kava studies have involved the full kava beverage (Aporosa, Atkins, & Brunton, 2020; Aporosa, 2017; White, 2018). As a result, the research that is currently available does not demonstrate kava effects comprehensively, and those studies concerning balance which have involved traditionally influenced kava have either assessed case studies retroactively and relied on anecdotal evidence (Ketola,

Viinamäki, Rasanen, Pelander, & Goebeler, 2015; Perez & Holmes, 2005; Tith & Lalwani, 2013), or implemented flawed methodologies (Prescott et al., 1993).

Research into whether kava has detrimental effects on balance has not been conclusive, although some papers imply it may have a significant impact (LaPorte et al., 2011; Nachbauer, Eigentler, & Boesch, 2015; Perez & Holmes, 2005; Prescott et al., 1993). This thesis aims to add to a recently developed field of research; clinical investigations of kava effects involving traditionally influenced consumption volumes and settings (Aporosa et al., 2020; Aporosa & Tomlinson, 2014). The thesis will also review the current state of kava literature, where body sway and body balance is situated within past studies (Berry, Gilbert, & Grodnitzky, 2019; LaPorte et al., 2011; Prescott et al., 1993), and provide quantitative data concerning kava and balance, in the form of a clinical study that is the first of its kind; using AMTI ACCUgait Force Plate (Advanced Mechanical Technology Incorporated) and a Y-Balance Test (Shaffer et al., 2013) in a traditionally influenced kava setting. The results of this study will add to the literature which continues to investigate whether kava has a deleterious effect on human balance and physiological performance (LaPorte et al., 2011). This study also acknowledges the pre-eminence of Oceanic communities, whose knowledge and cultivation of kava provide the basis for any interest from outside clinical researchers (Aporosa, 2014; Kautu, Phillips, Steele, Mengarelli, & Nord, 2017; Ratuva, 2009), including the current author.

This study forms the first-ever investigation of kava effects involving biomechanical measurement instruments – AMTI ACCUgait Force Plate (Miyata et al., 2015), and Y-balance Test (Shaffer et al., 2013). As a result, it fits within the kava literature as a preliminary investigation, with the fully acknowledged expectation that future researchers will likely be able to improve on the current thesis. Furthermore, this study becomes part of a small amount of kava research which has investigated clinical implications of kava in a traditionally influenced context (Aporosa et al., 2020; Aporosa, 2017; Aporosa & Tomlinson, 2014). Research has not addressed the issue of kava effects on human body balance, stability, and body sway adequately. However, because only one clinical study has investigated body sway in the past (Prescott et al., 1993), and meta-analyses continue to make references to increased body sway when no further studies have been conducted to confirm the hypothesis that kava consumption causes an increase in body sway (LaPorte et al., 2011; White, 2018), a more concerted effort to establish kava effects on human body sway is essential. This thesis provides a framework for how the kava 'body

Figure 2 Traditionally influenced kava beverage served in a bilo (Photographer: Todd Henry, 2019)

sway' literature can include research concerning traditionally influenced consumption volumes (Aporosa, 2014), based on the methodologies used in similar kava studies (Aporosa et al., 2020; Aporosa, 2017)

Thesis Overview

Chapter 2 provides a brief overview of kava pharmacology and includes a discussion on kava chemistry and how it is prepared (Lebot, Do, & Legendre, 2014; Teschke & Lebot, 2011), as well as pharmacological aspects of consumption (Ooi, Henderson, & Pak, 2018; Pittler & Ernst, 2003; Sarris et al., 2011). Clinical aspects of kava are also discussed in terms of treatment and safety (White, 2018). Chapter 2 then concludes with an introductory comment about how results from clinical trials may provide useful insights into kava's effect on human body balance.

Chapter 3 examines the subject of kava's effect on human body balance, and how the evidence is presented in research (Prescott et al., 1993). The discussion is opened with an explanation of historical accounts of how kava affects balance (Cawte, 1985; Showman et al., 2015; Turner, 1986), and how kava effects have been inferred from studies involving other substances. The discussion includes detailed examinations of individual studies which illuminate different aspects of kava effects on body sway (Prescott et al., 1993), acquired ataxia (Perez & Holmes, 2005), and drug interactions (Tawfiq, Nassar, El-Eraky, & El-Denshary, 2014; White, 2018). The chapter culminates in a discussion of the reliability of claims concerning how kava caused increased body sway or instability.

Chapter 4 describes this study's methodological approach, based on post-development frameworks outlined in Aporosa (2014). The chapter gives an overview of the participants, the analysis of the data used for the study, and the procedure employed. The discussion also includes an explanation of outsider-insider research concepts, ethics approval process and participant screening processes

Chapter 5 is a journal article, submitted to the *Pacific Health Dialogue: The Journal of Pacific Research* as part of the requirements for the University of Waikato master's Degree. The study was an experimental investigation of kava effects of COP and lower limber dynamic balance, taking place over a six-hour kava session.

Chapter 6 is a discussion of the results of the study present in Chapter 5 concerning the literature review in Chapter 2 and Chapter 3. The results did not find that kava increased body sway or lower limb dynamic balance. However, the results did demonstrate contrasting data to the previous study which found that kava increased body sway significantly (Prescott et al., 1993). There is also a

description of the limitations of the study, and observations of possible insights for future studies which may be drawn from the research project. The thesis is completed by a conclusion which forms a subsection of this chapter.

Chapter 2

Kava Psychopharmacology

Introduction

This chapter will examine the current state of knowledge in terms of kava psychopharmacodynamics. Kava is the beverage made from the kava plant (*Piper methysticum*) which is consumed throughout Oceania (Aporosa, S., 2019; Balick & Lee, 2002; Sarris et al., 2011). Current kava practices generally involve using dried kava rootstock (Teschke & Lebot, 2011). Historically, oceanic people understood kava's medicinal potential. Specific cultivars were chosen as targeted treatments (Lebot et al., 1997 pp.112-16; Whistler 2000, p.112; Sarris et al., 2013; Lebot et al., 1997, pp.112-16). However, kava is of interest to known Oceanic researchers searching for a potential replacement for BZDs as an anxiolytic treatment, which are known to have severe negative side-effects (Pittler & Ernst, 2003; Sarris et al., 2020; White, 2018). Research continues to address the unknowns of kava; the exact mechanisms of action, therapeutic potential, and safety concerns associated with consumption (Kautu et al., 2017; Sarris et al., 2020; White, 2018). This chapter briefly examines the kava literature to describe the active components of the kava rootstock, pharmacokinetics and pharmacodynamics of kava, as well as the limited evidence which describes neurobiological aspects of kava. This chapter concludes with a comment on the insights which can be drawn from clinical aspects of kava research when considering this study which focuses on the effect kava has on balance.

Kava Chemistry

According to Thompson, Ruch, and Hasenöhrl (2004), kavalactones readily cross the blood-brain barrier once they are ingested. According to the study, kavalactones reach maximum potency within the brain after 45 minutes. Lebot et al., (1997) have discussed how kavalactones are rapidly absorbed through the stomach membrane. Furthermore, early scientific investigations into kava suggested mastication broke down the starch into sugars which were then able to be effectively transmitted into the bloodstream (Lewin 1886, cited in Lebot et al., 1997). More recent research found that the psychoactive action of kava depended on the separation of active lipophilic resins when mixed with water – which was assisted by chewing (Lebot et al., 1997, pp. 57-58). The resin is insoluble in water; thus, the resinous particles are suspended in the water. When the drink is consumed, the resinous particles transition into the bloodstream through the stomach membrane.

The principle active ingredients within kava are kavalactones (LaPorte et al., 2011; Lebot et al., 1997; Prescott et al., 1993) which consist of 13 carbon molecules, attached to a lactone, a cyclical carbon ring, with double-bonded oxygen (Balick & Cox, 1996, pp. 161-162). Researchers have identified more than 20 separate kavalactones, of which six kavalactones account for >90% of the psychoactive properties in kava (Figure 3) - desmethoxyyangonin (DMY) [1], dihydrokavain (DHK) [2], yangonin (YAN) [3], kavain (KAV) [4], dihydromethysticin (DHM) [5] and methysticin (METH) [6]. Each cultivar is defined by descriptive analysis of kavalactone volumes (Lebot et al., 1997, p57; Rowe et al., 2011; Segone et al., 2020, pp.1-2; Showman et al., 2015, pp. 58-60). Kava is into four different varieties; noble, medicinal, tu dei, and wichmanni (Teschke, Sarris, & Lebot, 2011) Noble varieties have been defined as those cultivars which have; high concentrations of DHK (2), DYM (5), and KAV (4; (Lebot et al., 1997; Lebot, Michalet, & Legendre, 2019, p. 9). Isa – 254631, and Mahakea – 245361, are noble cultivars (Wang et al., 2015, p.1168).

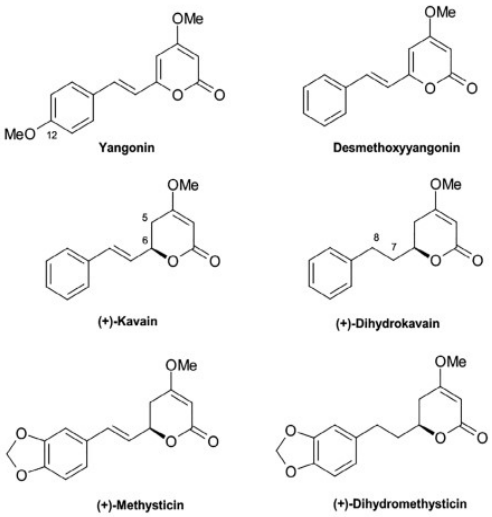

Figure 3 Chemical structure of the six principal kavalactones; Me=methal (Dragull, Lin, & Tang, 2006)

Aside from kavalactones, other compounds are present in kava, at varied volumes; flavokavin A, B, C other chalcones, and alkaloids (Lebot et al., 1997), which likely impact the psychoactive properties of a given cultivar (Lebot et al., 2014). Authors have stated that a drug's action is dependent on each of its active constituents, rather than just the principle compounds (Nutt, 2012). The effects and safety of substances are also altered by contaminants, which can cause harms or unwanted effects (Nutt, 2012; Teschke & Wolff, 2011). The kava botanist Dr Vincent Lebot has made related comments about kava, saying that if you extracted the peripheral compounds from the kava beverage, then the effect would be different to those experienced by consumers of traditionally influenced kava (Lebot et al., 1997; Goldberg 2019). This thesis focuses on traditionally influenced kava, which is defined by Lebot; "Kava is kava; it is the traditional beverage prepared by cold water extraction of the ground organs of the plant Piper Methysticum and nothing else (Lebot, 2018)".

There are a few reasons why kavalactones are given the most attention out of all the compounds present in kava. Firstly, kavalactones are the most abundant psychoactive element within naturalistic kava. The second reason is, specific kavalactones, either isolated from kava (Kautu et al., 2017), combined with other kavalactones (Sarris et al., 2009; Sarris et al., 2012), have been efficacious as anxiolytic substances, and have other promising properties; hypnotic, anti-cancer, and

neuroprotective (Celentano et al., 2019; Tzeng & Lee, 2015). Thirdly, research concerning drugs which have a clinically defined function (Kautu et al., 2017; White, 2018), is often restricted to fields where there have been past successes so that other perspectives and opportunities are ignored (Bille, 2019; LeDoux, 2019). Fourthly, companies interested in kava seem to be content to sell their products based on the current incomplete literature and limited evidence (Fiji Rugby Union 2019; Goldberg 2019; Sarris et al., 2012; White 2018). As a result of the bias towards kavalactones in the kava literature, the other compounds which likely influence the effects of the traditionally influenced kava beverage may have been overlooked (Goldberg, 2019; Lebot et al., 2014; Lebot et al., 1997).

The most prominent alkaloid present in the kava plant is pipermethystine (Lebot et al., 1997). it is toxic and predominantly found in the aerial parts of the plant (Cock & Cheeseman, pp.506-08). Importantly, pipermethystine is not present in the rhizomes or roots at high volumes, so should not be of concern provided the kava is produced correctly, as per recommendations made by Teschke and Lebot (2011). The current understanding is that lateral roots contain the highest concentration of kavalactones (Rowe et al., 2011, p.1; Wang et al., 2015, p.1167), while the concentration diminishes towards the aerial part of the plant (Lebot et al., 2014). Flavokavains, a class of chalcone, are also found in kava, at low volumes in the lateral roots (Lebot et al., 2014). Chalcones are open-chain flavonoids, which are also found in; liquorices, willow, apples, and oranges (Schnekenburger & Diederich, 2015). They possess high therapeutic potential as an anti-inflammatory, antioxidants, anti-infective substances, and chemo-preventive properties have been associated with various chalcones (Zenger et al., 2015, p.6376; Celentano et al., 2019). Three well-known chalcones are present within kava; Flavokavain A, B, and C (Lebot et al., 2014). However, research also suggests flavokavain B may be hepatoxic (Lebot et al., 2014; Teschke & Lebot, 2011; Teschke, Qiu, et al., 2011). In research concerning physiological effects of kava, kava must be understood as a compound substance containing various volumes of separate chemicals (Kautu et al., 2017, pp.4-5; Teschke et al., 2011, 99-100), instead of a single entity. Therefore, many of the studies discussing are limited by their focus on kavalactones, as well as their use of pills or capsules (<400mg kavalactone) rather than traditionally influenced kava beverage (Aporosa et al., 2020).

Mechanisms of Action

Kava appears to have multiple mechanisms of action within the Central Nervous System (CNS) and Neuromuscular Junctions (NMJ). Pharmaceutical mechanisms of action can often be somewhat tailored (LeDoux, 2019; Miller, 2013; Nutt, 2020). However, In a recent interview, the neuroscientist Joseph Ledoux explained that there is no such thing as targeted drug therapy (LeDoux, 2019). Kava

may be described as GABAergic because it interacts with GABA receptors (Wang et al., 2020; White 2018), but all GABAergic substances have a mechanistic effect which spans the whole body as well as the targeted areas of the brain (Kautu et al., 2017; LeDoux, 2019). Some GABAergic substances, i.e. BZDs, are associated with dizziness, grogginess, nausea, slurred speech, and excessive next-day tiredness (LaPorte et al., 2011; LeDoux, 2019; White, 2018). Due to these issues, researchers suggest kava may have the potential to be a non-addictive alternative to BZD (Pittler & Ernst, 2003) because it has a milder effect on the body and does not result in any of the negative side-effects associated with BZD treatments (Showman et al., 2015).

Kava has the potential to be a GABAergic substance set apart from past anxiolytic, anti-depressant, and hypnotic treatments. Evidence suggests it is not addictive (LaPorte et al., 2011; Pittler & Ernst, 2003) and demonstrates reverse tolerance (Singh, 2004) characterised by long time consumers not developing a need for more kava to obtain the same effects (p.132). However, there are several studies which suggest that one of the mild side-effects which may be associated with kava is increased body sway (Prescott et al, 1993; Singh 2004; LaPorte et al., 2011). Although GABA is suggested as the neurotransmitter responsible for that effect (LaPorte et al., 2011), there are other mechanisms which influence kava pharmacodynamics that may influence kava's effects on human physiology (Lebot et al., 2019).

Several neurotransmitter interactions seem to form the basis of kava pharmacodynamics (White, 2018). However, research does suggest that precise mechanisms of action remain uncertain (Kautu et al., 2017). This is compounded by two factors; the majority of studies have used low volumes of kavalactones (<400mg/day), or an isolated selection of individual kavalactones (Aporosa et al., 2020; White, 2018), and kava effects are often inferred from *in vivo* and *in vitro* studies using animal models are not likely to be reliable (Aporosa et al., 2020; Kautu et al., 2017; Raza, 2015). However, there are numerous useful insights to be gained from examining past studies, particularly in regard to hypothesising possible effects caused by consumption of noble kava, which contain high volumes of KAV and DHK (Teschke & Lebot, 2011; Wang, Qu, Bittenbender, & Li, 2015).

Kava's mechanism of action is allosteric, which means it facilitates neurotransmission rather than imitating endogenous neurotransmitters (Miller 2013). Kava seems to interact with GABA (A) receptors in a similar way to BZDs, except at a different binding site, because BZDs do not interfere with kava efficacy (White 2018). One recent study has suggested that a key to understanding kava's neurobiological effects is quantifying changes in GABA metabolites in the limbic region of the brain as a product of kava treatment (Savage et al., 2015, p10-11). Kavalactones also operate for ligand-gated chloride ion (Cl-) channels (Chua et al., 2016, pp. 1-2; Shi, Dong, Zhao, Tang, & Zhang, 2014, p. 289; White, 2018). Additionally, kava also inhibits the release of excitatory neurotransmitters – e.g.

Glutamate – via blockade of the calcium ion (Ca+) channels, therefore further facilitating transmission of GABA (Savage et al., 2015). Another suggested kava mechanism is modulation GABAergic networks via blockade of sodium (Na+) ion channels (LaPorte et al., 2011). One study which investigated the efficacy of kava as an anti-convulsant substance found that Kava (100mg/200mg) had an anticonvulsant effect in rats, although the mechanism is not well understood (Tawfiq et al., 2014, p.588). Kava modulation of GABA (A) receptors suggests a concentration-dependent interaction with GABA (A) receptors, a mechanism that is also suggested in other reviews and studies (Tawfiq et al., 2014, pp.591-2; Chow Chua et al., 2016, p.6; Singh 1983; p.274). Several studies have suggested that kavain is the major GABAergic component of kava, along with DHK (Chua et al., 2016; Çiçek, 2018). However, this may not properly account for other active ingredients (Lebot et al., 2014; Lebot et al., 2019), which are certain to impact the effect kava has on human patients.

The majority of neurobiological kava studies have used animal models to measure effects of kavalactones on different neurotransmitters, describing several mechanisms of action (Rowe et al., 2011, p.3; Shi et al., 2014, p.293); agonism of Acetylcholine (ACH) receptors via ligand binding (Kautu et al., 2017), and GABA (Kautu et al., 2017), blockade of Noradrenaline - which may account for kava's lack of side-effects (Showman et al., 2015; (Savage et al., 2015), ligand binding with cannabinoid receptors (Cock & Cheesman, 2016), monoamine oxidase B (MOA-B) – reversed inhibition (Showman et al., 2015), and blockade of Glutamate release (Schanzer, Rivas-Grajales, Khan, & Mathew, 2019). However, it is not clear how useful animal models are for drug research. Azra Raza has stated: "An obvious truth… is that mouse models don't mimic human disease well and are essentially worthless for drug development"(Raza, 2015, p. 201). Therefore, while kava studies using animal models have illuminated certain aspects of kava pharmacodynamics in human models (Cock & Cheesman, 2016; Kautu et al., 2017; White, 2018), there are always limits to animal models when they are used for drug discovery, as is the case with the study of kava (Wang et al., 2020; Wang et al., 2015). The problem is further compounded by the nature of the kava compound.

Kava is not a single compound or a tailored synthesised pharmaceutical, but a non-water-soluble conglomerate of compounds, suspended in water (Lebot et al., 1999). Each kavalactone – the major psychoactive constituents of kava (Segone et al., 2020, pp.1-2), has unique properties which could be analgesic, anxiolytic, hypnotic, or muscle relaxing in effect (Çiçek, 2018; Cock & Cheesman, 2016; Schanzer et al., 2019). The same could be said for any specific combination of compounds in kava – studies continue to isolate so-called 'psychoactive proponents' from kava rather than extracting the whole substance (Kautu et al., 2017; Wang et al., 2020). At this stage, evidence for kava mechanisms of action is incomplete (Kautu et al., 2017), and there may be interesting findings which illuminate the combined mechanism of action in future research. For example, in combination with

GABA network modulation, noradrenalin mechanisms may explain the feeling of relaxation with slight hedonistic tones, without the deleterious effects on cognition. It may be the varied mechanisms of action which may counterintuitively make the substance safer for consumers (Savage et al., 2015).

Conclusion

Researchers' current understanding of kava mechanisms is based on inferences and extrapolation from *in vivo* and *in vitro* animal models (Rowe et al., 2011, p.3; Shi et al., 2014, p.293; Kautu et al., 2017). Furthermore, majority of studies which have been conducted using animal models to describe kava mechanisms of action and interactions with separate neurotransmitters are not likely to reliably represent the effects of traditionally influenced kava studies (Cock & Cheesman, 2016; Kautu et al., 2017). Another issue with the current state of kava studies is that researchers have predominantly administered a tailor kava product which contains either a specific selection of kavalactones or a very low volume of powdered kava rootstock (<400mg) in the form of capsules (Aporosa et al., 2020; LaPorte et al., 2011; Sarris et al., 2013; White, 2018). Finally, the literature demonstrates that kava effects are still relatively unknown, especially regarding specific neurobiological mechanisms of action (Kautu et al., 2017). Therefore, while the next chapter discusses what is understood about kava effects on human body balance, research currently remains unclear which mechanisms of action influence kava's effects on human consumers in numerous domains (White, 2018), including body balance and stability.

Chapter 3

Kava and Balance

Introduction

The current chapter will address kava effects on human physiology with a focus on balance. Based on research, kava is assumed to cause increased body sway, instability, or ataxia. The discussion begins with a description of how kava intoxication has been described, and how it is was historically compared to opium and alcohol despite the effects being demonstrably different. However, in the current context kava has been compared with BZD drugs which have been shown to have negative side-effects. These are discussed in relation to comparisons to kava because the respective GABAergic mechanisms of action have been associated with each other, although evidence suggests that kava

does not cause any of the side-effects associated with BZD. Indeed, kava may be combined with diazepam to decrease the negative side-effects associated with the latter, although other authors have disputed this suggestion and mixing kava with other substances may not be advisable. Due to the recent interest in kava possible link to ataxia, there has been a repeated claim that kava does cause increased body sway, justified by citing Prescott et al., (1993), which found kava causes significantly increased body sway. This chapter investigates the kava literature with a focus on the claims made about kava inducing instability or body sway, to establish whether current evidence is reliable.

Historical Perspectives

Kava is a substance which has been known to cause a kind of all body effect; that has been variously described as doped, mateni (Lindstrom, 2004, pp. 12-13), or kona (Lynch, 1996, pp. 35-36) by different scholars and writers. Early commentators noted that kava has often been associated with alcohol (Turner, 1986, p. 204), and even opium (Lewin 1886, cited in Lebot et al., 1997, p.59). More recent research has dispelled these claims (Lebot et al., 1997, p.58-60). However, it is worth noting that BZDs such as oxazepam (Ox-Pam®), lorazepam (Ativan®), and alprazolam (Xanax®), have all been shown to cause instability in those patients taking them regardless of whether they are elderly, injured, or young and healthy (Dassanayake, Michie, Carter, & Jones, 2011; de Vries et al., 2013; Stanley et al., 2005). Joseph Ledoux has stated in an interview that BZDs affect the whole body, and their function is dull the nervous system's responses as part of the treatment of anxiety (LeDoux, 2019). It has been suggested that kava acts in a similar way to BZDs within the body and the central nervous system (Lebot, 1997; Sarris et al., 2011; White, 2018), as a GABA neuroreceptor agonist, increasing inhibitory actions within the nervous system. BZDs act as useful analogues for kava in the respect that they increase the transmission of GABA across the synaptic membrane, thus causing inhibition, though kava does not have the same binding site (Sarris et al., 2013, pp.643-644; White, 2018, p.1397). Although early comparisons to alcohol and opium were misguided, a reasonable conclusion is that kava will have a detrimental effect on balance and coordination due to inhibitory action, although there is a lack of evidence to support the claim.

There is no one study which focuses primarily on balance, body sway, or stability. If the history of anecdotes is considered, then the gap seems important, as past accounts of kava have often included comments on the consumers being in a stupor or heavily intoxicated (Lebot et al., 1997; Turner, 1986). Singh (2009, pp108-113) describes early missionary accounts of kava; they first tried to ban the consumption of kava, and then set about buying up and destroying kava plantations, to

impose Christianity and European social standards on people. The justification for these actions came in the form of concerns about people chewing kava roots, which missionaries said was unhygienic (p.109). Another justification was drawn from accounts given about people staggering home, falling over as if in an opium stupor, and being unsavoury due to intoxication. Excessive kava consumption was considered dangerous; akin to abuse of alcohol (Turner, 1986, p. 204) and opiates (Lewin 1886, cited in Lebot et al., 1997, p.59), even within recent literature, there is no understanding of what excessive consumption of kava is, and what levels of consumption of kava causes people to be a danger to themselves and others. Due to recent studies, we know that kava causes negligible harm and its effects have not caused any health issues, as has been stated previously (Bonomo et al., 2019; White, 2018). Past results concerning cognitive abilities have shown that kava improves certain aspects (Thompson et al., 2004). However, a rigorous understanding of how kava affects balance is still needed to account for a substance that is drunk routinely before driving, working, and playing professional sport (Aporosa et al., 2020; Aporosa, S., 2019; Tecun, 2017), as much of the data is incomplete (Kautu et al., 2017; LaPorte et al., 2011; Prescott et al., 1993) or based on anecdotal commentaries.

Neurobiological and Clinical Investigations

A study has investigated the muscle actions in frogs (Singh, 1983). The Sartorius muscles of a frog were administered with kava (2mg/ml) to investigate the effect of kava on Na+ channels within the muscle. The author found that kava affects the muscle action of mice, causing noticeable contraction and inhibition over 1 -1 ½ hour after kava was incubated within the muscle (Singh, 1983, p.273). These results seemed to confirm earlier studies in which kava extracts caused contractions in frog heart muscles (Meyer & May, Cited in Singh 1992, p.40). The studies inform the current research because they show that the inhibitory effects do indeed extend to the body, rather than being isolated within the CNS, which is still the primary focus of kava research (White, 2018). Questions have been raised in a recent paper about how much we understand about kava pharmacodynamics (Kautu et al., 2017). In an investigation into kava influence on acetylcholine transmission Kautu et al., (2017) showed that although we know little about kava within the human body, kava does seem to cause convulsions leading to partial paralysis at higher volumes at receptors operating for ACH in the NMJ of the Caenorhabditis elegans (Roundworm). Though these studies have been done on neuromuscular receptors within worms, the results are meaningful in the human context because ACH is also present in the human body and mediates muscular and motor actions within the body (Kautu et al., 2017; Kuo

& Ehrlich, 2015). The literature suggests there is a direct link between kava pharmacodynamics and muscular contraction, as multiple animal studies

Although these studies infer that NMJ in the human body may be impacted by kava, only Singh (1983) investigated human cases extensively. As his research shows, the extent of what we know about kava's effect on synaptic junctions in the body is that the twitch reaction is delayed after the consumption of kava. The main body of work relating to kava's effect on balance consists of a single study which considered balance, body sway, or instability as secondary subjects (Prescott et al., 1993). Elsewhere, kava has been linked to acquired ataxia, a form of coordination and motor control deficiency (Perez & Holmes, 2005), which some studies suggest is being mediated by the cerebellum (Nachbauer et al., 2015; Teive, Munhoz, & Werneck, 2012). For this reason, it is suggested that kava may cause inhibition in the cerebellum region, thereby reduced coordination in consumers (Perez & Holmes, 2005). These studies, although interesting, do not focus on kava as a traditionally influenced drink as described by (Lebot, 2018), but rather a potential pharmacological therapy (LaPorte et al., 2011; Perez & Holmes, 2005), or concoction of a limited number of kavalactones - < 400mg/day (LaPorte et al., 2011; Sarris et al., 2020; Sarris et al., 2012; White, 2018). Furthermore, these studies are discussing ataxia, which can elicit several symptoms, of which a lack of balance or increased body sway is only one, while others such as hand-eye coordination or decreased performance in acute motor tasks may be far more prevalent in any given person (Klockgether, 2010). Therefore, although kava may be linked to acquired ataxia (Klockgether, 2010; Perez & Holmes, 2005), it is not yet clear how much kava would affect a person's balance.

A case study published in the mid-2000s claimed that kava ingestion caused acute ataxia which influenced a patient's ability to complete motor tasks and stand properly (Perez and Holmes, 2005, p.49-50). It appears that the patient consumed a herbal tea infused with kava that was "too strong" (Perez and Holmes, 2005, p.50), and as a result was unable to stand after dinner shortly afterwards. None of the other people present had drunk the tea as well, and they were fine, so the tea appeared to be the toxic agent (p.50). The authors state that this is just an introductory paper, designed to warn Emergency Physicians of the possible harms of herbal medicines, and it is not a rigorous study of kava itself (Perez & Holmes, 2005). However, here are several issues with the study. Firstly, all the assumptions that the patient had taken nothing else were derived from self-report, which may be false if he had taken something illegal (Magura, 2010; Uvacsek et al., 2011). Second, we do not know what type of kava he had ingested, and though it could be a traditionally influenced aqueous mix, it is more likely to be a much more potent commercially extracted (using ethanol or acetone) version (Munsell 2019; Teschke et al., 2011; Wang et al., 2015), or even a version where a company has misused the plant meaning it is contaminated (Teschke & Wolff, 2011). It is not clear that kava has

caused the ataxia present in the Perez and Holmes (2005) study. However, the case is of some use as it shows kava may have some acute impacts on balance and coordination.

An early study attempted to quantify the amount which body sway increased after the consumption of kava (Prescott et al., 1993). The kava consumed is of interest to the current thesis; "200 gm of commercially available Fijian powdered kava root, held inside a permeable nylon sack, was infused into 1000 ml of water for 10 minutes" (p.50), because the authors appear to have used kava in its complete, traditionally influenced form – although the cultivar is not defined (Prescott et al., 1993, p. 50). This is the first study to address the link between naturalistic kava and balance in a quantitative setting. The researchers assigned their participants (11 male, 13 female) blindly to one of two conditions; kava drinking or non-kava drinkers. Kava drinking participants received 500ml of kava mixed with 500ml fruit juice (1000ml), while non- kava drinkers received 1000ml of fruit juice (p.50). It is unclear why the kava was mixed with fruit juice; however, it is worth noting that the sugar in the fruit juice may have augmented the results as kava is a mildly psychoactive substance (Ahmed, Guillem, & Vandaele, 2013; Pittler & Ernst, 2003). Two reasons for the mix are plausible. Either; the juice was mixed with kava to make the taste more appealing, or the juice was designed to mask the taste of kava in some way, though it is unlikely to have succeeded. The researchers analysed a range of measures; cognitive performance, physiological recordings (including body sway), mood, heart rate, respiration rate, and blood pressure (Prescott et al., 1993, pp50-51).

In their discussion, Prescott et al., (1993) describe their results with justified caution. Their experiments found no effects of kava on naïve kava drinkers in all the measures, excepting a body sway (p.52), which was shown to increase significantly ($t = 2.75$, $p = 0.016$). The methods used in the study are not ideal, as the tools used were a vertical metric scale attached to a bench. The researchers used a vertical pulley system that hung in line with the metric scale and was attached to the participant's collar. The pulley was also attached to a weight, and researchers observed the amount the weight moved from its position to measure the amount of body sway (p.51). An issue may be that the experiment did not measure the COP – postural sway (Miyata et al., 2015; Rodrigues et al., 2015), of participants, who may have shifted their weight in some way during the session. Therefore, although the authors do state that the pulley system has been used extensively and accurately measure upper body movement, there is no way of knowing if the resultant body sway results are entirely reliable. However, the instructions given to the participants were clear and replicable. Participants were instructed to "stand motionless with their feet together and eyes closed" (Prescott et al., 1993, p.51), which are instructions that have been used in more recent Force plate studies measuring COP (Miyata et al., 2015, p. 2132; Murray, Beaven, & Hébert-Losier, 2019, p. 342). The methods of the Prescott et al., (1993) study are questionable, and the volumes drunk are also

incomparable to this study (500ml vs. 3600ml); the volume could not be increased because three participants complained of nausea (p.52). However, the researcher did manage to gain statistically significant results. Participants, who are not regular kava drinkers, complained of being mildly intoxicated, though it is unclear what 'intoxication' means in this instance. The participants who were drinking kava showed a statistically significant increase in the extent of individual body sway ($t = 2.75$, $p = 0.016$), against the non-kava drinking group (p.52). A final point is that the body sway increase could be associated with nausea or mild discomfort experienced by some participants (p.52)

Outside of the previously mentioned studies, there are no studies that have addressed body sway directly in recent years. There have been a few systematic reviews in recent years, all from a clinical perspective, where balance or sway is not the major focus (LaPorte et al., 2011; Ooi et al., 2018; White, 2018). White (2018) focused their attention on the pharmacology, pharmacokinetics, and adverse effects. While they noted that kava has a broad safety profile, meaning long term harm is unlikely, the author also suggested that the clinical trials reviewed all operated with strict kava consumption limits. LaPorte et al. (2011) reviewed the clinical literature thoroughly, and though they too mostly focused on cognitive function and medical issues such as dermopathy and hepatotoxicity, the authors do make a brief mention about kava's effect on balance (p.107). However, though this is an updated paper, the authors only found any mention of increased body sway in the Prescott et al., (1993) study (LaPorte et al., 2011, p.107). Sarris, LaPorte and Schweitzer (2011) Also noted the Prescott et al. (1993) paper regarding body sway, and no other studies were mentioned. Another example of a clinical review comes from a book by Singh, Singh and Singh (2004, pp.140-159). The authors describe studies which found that dizziness is a side effect associated with kava consumption, though one study used a commercially available kava extract (Laitan®), so it is unclear whether the study, or any others that use limited volumes, or specific extracts, can be compared to the present study which will use a common noble traditionally influenced naturalistic beverage (Aporosa and Tomlinson 2014; Aporosa et al., 2020). The systematic reviews were also limited because of their restriction to clinical trials and did not extend their findings to other disciplines. Even so, the systematic reviews further demonstrate the gap in the kava body sway literature, and that researchers should investigate the issue further, as the study which found kava increased body sway (Prescott et al., 1993), implemented consumption levels which are much lower than the standard consumption rates in New Zealand and other parts of Oceania (Aporosa et al., 2020; Aporosa & Tomlinson, 2014).

One important study which addressed the psychopharmacodynamics of kava was conducted using the NMJ of Caenorhabditis Elegans, a worm which uses the same neurotransmitter as humans to mediate NMJ action (Kautu et al., 2017). ACH is an excitatory neurotransmitter which is associated with human muscle contraction (Kuo & Ehrlich, 2015). Human musculature only can pull and release

(Wilkinson, 2016). ACH is associated with pulling mechanisms, which is achieved through the process of release of ACH into the synaptic cleft, which eventually results in muscle contraction (Kuo & Ehrlich, 2015). Past studies have demonstrated kavalactone potency regarding interactions with GABA. Very few studies have discussed possible mechanisms for the action of kavalactones on NMJ, and ACH transmission and the modes of action are still not well understood (p.1). Kautu et al. (2017) showed that kavalactones increased the transmission of ACH, thus inducing muscle contractions. Kavalactone dose of 0 and 0.2mg/ml had no effects, while kavalactones had a progressive effect profile at concentrations of 0.4,.06, 0.8 and 1.0mg/ml. some of the worms exhibited anterior convulsions and repetitive muscular contractions, however, the effects seem to begin with convulsions and move to paralysis over time, or as the concentration was increased. In some cases, kavalactones caused instant paralysis, though this was prominent in only higher doses (pp.2-3). The authors also noted that while kavain didn't cause convulsions, muscle paralysis ensued in 62% of cases. Past studies have shown that worm NMJ exhibit convulsions and paralysis due to the increased transmission of ACH, as demonstrated by activating mutation in nicotinic ACR-2 receptor. The authors postulate in this study that kavalactones intensify ACH neurotransmission (p.3).

Further support for kavalactone enhancement of ACH is provided by an investigation into the sensitivity of worms treated with kavalactones to aldicarb, a reagent used to inhibit Acetylcholinesterase (ACHE) – the inhibitory counterpart to ACH. Through ACHE inhibition, aldicarb induces muscarinic paralysis via an increased concentration of ACH. Worms treated with kavalactones exhibited a significantly increased hypersensitivity to aldicarb when compared to the untreated control worms (p.3) For investigations into the effects of kavain alone, the authors used a 1.0mg/ml solution kavain, which was administered to the same Caenorhabditis elegans NMJ as the previous complete compliment of kavalactones (p.1-2). The result was unexpected, while they found that 0% of the 40 worms analysed experienced paralysis under the control condition, 1mg/ml of kavain induced paralysis in 62% of the worms analysed (pp.4-5). The authors recommend further research into kavalactone interactions with NMJ receptors because their results are not conclusive. Although the results demonstrate that ACH increase could cause paralysis (Kautu et al., 2017, p. 5), other research demonstrates kava's GABAergic properties (LaPorte et al., 2011), a factor which could mean that ACH is not the only neurotransmitter kava interacts with at the NMJ. Research needs to establish the precise interactions between kava, ACH, and GABA to establish a more reliable model for kava NMJ action.

Due to the GABAergic mechanisms of kava (Kautu et al., 2017), which may also be associated with kava cholinergic mechanisms, there is a possible explanation for the continuous reference to kava induced ataxia (Perez & Holmes, 2005), or increased body sway (Prescott et al., 1993). In one study

ataxia is the term used as synonymous with intoxication (Cawte, 1985), suggesting that the extent of kava effects may be extrapolated by how unsteady a kava drinker looks. As a result, it is important to establish whether kava is the causative variable which induces ataxia as suggested by one case study (Perez & Holmes, 2005); rather than fatigue, other substances, or 'dead legs'. Past researchers have suggested that kava does not cause the ataxia in European novices, rather the dense tobacco smoke present – perhaps synonymous with men sitting together, and having to sit cross-legged the whole time (Cawte, 1985). However, Turner (1986) suggests that it is likely that kava has some effect because it seems to cause ataxia even inexperienced Fijian kava drinkers. Kava's reverse tolerance index (Singh 2004b) would suggest that both cases might be true, novice Europeans may not feel as many effects as Fijian kava drinkers, and it is worth considering how influential the sitting position is for the apparent physiological effects of a kava session (Cawte 1985).

Fu et al., (2008) discussed the transient ataxia caused by kava, defined as an altered and uncertain gait when participants walk, although they administered an ethanol extract. Perez and Holmes (2005) found that kavalactones possibly induce ataxia. a study on Iowa drivers found that kava may have been associated with observed ataxias in three cases, however, the methodology of the study was questionable and the results are likely not reliable (Berry et al., 2019). Furthermore, Fajemiroye et al., (2016) warn that one of the common side effects for any treatment of anxiety and depression is increased instability, often defined as ataxia, which is associated with unsteady gait or a decrease in motor ability (Klockgether, 2010). The observations made about possible kava induced ataxia are not extensive (Fu, Korkmaz, Braet, Ngo, & Ramzan, 2008; Perez & Holmes, 2005), and the definition of ataxia is not reliable in this context (Klockgether, 2010). However, there is a certain amount of evidence within the kava literature which suggests future research into acquired ataxia will be worthwhile for kava research.

Conclusion

This chapter has reviewed literature which addresses kava's effect on human psychology and physiology, with a particular focus on kava's effect on balance. Overall, the literature does not support the claims that kava likely causes significant increases in body sway or instability. Furthermore, instability is not well defined and is an often falsely conflated with alcohol intoxication, or included in the umbrella definition of 'ataxia' which is unreliable due to symptomatic variability included in ataxic definitions. Another issue with the current body of evidence is that many of the accounts are anecdotal, or based on accounts collected retrospectively. However, clinical research into kava effects has tended to involve kava pills of capsules, which contain a specific cocktail of certain kava compounds, rather than the entire beverage. This means that the bulk of research conducted so far

cannot be generalised to the reality of everyday kava sessions in Aotearoa New Zealand and elsewhere, except for recent studies conducted by Aporosa (2017) and Aporosa et al., (2020). Indeed, the only previous study which found kava significantly increased body sway, was undermined by a flawed methodology and has not been replicated. Although the research does not support the claims that kava causes increases body sway or instability, there is no body of evidence that firmly disputes the claims made on the subject, and the weight of anecdotal evidence has influenced perceptions about kava. Therefore, the current study will attempt to investigate traditionally influenced kava's effect on human balance for the first time, to establish whether statistically significant results from past studies are reliable.

Chapter 4

Methodology

Introduction

This chapter will provide an overview of the methodological framework used in this study, which is based on The Post-Development Framework described by Aporosa (2014). It will also describe the data collection process and design. The participant recruitment process, eligibility and exclusions criteria will be described in detail. The chapter also discusses the process of obtaining informed consent, which is described in relation to the consent form provided (Appendix A). The researcher discusses the impact of being an outsider to kava culture, and how that shaped the research process. Finally, the testing instruments; an AMTI ACCUgait Force Plate and a Y-balance test are discussed, and the respective testing batteries associated with each test is explained. The chapter ends with an introduction to Chapter 4, which is a journal article, submitted to *Pacific Health Dialogue: The Journal of Pacific Research* as part of the current research project.

Methodology

The methodology for the current research is based on the Post-Development Pacific Research Framework described by (Aporosa, 2014). This framework was established to provide a Pacific centred approach to research which focused on maintaining the Pacific values of respect as central parts of any research project. Nabobo-Baba (2008) added further weight to the framework in her discussion of Vanua Research, where the concept of 'being the best person you can be' comes to the forefront for both the researcher and the participants. Cultural standards such as those proposed by the Post-Development Pacific Framework and Vanua Research have been shown to be more effective in Oceania contexts than other academic standards of research (Aporosa, 2014; Nabobo-Baba, 2008).

The methodology is appropriate for the research as it focuses on pacific influenced kava consumption. The literature also suggests that the 'gold standard' for clinical trials involves a placebo condition, however, this study does not include such a condition. The decision to forego a placebo control condition is based on recommendations made by past researchers. Aporosa and Tomlinson (2014) stated that cultural imperatives and restrictions would prevent a placebo being presented as 'real' kava beverage, while more recent studies have discussed further problems with using 'gold standard' placebo conditions for kava research; kava consumption causes the mouth to tingle and

over a 6-hour kava session an experienced kava drinker would question its absence (Aporosa et al., 2020). Furthermore, kava is not a consistent compound, and it is routinely mixed to preferred consistency (Aporosa, 2014). A placebo condition which mimicked the process of kava consumption, including regularly mixing the beverage to the preferred consistency would involve unacceptable deception in a traditionally influenced kava consumption setting (Aporosa & Tomlinson, 2014; Tecun, 2017).

The current methodology is also based on past clinical trials and experimental investigations involving kava. The study presented in Chapter 5 is an updated investigation of kava's effect on body sway with a modified methodology, based on a previous study (Prescott et al., 1993). The original study conducted using novice kava consumers (n=12) obtained results which showed significantly increased body sway caused by kava consumption (Prescott et al., 1993). The study has not been repeated, and this study attempts to update the results of that have not been supported by more recent data assessing kava effects in clinical and cognitive domains (LaPorte et al., 2011; Pedrosa, Bezerra, da Costa, Pinheiro, & Guzen, 2020). This study will implement two novel assessment tools for kava research; an AMTI ACCUgait force plate and a Y-balance test (Shaffer et al., 2013), to determine the effects of kava on human body balance during and after a traditionally influenced 6-hour kava session.

Outsider Research

The researcher or this project is palagi, defined as someone who does not identify as part of an Oceanic ethnicity (Seiuli, 2016, pp. 203-204; Teaiwa, 2007). The Pacific Post-Development Framework prioritised respect for culture and relationships (Aporosa, 2014), and on that basis, the current research can be conducted because the kava community has invited the researcher to do so. In a project such as this one, the researcher can only bring so much knowledge to the data collection session (De la Cadena, 2015). They must then accept that kava is an object of culture which the participants are more knowledgeable about, a ubiquitous part of their culture which the researcher and their equipment would risk trespassing on if they did not have the groups permission to conduct the research (De la Cadena, 2015; Ratuva, 2009; Tecun, 2017; Tversky, 2019). Separating the testing area from the drink areas by a partition wall was also a method of ensuring that the 'outsider' equipment was used to measure specific physiological indicators while remaining in the general vicinity of the kava session while interfering with it as little as possible.

Participants

The participants (n=6) consist of regular kava drinkers who consume kava at least twice a week. As regular and experienced drinkers, they are regular attendees at kava sessions of six hours and longer, familiar with sitting for extended periods at lengthy kava sessions. The original number of participants obtained for the study was 12, based on a previous study in which kava caused a significant increase in participant body sway (Prescott et al., 1993). However, due to the COVID-19 crisis, the number of participants who confirmed their involvement was reduced to 6. The group will largely comprise of Pacific Island peoples, however, due to the increasing number of Maori and Pakeha kava drinkers, experienced users from these ethnicities are also included. All participants are over the age of 18. All participants of the current study are male, which is due to most kava drinkers being of the male gender (Balick & Cox, 1996). All participants will be given an information sheet which describes the procedure and informs participants that they can withdraw from the data collection at any point during the kava session (Appendix C). Participants who volunteer for the data collection will be required to give informed consent by completing the consent attached consent form (Appendix A), and the researcher has provided an eligibility screening form which will be completed for each participant (Appendix B). Eligibility screening is essential because the presence of existing mental illnesses, physical disabilities, and concurrent prescriptions of pharmaceutical drugs will mean that the safety of participants may be compromised and that the results will be unreliable (Sarris et al., 2013).

Setting

The data collection for this research will take place during a traditionally influenced kava use session (Figure 4), or kalapu (Tecun, 2017). The venue will be a seminar room at the University of Waikato, which is arranged according to kava consumption practices which are common in Aotearoa, New Zealand During such a session, participants typically sit on the floor, cross-legged, on a woven mat, while the powdered kava is strained through water and mixed in the kava bowl (Aporosa et al., 2020). The resulting liquid is a light-brown coffee-like colour (Aporosa, S. A., 2019). Typically, the strength is established visually, based on the experience of those mixing the kava. The kava is served in 'bilo' (cups made from half coconut shells)

Figure 4 A example of a traditionally influenced kava session in Aotearoa New Zealand (Photographer: Todd Henry, 2018).

and served at regular intervals. The average duration of a kava session is 6 hours, in which time, drinkers will typically consume 6 x 100ml bilo each hour (Aporosa, 2019. pp 22-23).

Equipment

A Force Plate (AMTI ACCUGait Optimized) will be used during the data collection testing batteries to ascertain COP data for later analysis using Balance Clinic. A Y-Balance Test was also used as a secondary measure for dynamic lower-limb balance (Hoch, Welsch, Hartley, Powden, & Hoch, 2017; Shaffer et al., 2013). Both instruments belong to the University of Waikato. Neither of these instruments has been used to assess kava effects in previous studies. However, there are several studies which implement force plate tests to assess the effects of BZD on balance and stability in humans (Miyata et al., 2015). Therefore, because BZD are kava's closest pharmaceutical analogue, force plate tests were used in the current study. The Y-balance test has also been used extensively to assess human balance (Hoch et al., 2017), and is used in this study as a secondary measure of human body balance.

Kava

Dried powdered kava root/basal stump was purchased from a popular retailer in Hamilton, New Zealand. Before testing (approximately 1 hour), 36L of kava was mixed to the average potency consumed at a kalapu in New Zealand (Aporosa et al., 2020). A sample of this dried kava powder was analysed by The Institute of Environmental Science and Research (ESR), New Zealand's Crown Research Institute. The kava was found to contain no adulterants, with a strength rating of 5% total kavalactones by dry weight, a chemotype of 245163, and a mean kavalactone content of 115mg per 100ml of kava beverage. The chemotype for the kava used in this study demonstrates the characteristic of noble kava, as Kavain (4) and Dihydrokavain (2) are present in the highest volumes. These cultivars are well-tolerated and safe for regular consumption (Lebot et al., 2014; Lebot et al., 1997; Teschke & Lebot, 2011).

Procedure

The research will take place during a 6-hour traditionally influenced kava session. The session's duration will be six hours and held in a lab at Te Huataki Waiora School of Health. The participants will sit and drink kava throughout that time, though chairs or cushions can be provided for comfort if necessary. The participants will be served six bilo of kava every hour for the duration of the session (1 bilo = 100ml). At three points during the kava session, the participants will under-go 3x COP testing using a force plate (closed eyes, closed stance condition), and anterior y-balance testing, 3 x right leg then 3 x left leg. Testing will occur immediately before the kava session (0-hour), at the mid-point (3-hours) and immediately after the kava session (6-hours). The participants will be thanked and debriefed once all the participants have finished their testing and each participant will be given a meal. The researcher will ensure that every participant can travel home safely.

Centre of Pressure Test

Postural balance was assessed using an AMTI AccuGait optimised force plate, sampling at 150Hz, and Balance Clinic software v.2.03.00 (both developed by Advanced Mechanical Technology Incorporated).

Participants stood barefoot in the middle of the force plate, with their feet together, arms by their side, and head facing directly forwards, in a closed eye, closed stance condition. Three 30 second trials recorded four COP parameters for each participant: $COP^{\text{path length}}$, $COP^{\text{area 95\% ellipse}}$, $COP^{\text{AVG Velocity}}$, and COP^{Range}.

The trial length was chosen as a result of past research which demonstrated its reliability (Murray et al., 2019; Ruhe, Fejer, & Walker, 2010). Using a similar postural balance test, (Bauer, Gröger, Rupprecht, & Gaßmann, 2008) found these measures had acceptable reliability, with corresponding intraclass correlation coefficient (ICC) values of 0.945 and 0.710.

The researcher ensured that participants felt comfortable enough to complete each trial. All participants completed the full force plate testing. An example of the COP data plotted during the trials is shown in Figure 3.

Y-balance test

An abbreviated y-balance test will be implemented, where only the anterior condition was measured for each foot (Shaffer et al., 2013). Trials were repeated three times for each leg. The right leg was tested, followed by the left leg. The process was repeated at each testing cycle: before, in the middle and after the kava session.

Data Analysis

The force plate data will be exported and analysed using Balance Clinic software v.2.03.00, which will measure changes in the mean scores and standard deviations between the pre-, mid- and post-tests.

Y-balance test results will be analysed as percentages of leg length. Changes between the pre-test mean and post-test mean scores were measured.

Hypothesis

Traditionally influenced kava consumption will cause an increase in COP test scores and a decrease in y-balance test scores.

Ethics Approval

This thesis and research project obtained ethical approval from the University of Waikato (New Zealand) Human Research Ethics Committee (HREC [Health] #34). See Appendix D.

Conclusion

This chapter has outlined the methodological aspects of the research. The framework of the research project is based on The Pacific Post-Development Framework established in past studies *(Aporosa, 2014).* This discussion also acknowledged past research which used traditionally influenced kava consumption volumes (Aporosa et al., 2020; Aporosa, 2017). The research instruments were introduced and included kava – the chemotype and chemical analysis was outlined, the setting – including a discussion surrounding appropriate kava drinking venues, measurement instruments – an AMTI ACCUgait force plate (Murray et al., 2019) and y-balance test (Shaffer et al., 2013). The testing procedure was explained, as well as the study hypothesis: traditionally influenced kava consumption

will cause an increase in COP test scores and a decrease in y-balance test scores. Finally, the ethics approval for the research project was presented. The following chapter is a journal article which has been submitted to the *Pacific Health Dialogue: The Journal of Pacific Research and* includes the discussion and conclusion of the results that will be obtained during the data collection session.

Chapter 4

Journal Article

Traditional kava use and body sway: Preliminary findings

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Keywords: psychopharmacology, kava, body sway, balance, naturalistic test setting

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TRADITIONAL KAVA USE AND BODY SWAY: PRELIMINARY FINDINGS

ABSTRACT

Introduction

The (traditionally influenced) consumption of kava (*Piper methysticum)* has been associated with increased body sway, leading to a greater risk of falls and incidents while driving. However, systematic studies involving centre of pressure (COP) and anterior y-balance tests had not previously been conducted with experienced kava drinkers. This preliminary study investigated the effects of naturalistic kava consumption over a 6-hour period on body sway and lower-limb dynamic balance but did not reveal any significant impairment.

Methods

It was hypothesised that naturalistic kava consumption would cause an increase in COP test scores, and a decrease in y-balance test scores, indicating postural instability. Data collection took place over a 6-hour kava session, where 100ml of kava was served to participants every 10 minutes.

Pre-test (baseline), mid-test and post-test measures were taken, consisting of:

- COP tests – three 30 second tests with eyes closed, and a closed stance test
- abbreviated y-balance test – three anterior direction tests.

The tests were conducted with six experienced kava drinkers in a culturally appropriate setting. The data was supplemented by quantitative and qualitative observations.

Results

There were no significant increases in the COP parameters (COP $^{\text{path length}}$, COP $^{\text{area95\% ellipse}}$, COP AVG$^{\text{Velocity}}$, and COP$^{\text{Range}}$) between the pre-tests and post-tests. A slight increase in the Area$^{95\%}$ ellipse parameter was observed, but the change was not statistically significant. Similarly, y-balance test scores were not significantly affected.

Discussion

The results of this study did not support the hypothesis that kava consumption increases COP test scores and decreases y-balance test scores. This contrasts with previous research, which found that kava consumption significantly increased body sway. This suggests the perceived risks of naturalistic kava consumption may be overstated and further study on the physiological implications of kava consumption to determine the associated risks is needed.

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INTRODUCTION

This study investigates a possible link between kava consumption and increased risks of falls or unsteady gait, based on research which has found kava increases body sway.[1]

Dried kava plant (*Piper methysticum*) roots are ground into a powder and mixed in water to make the kava beverage.[2,3,4] Kava is a Pacific ceremonial, social and medicinal beverage, and is regularly consumed without any associated health risks or side-effects.[5,6] However, research suggests that kava may impair human balance.[1,7]

The current study measured body sway in kava users, using centre of pressure (COP) tests; and lower-limb dynamic balance, using an instrument new to kava research (namely an AMTI ACCUGait force plate, developed by Advanced Mechanical Technology Incorporated) and an abbreviated y-balance test.[8]

Participants attended a 6-hour kava session in a naturalistic (traditionally and culturally influenced) consumption setting.[9,3] The design of the study focused on kava as a beverage,[10,11] rather than a pill or capsule,[12] with the beverage consumed at high volumes to replicate the conditions of a typical Pacific influenced kava session.[13] Participants underwent a battery of tests before, during and after the session.

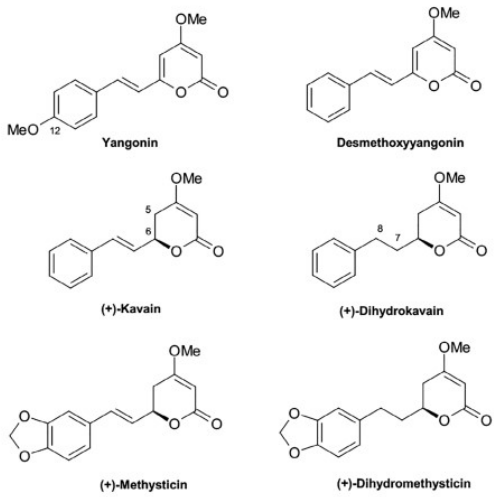

Figure 1 The structure of the six principle kavalactones.[3]

The results showed a lack of change in the participants' balance, between their baseline pre-tests and post-tests.

This study, supporting a Master of Health, Sport and Human Performance at the University of Waikato, New Zealand, was initially designed to test 12 participants, based on a previous study which investigated kava's effects on balance.[1] Unfortunately, the data collection phase of the study coincided with the COVID-19 lockdown in New Zealand, reducing participant numbers to six. Therefore, this paper presents a preliminary study aimed at providing a pathway forward for future researchers investigating the effects of kava using biomechanical measurements.

KAVA EFFECTS

Kava is a compound containing various active ingredients, including kavalactones, flavokavains and alkaloids.[11] Six kavalactones account for upwards of 90 per cent of kava's psychoactive properties: 1. dimethoxy-yangonin (DMY,), 2. dihydrokavain (DHK), 3. yangonin (YAN), 4. kavain (KAV), 5. dihydromethysticin (DHM) and 6. methysticin (METH). Their chemical structure is shown in Figure 1.

However, kava effects are also influenced by other chemicals present. Kava's various effects – anxiolytic, hypnotic, analgesic and soporific – have been associated with numerous mechanisms:

- blockade of ca+ ion channels[12]
- GABA potentiation via ligand binding to GABA(A) R receptors – similar to benzodiazepines, though via separate binding sites[14]
- inhibited reuptake of noradrenaline[12,14]
- reversal of monoamine oxidase B (MAO-B) inhibition[15,16]

However, kava's effects are not well understood, and past assumptions have been inferred from limited clinical data, *in vitro* and *in vivo* experimental assays, and historical and anecdotal assumptions.[7] Recent clinical studies have demonstrated the anxiolytic efficacy of kava,[10] when administered at pharmacologically recommended doses, although the effect has not been significant. Pharmacological researchers have suggested kava may be a non-addictive alternative to benzodiazepines, without exhibiting severe side-effects.[17] One study also suggests that kava may be used alongside diazepam, as a way of limiting adverse side effects. Clinical trials have investigated the medicinal value of pharmacological kava dosages.[10,]

A further complication in kava research is the use of pills or capsules versus naturalistic kava. Naturalistic kava is often consumed at volumes up to 30 times the recommended pharmacological dose.[3] Naturalistic kava sessions consist of drinkers sitting cross-legged on the floor, often consuming numerous cups made from half coconut shells (bilo) of kava over several hours, in settings known by various names, such as faikava, kalapu or 'grog sessions'; as shown in Figure 2. As a result, studies conducted on low volumes of naturalistic kava, or tailored cocktails of kavalactones in pill form, do not provide reliable representations of the effects of the kava beverage.

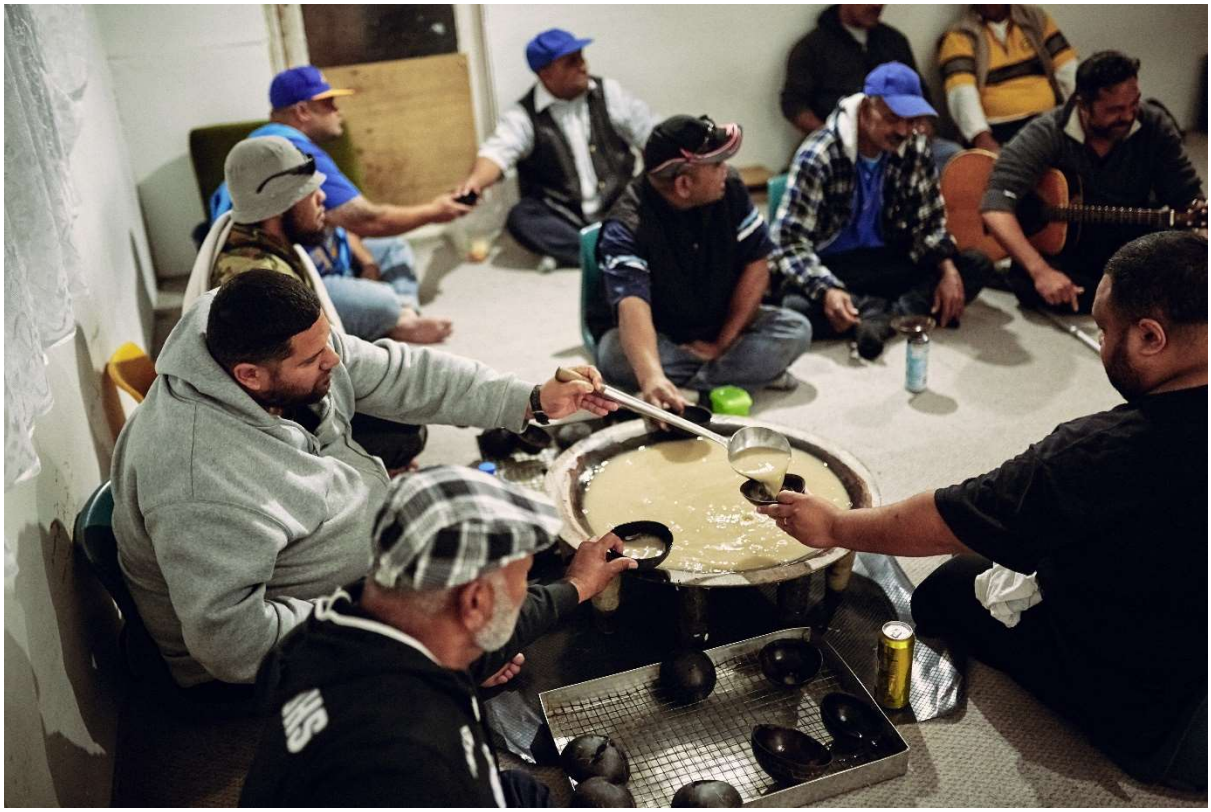

Figure 2: Naturalistic (traditionally influenced) kava session in New Zealand (Photographer: Todd Henry, 2018)

Cognitive assessments of kava users have determined that kava does not have a significant negative effect on cognition[3]., Another study suggests that kava's modulation of noradrenaline may mean it can reduce deleterious cognitive side-effects associated with benzodiazepines.[2] Research also reports the potentiation of kava when used with alcohol and opioids., Kava 'intoxication' is characterised by mild sedation, a slightly hedonistic relaxed feeling and mild somnolence[6].

Suggestions of kava induced imbalance persist, particularly in ethnographic commentaries,[18] with researchers suggesting it may be caused by sitting cross-legged for long periods, GABAergic mechanisms,[1] or cholinergic mechanisms associated with neuromuscular junctions.[19] Prescott et al.[1] did record significant body sway increases ($t=2.75$, $p=0.016$), although the kava used was heavily diluted and mixed with orange juice, with some participants complaining of nausea and declining to consume the recommended study volume, making the results inconclusive (pp.50-52).[1] More concerning is a report about the death of a man who, after consuming kava together with alcohol, fell into a wall which subsequently collapsed on him.[20] Regardless that the deceased instability

may have resulted from an alcohol kava cocktail, the report tends to focus on, and therefore infer, kava use as the dominant factor leading to imbalance, fall and death.

Despite this, kava's effects of human balance, including postural sway and lower limb dynamic balance, are relatively unknown. This study aimed to investigate the veracity of persisting suggestions that kava may increase body sway or general instability in human participants, as there is a lack of empirical data supporting the claim.

METHOD

Ethics

The current study obtained ethical approval from the University of Waikato (New Zealand) Human Research Ethics Committee (HREC [Health] #34) and was completed as part of the requirements for a master's degree.

Hypothesis

Naturalistic kava consumption would cause an increase in COP test scores and a decrease in y-balance test scores.

Participants

Participants were recruited through word of mouth and social networks (n=6). All participants completed an eligibility form and provided informed consent. Participants were told that they were free to withdraw from data collection at any stage. All participants were aged 18 or over, were of Pacific Island descent and were male. Exclusion criteria included inexperience of drinking kava; current or recent neurological, psychological, or physiological conditions; current psychoactive prescribed medications; diagnosed psychotic disorders; and conditions that may affect the participant's ability to complete balance testing, e.g. vertigo, a history of falls. The researcher requested that participants did not drink kava for four days before the data collection to provide a wash-out period.[3]

Materials

Kava

Dried powdered kava root/basal stump was purchased from a popular retailer in Hamilton, New Zealand. A sample of this dried kava powder was analysed by The Institute of Environmental Science and Research (ESR), New Zealand's Crown Research Institute. The kava was found to contain no adulterants, with a strength rating of 5% total kavalactones by dry weight, a chemotype of 245163, and a mean kavalactone content of 115mg per 100ml of kava beverage.

Prior to testing (approximately 1 hour), 36L of kava was mixed to the average potency consumed at a kalapu in New Zealand.[3,9]

Procedure

The data was collected in a seminar room on the University of Waikato campus, over a single 6-hour kava session.

Testing was conducted at three intervals: pre-test (individual baseline testing), mid-test and post-test. Pre-testing was conducted as each participant arrived, at which point the researcher explained the testing procedure to the participant.

Kava was served in bilo's at volumes of 100ml at intervals of 10 minutes from a kumete (kava bowl) to the participants who sat cross-legged on the floor on woven mates. In total, each participant drank 3.6L (or 7.61 pints) of kava. Based on ESR kava analysis, each participant consumed 4,140mg of kavalactones over the six-hour test period. Participants could leave the kalapu area to stretch their legs or use the toilet. However, they were required to be present at each drinking and testing interval. Snacks and water were provided for the participants, as would be expected at a typical kalapu elsewhere. The snacks did not contain sugar or caffeine.

Once the final post-testing was completed, participants were supplied with a meal and the researcher ensured each participant was able to travel home safely.

Centre of pressure test

Postural balance was assessed using an AMTI AccuGait optimised force plate, sampling at 150Hz, and Balance Clinic software v.2.03.00 (both developed by Advanced Mechanical Technology Incorporated).

Figure 3 AMTi Force Plate. (https://amti.biz/optima.aspx)

Participants stood barefoot in the middle of the force plate, with their feet together, arms by their side, and head facing directly forwards, with their eyes closed. Three 30 second trials recorded four COP parameters for each participant: COP $_{\text{path length}}$, COP $_{\text{area 95\% ellipse}}$, COP $_{\text{AVG Velocity}}$, and COP$_{\text{Range}}$.

The trial length was chosen as a result of past research which demonstrated its reliability.[21] Using a similar postural balance test, Bauer et al. (2008)[22] found these measures had acceptable reliability, with corresponding intraclass correlation coefficient (ICC) values of 0.945 and 0.710.

The researcher ensured that participants felt comfortable enough to complete each trial. All participants completed the full force plate testing. An example of the COP data plotted during the trials is shown in Figure 3.

Y-balance test

An abbreviated y-balance test was implemented, where only the anterior condition was measured for each foot.[8] Trials were repeated three times for each leg. The right leg was tested, followed by the left leg. The process was repeated at each testing cycle: before, in the middle and after the kava session.

Figure 4 A example of the anterior condition of the y-Balance Test (https://www.functionalmovement.com/)

Data analysis

The force plate data was exported and analysed using Balance Clinic Software v.2.03.00. Changes in the mean scores and standard deviations between the pre-, mid- and post-tests were measured.

Y-balance tests were analysed as percentages of leg length. Changes between the pre-test mean and post-test mean scores were measured.

RESULTS AND DISCUSSION

Centre of Pressure

The postural sway analysis was inconclusive, as there were no significant changes between the pre-test individual baseline and the post-test data collection results, for any of the chosen measures: $COP^{\text{path length}}$, $COP^{\text{area 95\% ellipse}}$, $COP^{\text{AVG Velocity}}$, or COP^{Range}. These measures were chosen as they had been demonstrated to be reliable in past studies.[22]

There was only one measure which changed during testing: area 95% ellipse. This parameter had pre-test and post-test means of 11.94cm^2 and 16.113cm^2, respectively.

It is suggested that either the force plate lacked sensitivity to the capturing of kava effects, or kava may have no major effect on body sway, despite past research claiming otherwise.[1,17] Further, the low participant numbers are acknowledged. The original number of participants (n=12) was chosen based on a previous study.[1] As previously noted, the number of available participants was heavily constrained by the outbreak of COVID-19.

This preliminary study investigated the effects of kava, drunk at naturalistic volumes (>3,000mg/kavalactone)[3], on human body sway and lower limb balance, during and immediately after a traditionally influenced kava session. The study incorporated novel experimental techniques regarding kava research: namely, force plate and y-balance tests as quantitative measures.[22,8]. There were no conclusive results in either testing condition, despite observations of the slowed gait of participants. There was a small increase in the $COP^{\text{area95\% ellipse}}$ score. However, the effect was not significant.

The study was initially designed for a minimum of 15 participants to ensure statistical significance. With fewer than half the original participants, due to circumstances imposed by the COVID-19 lockdown, this created an obvious study limitation.

The literature suggests the absence of a placebo condition may limit study sensitivity. However, Aporosa and Tomlinson[23] and Aporosa et al.[3] argue that placebo conditions are not suitable for kava research conducted in naturalistic settings. This is because of kava's spiritual and ceremonial significance which prevents deception associated with placebo.[24,13] Additionally, the taste and effects of kava are unique and well known to experienced kava drinkers, further limiting the use of placebo.[3]

Simple and straight-forward instructions recommended in earlier research were used in the study.[22] Despite this, participants approached the force plate testing in various ways. One participant saw it as a competition, while other participants appeared to be nonchalant.[25] Variations in behaviour during testing could be mediated by varying the order of tests throughout the session. However, to obtain a result based on the individual participants' baseline scores, repetition is necessary. Therefore, possible reactions to that repetition from the participants cannot be avoided.[25]

Y-balance test

The raw data from the abbreviated y-balance test was converted to percentages of leg length. There were no statistical significant differences between individuals or the entire group score in the pre-test (right-leg mean = 0.60, standard deviation = 0.16; left-leg mean = 0.59, standard deviation = 0.04) and the post-test (right-leg mean = 0.62, standard deviation = 0.10; left-leg mean = 0.61, standard deviation = 0.09).

Past studies suggested kava increased body sway,[1,7] and one study observed falls induced by kava.[20] However, in this study, there is no evidence of kava altering lower limb dynamic balance, either positively or negatively.

It is possible, that naturalistic aqueous kava does not affect lower limb dynamic balance. Other possible explanations for the lack of change in dynamic lower limb balance are:

- the measures were not sensitive enough to the effects of kava, due perhaps to the fact that the y-balance test is a novel assessment tool when applied to the effects of kava consumption
- although straight-forward instructions were given, it is possible those instructions resulted in the task not being strenuous enough to produce significant results
- although evidence of muscle relaxant and sedative effects have been found during *in vitro* and *in vivo* studies,[19] the effects may not be apparent during naturalistic kava consumption
- the data collection session did not have enough participants.

Observations

All six participants completed the kava session. The researcher observed a mild slowness in gait in some of the participants towards the latter part of the 6-hour session. One participant suggested his mild feeling of unsteadiness was possibly caused by 'dead legs', as a result of sitting cross-legged for several hours. There were no observations of slowed or slurred speech, which is inconsistent with results from past research

CONCLUSION

This preliminary study into the effects of kava on balance, based on data obtained from regular kava drinkers during and after a 6-hour naturalistic kava session, has not produced results that support the proposed hypothesis.

Future researchers can use this study as a marker for how more controlled studies might be achieved. Future researchers are encouraged to investigate the interactions between kava and human body balance, as past research and anecdotal evidence has pointed to possible connections.[1,7,17]

The current study has not undermined past claims of increased body sway and unsteadiness. However, the lack of alteration between the pre- and post-test data does suggest a mild kava effect.[26,27] The results also imply support for comments that kava is a safe substance that exhibits minimal side-effects.[16,28] More research is needed to better understand the effects of naturalistic kava on human consumers and particularly on stability and balance.

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Chapter Five

Discussion and Conclusion

Introduction

This chapter discusses the results of the paper presented in Chapter 4 in relation to the earlier literature review and methodology chapters. The paper has been prepared for publication in the *Pacific Health Dialogue: The Journal of Pacific Research*. The following discussion will address the study of (Prescott et al., 1993), as this current research was an attempt to repeated the aforementioned study with a few methodological differences. This chapter will discuss the results of the study presented in Chapter 4. The current research's hypothesis that kava increases body sway and decreases lower limb dynamic balance was not supported because there was no statistically significant change in any of the four COP parameters, or in Y-balance test scores during and after the data collection session. Furthermore, the discussion involves an analysis of the possible limitations which affected the data collection process while explaining the methodological decisions made in this research project. Finally, the thesis ends with a conclusion which reflects on this discussion section in relation to the entire thesis and makes some comments regarding possible future avenues for kava research.

Observations

During the experimental period of this study, the researcher has made numerous observations. Firstly, the kalapu sets the tone for the afternoon or evening of kava drinking (Aporosa et al., 2020; Tecun, 2017). There is an important element of the kava session which is missing in kava bars (Figure 5) where people sit on chairs or stand at the bar (Christian, 2019). In a kava session, participants generally sit with crossed legs on woven mats on the floor (Aporosa et al., 2020; Tecun, 2017; Turner, 1986). One of the participants in the study (Chapter 4) commented that they felt their legs were dead, which was likely caused by sitting cross-legged for many hours. It may be possible that the effect would not have been felt if they had

Figure 5 Four Shells Kava Bar in Auckland, New Zealand (Photographer: Todd Henry, 2020), the location described by Christian (2019).

consumed kava on the stools or chairs available at kava bars (Christian, 2019). One author suggests that ataxias demonstrated in kalapu are accounted for by tobacco smoke inhalation rather than kava effect (Cawte, 1985). Although it is still possible and somewhat likely that kava affects human body balance (Prescott et al., 1993), there is also research which suggests that the kava effect is indeed mild and may not be accountable for observable effects (Pittler & Ernst 2003). Overall, the researcher did not observe any significant differences in kava drinkers throughout the data collection process. Therefore, research which suggests that kava has mild or negligible effects has been somewhat supported by researcher observations in the current study (Cawte, 1985; Pittler & Ernst, 2003; Thompson et al., 2004).

However, the cross-legged position may not be insignificant (Cawte, 1985). It is possible that the cross-legged position does not undermine the effects of kava as stated by Cawte (1985), but compounds the effects as physical manifestations of sedated limbs. By extension, crossed may cause genuine increases in body sway supplemented by the psychopharmacological effects of kava (Perez & Holmes, 2005; Prescott et al., 1993; Turner, 1986), which are thought to be GABAergic, as stated by numerous studies and reviews which were described in Chapter 2 and Chapter 3 (Prescott et al., 1993; White 2018; Turner 1986: Singh 2004a). However, the current study did not support earlier suggestions that kava causes increased instability, body sway, or induces acquired ataxias (LaPorte et al., 2011; Perez & Holmes, 2005; Prescott et al., 1993). There was no statistically significant change in the Force Plate parameters or the Y-Balance Test scores during or after the kava session. As a consequence of supporting the null hypothesis, the study presents a requirement for future research to establish the link between kava and increased body sway suggested in the literature (LaPorte et al., 2011; Perez & Holmes, 2005; Prescott et al., 1993), which remains unsupported.

During Chapter 2 and Chapter 3, this thesis described how Kava psychopharmacodynamics is not well understood (Kautu et al., 2017). Kava effects have only thus far been inferred from *in vivo* and *in vitro* studies or clinical trials involving low doses, 120mg -480mg kavalactones per day, which is in line with pharmacologic recommendations (LaPorte et al., 2011; Sarris et al., 2020; White 2018). As a result, the exact mechanism of action which might induce ataxia or increased body sway can only be speculated upon. Research suggests possible mechanisms of action are potentiation of GABA or ACH via ligand binding to the post-synaptic receptor (Kautu et al., 2017; Singh, 2004). Alternatively, it is likely to involve kava compounds other than kavain, as well as other neurotransmitters – perhaps dopamine (Tith & Lalwani, 2013), the effects of which are not as well understood. Furthermore, Prescott et al. (1993) assumed that the increase of body sway was associated with novice kava drinkers, some of which experienced nausea throughout the experimental process, which is likely one reason the body sway increase was significant ($t = 2.75$, $p = 0.016$).

However, Singh (2004a) has outlined how kava has a reverse tolerance index, which means that although kava is psychoactive, unlike other substances such as benzodiazepines and opiates (Nutt, 2012), consumers do not become addicted because they do not more tolerant to kava (Singh, 2004).

The implication of kava reverse tolerance for this study is that kava effects are likely to remain equivalent in novice drinkers and experienced kava drinkers (Singh 2004a), thereby negating possible differences between result caused by the previous study's use of novice consumers (Prescott et al., 1993). Concerning dead legs from sitting cross-legged which was observed during the current research project, the reverse tolerance of kava means that Turner's account is likely to have some veracity. Turner (1986) noted that ataxia induced by kava was likely to still be a factor due to experienced Fijian kava drinkers exhibiting the same instability demonstrated by palagi consumers – likely novices such as the participants in the study by (Prescott et al., 1993). The nature of this effect would be derived from the reverse tolerance of kava (Singh 2004a; Singh 1983; Prescott et al., 1993), but also possibly from the dead-legs participants experience due to the traditionally influenced cross-legged sitting position. The cross-legged sitting position may not be one of kava psychoactive effects, but its possible brief effect after kava consumption should not be completely ignored, as it remains part of the kava 'set and setting', due to the way naturalistic kalapu are usually prepared so the appropriate behaviour of drinkers is to sit on the woven mat with their legs crossed (Tecun 2017; Nutt 2012; Hari 2015). Nevertheless, experienced kava drinkers are more likely to become accustomed to the effects, as is apparent with most psychoactive substances, which may result in the appearance of being able to compensate for the increased instability they feel (Aporosa et al., 2020; Mets, Volkerts, Olivier, & Verster, 2010; Nutt, 2020). However, such compensation is not likely based on previous accounts of kava effects, in which experienced kava drinkers are described as experiencing the same effects as novice consumers (Singh, 2004; Turner, 1986)

Kava and Human Body Balance

The kava body sway literature remains incomplete. The one study conducted specifically to test kava body sway effects found a significant body sway increase ($t = 2.75$, $p = 0.016$), which is intriguing because it fits with a previous commentator's perspectives on kava (Prescott et al., 1993; Turner 1986). A further study has described an event where a man fell into a wall, which fell on him and killed him (Ketola et al., 2015; Tarbah et al., 2013). Ketola et al. (2015) described anecdotal evidence for the fact kava could be lethal, even though the victim injected multiple substances intravenously at the same time, of which kava was the least harmful substance, and therefore not the principal cause of harm (Hart, 2015; Ketola et al., 2015). Even so, the authors felt it was appropriate to imply that kava

effects are potent enough to be significantly harmful by alluding to the incident with the wall described in Tarbah et al. (2013), despite it being an anecdotal account which has never been observed subsequently. The Ketola et al. (2015) account has not been of much interest for this thesis as it is unscientific in its methodology and accuracy, but it serves to expand on the issue of kava intoxication and the often implied extent to which kava affects psychomotor performance, body sway, and steady gait in human participants (Ketola et al., 2015; Perez & Holmes 2005; Prescott et al., 1993). Although there is very little convincing evidence for a significant ataxic effect in kava consumption, multiple studies have implicated kava in situations where is it unclear whether there is a causal connection (Berry et al., 2019; Ketola et al., 2015; Perez & Holmes, 2005). Unfortunately, a few more rigorous literature reviews and meta-analyses are also implicated in the misrepresentation of kava's effect on body sway. Although their comment is brief, (LaPorte et al., 2011) make a direct statement suggesting that kava likely increases body sway, which is repeated once more in Sarris et al. (2011), and again in White (2018). The reviews are focused on cognitive effects and clinical efficacy of kava, and when they discuss kava body sway effects, it is as a tangential justification of a cautious approach to kava clinical research, via a suggestion that kava induced body sway may statistically significant (LaPorte et al., 2011; Ooi et al., 2018; Sarris et al., 2011; White, 2018). The current critique is not intended to rebuke the authors for suggesting possible effects. This current research found no significant association between kava and positive or negative body balance impacts, in either the body sway or dynamic lower limb balance condition. Therefore, the conclusions which are drawn from Prescott et al. (1993) cannot be completely ruled out, despite that fact that the study was methodologically flawed because the kava beverage was diluted and mixed with orange juice – which likely skewed results and perhaps caused the cases of nausea described by the authors (Ahmed et al., 2013; Prescott et al., 1993). Therefore, until further studies are conducted in naturalistic kava settings, there is insufficient evidence to support or refute claims made by past researchers completely (Perez & Holmes, 2005; Prescott et al., 1993; Turner, 1986), although this study does provide evidence that any kava effect on human body balance is likely to be mild.

In terms of neurobiological accounts for kava's effect on human body balance, there are two prominent candidates for mechanisms of action. Due the wealth and breadth of the research which accounts for kava's anxiolytic effects by implementing GABAergic effects (Chua et al., 2016; LaPorte et al., 2011; White, 2018), it is still highly likely that should kava be proved to cause increased instability or human body sway, GABA will be one of the neurotransmitters which operate for kava in the CNS and NMJ (Kautu et al., 2017; White, 2018). Past research has shown that medications which are designed to modulate GABA(A) receptors have been associated with increasing the likelihood and rate of patient falls as a product of side-effects and sedation associated with consuming them (Mets

et al., 2010; Tapper, Risech-Neyman, & Sengupta, 2015). These drugs include BZDs, which is considered the closest pharmaceutical analogue to kava (Pittler & Ernst, 2003; White, 2018), and are substances which are readily prescribed for anxiety (Tähkäpää et al., 2018; Sarris et al., 2013). Kava and BZDs do not operate for the same binding sites at GABA (A) receptor (White, 2018), a fact which was described in Chapter 2, during the discussion of GABAergic mechanisms of action (Chua et al., 2016; Miller, 2013; Singh, 2004). How kava interacts with BZD is not well understood (White, 2018). Further research needs to be address possibilities of using kava and certain BZDs concurrently for clinical purposes. Other studies have suggested that kava likely potentiates BZDs, likely potentiating the effects and the harms caused by the latter (LaPorte et al., 2011; Sarris et al., 2011; Teschke & Lebot, 2011). However, as Tawfiq et al. (2014) demonstrate, kava appears to decrease the severity of side effects of Diazepam in a dose-dependent manner when it is administered concurrently. Kava is known to have none of the side-effects associated with BZDs; researchers speculate that the reason for this is kava blockade of noradrenaline and dopamine reuptake (Showman et al., 2015). However, the minor side-effects of a substance should not be ignored, and it is worth noting that kava has been associated with increased body sway based on its GABAergic mechanisms observed and inferred by researchers (LaPorte et al., 2011; Prescott et al., 1993). It is possible that even if kava has a beneficial influence on cognitive aspects of benzodiazepine treatment (Tawfiq et al., 2014), concurrent treatments may also potentially increase the risk of falls already associated with benzodiazepine treatments (Mets et al., 2010). Overall, kava's psychopharmacodynamics are complex and not well understood, and although GABAergic influences on muscular control is a candidate for body sway effects (LaPorte et al., 2011; Prescott et al., 1993), other studies have suggested other mechanisms of action.

Kautu et al. (2017) found that kavalactones which were administered to NMJ receptors increased the volume of ACH transmission in a dose-dependent manner, causing mild convulsions leading to muscular paralysis when the dosages were increased (p.3). These results might suggest that the nature of kava's interaction with the human muscular system is agonistic and that cholinergic mechanisms of action are responsible for muscular contractions which have been associated with kava from previous studies using frog sartorius muscles (Singh, 2004). However, (Kautu et al., 2017) also noted that there is a large bod for research which suggests kavain GABAergic action may cause muscular instability and sedation (pp.4-5). Therefore, the exact mechanism of kava action at the NMJ is still not well understood and more research will be needed to understand the molecular properties of kava pharmacodynamics which influence potential effects of human body balance.

Limitations and Suggestions for Future Research

As of writing, evidence concerning kava's effect on human body balance is not extensive (Kautu et al., 2017; Perez & Holmes, 2005; Prescott et al., 1993). This study was an attempt to address unknown effects of high volume kava consumption (Aporosa et al., 2020; Aporosa & Tomlinson, 2014), as much of what is understood about how kava affects human body balance is often inferred from a single study (Prescott et al., 1993), which has never been replicated to any satisfactory degree and involved low kava consumption volumes (500ml) compared to the current study (3.6L). This study did support the current research's hypothesis that kava increases body sway and decreases lower limb dynamic balance because there was no statistically significant change in any of the four COP parameters; although, $COP^{area\ 95\%\ ellipse}$ did increase slightly the change was not significant (pre-test mean= 11.94cm^2 and post-test mean= 16.113cm^2; see Appendix F). Furthermore, there was no statistically significant change in Y-balance test scores during and after the data collection session (pre-test: right-leg mean = 0.60, standard deviation = 0.16; left-leg mean = 0.59, standard deviation = 0.04, and post-test: right-leg mean = 0.62, standard deviation = 0.10; left-leg mean = 0.61, standard deviation = 0.09). However, some useful suggestions can be made for future research, based on the attached journal article.

Firstly, staggering testing cycles at different intervals over the 6-hour kava session (Aporosa et al., 2020), may enable time to be used more efficiently, while there should be accommodations made for strolls before each testing cycle in future data collection processes to address the issue of dead legs which one participant complained about, which is also mentioned in the kava literature (Cawte, 1985; Turner, 1986). This was not possible for the current research project due to time constraints. Furthermore, a tentative conclusion that might be drawn as a result of this study is that the Prescott et al. (1993) results may not be as reliable as the literature implies (LaPorte et al., 2011; White 2018). Prescott et al. (1993)also used a low number of kava drinking participants (n=12), the volume of kava consumed (500ml) were far lower than the current study (3.6L), and the kavalactone content was lower, meaning that it was reasonable to assume that the current results would demonstrate an increase in body sway as was documented by Prescott et al., (1993).

One possible reason for the deviation in the current study from the Prescott et al., (1993) study is, the two studies used different measurement tools. Prescott et al., (1993) attached a weight to the participants via a pulley system clipped to the participant's back. The instrument used in Prescott et al., (1993) may have been able to measure body sway from the upper torso which was not observed in the results of the current study because the point of contact with the instruments was the feet – directly on top of an AMTI ACCUgait force plate and y-balance-test (Miyata et al., 2015). It is also possible that kava does not cause a significant increase in lower limb instability and body sway

but does affect upper torso body sway (Prescott et al., 1993). In this instance, the instruments used in the current study would not be sensitive to those body balance alterations and future studies could account for that issue by using another method of measurement to better establish the body sway changes across the whole body throughout the kava session. One candidate may be a camera-based approach, where the participant is monitored via motion capture, as the method has been demonstrated in previous studies concerning balance (Maudsley-Barton et al., 2020; Yeung et al., 2014). Although this approach is more complex and more challenging for the researcher and participants, there may be a better chance of capturing kava effects more accurately when there are a low number of participants. At the very least, a variety of approaches which accounts for the entire human anatomy may be an advantageous approach.

Dr Vicnent Lebot's comments about the nature of kava; a compound of various chemicals, combined with the setting, presented in a culturally appropriate way (Goldberg, 2019; Lebot & Legendre, 2016; Lebot et al., 1997) may allude to a problem within the wider project within psychology and neuroscience. Human biology is not always amenable to strictly deterministic approaches due to high levels of complexity (Damasio & Carvalho, 2013; McFadden & Al-Khalili, 2016). For example, it is rarely possible to take one gene from the whole and predict what effect that might have (Rutherford 2018). The problem is exponential, whereby the number of outcomes becomes increasingly more complex as the number of compounds in any substance is increased. Kava contains over 30 psychoactive substances, which means the way those substances are combined likely effects the properties of any kava beverage which is drunk (Lebot et al., 2014; Lebot et al., 1997; Teschke & Lebot, 2011). By isolating single or small amounts of the kava beverage, the reductionist approach to kava science; e.g. kavain has GABAergic properties (Cock & Cheesman, 2016; Kautu et al., 2017), may be misinterpreting kava beverage effects which are much more probabilistic; based on how much of various kavalactones and other compounds are absorbed and at what rate (Lebot et al.,1997). However, at present researchers are limited to making inferences from limited neurobiological studies administering isolated or tailored kavalactone compounds (Aporosa et al., 2020; Kautu et al., 2017). These are not likely to reliably reflect traditionally influenced kava beverages which; vary in potency, chemotype, quality, and consistency, based on consumer preference (Aporosa & Tomlinson, 2014). Improvements to the bio-medical model can be made in future research to a certain extent – although comprehensive cultivar wide understanding is likely to be impossible (Kautu et al., 2017). However, as it stands, kava researchers continue to be limited by being restricted to the phenomenological domains available to them.

Even so, Traditionally influenced kava will likely never be fully understood through neurobiological approaches alone (Kautu et al., 2017), as it has been resistant to that approach in the

past (Goldberg 2019). Furthermore, some scientists reject the bio-medical model as it currently stands – without acknowledging the psycho-social-spiritual aspects of substance use (Mate, 2019). A more promising approach to naturalistic kava research seems to be to think about the beverage (Goldberg 2019; Aporosa et al. 2020), in a manner which reflects the variability of kava practices (Tecun, 2017), and accepts that one experiment will not illuminate the total extent of kava effects. An approach which accumulates data will likely be the most effective, as each whole kava mix is likely to be subtly different to the last (Lebot et al., 2014; Aporosa & Tomlinson 2014). Eventually, a probabilistic model of kava effects may become apparent to future researchers, but that will require a concerted approach consisting of neurobiological, experimental, and clinical research which goes beyond the current state of kava research that largely relies on inferences made based on past research which has not been sufficiently replicated (Kautu et al., 2017; LaPorte et al., 2011; Ooi et al., 2018; Perez & Holmes, 2005; Prescott et al., 1993; White, 2018)

The data collection for this research was very convoluted. Given the circumstances of the research process, which proceeded amidst a pandemic. In early February, the novel coronavirus SARS-COV-2 outbreak resulted in the initial research experiment not taking place on March 13 Friday. Because of the disease associated with SAR-2, COVID-19, New Zealand was placed under a 1-month Lockdown, denoted LEVEL 4, which eventually lasted 5 weeks. However, this research was only permitted to be carried out when New Zealand entered a lesser security level of LEVEL 1, which only occurred on June 5. As a result, whereas there originally 12 participants ready to take part in the initial research in March, only 6 were able to participate in the later experiment on June 12. The original participant number (12) was decided on as the current research is an updated study based on Prescott et al. (1993) with a modified methodology. As this project also contributed to a master's thesis, there were further time constraints placed on the study which meant that the researcher was impelled to complete the experiment as early as possible based on an imposed completion date, and with the known risk that ethical approval for the experiment may be revoked should there be local or national outbreaks of COVID-19 at a later date. Due to the way a New Zealand master's degree is structured and scheduled, there were limited options, and the decision was made by the researcher to conduct the data collection at short notice, as soon as possible after the nationwide lockdown was lifted by the New Zealand Government.

There were a few consequences for that decision. The first is that the researcher had planned to include a research assistant when the data collection was first devised, but due to the pandemic it was not possible to obtain an assistant. The assistant's role would have involved managing one of the testing sites; either the abbreviated y-balance test or the force plate. The result of conducting the research alone was that the session did not run as smoothly as it could have done. If there were

20participants present at the data collection, as originally expected, then the session would have run for far longer than the allotted 7 hours (including briefing and debriefing). The second consequence of the pandemic for this research was the low number of participants, which is the likely reason for the lack of significance observed during the analysis of the data. The researcher acknowledges that the constraints of the research may mean the study is limited by the number of participants. However, the results of this study will still be valuable as no similar study has been performed before, therefore information related to methodology, procedure, and analysis of results will provide useful data for future research.

Another aspect of the research which could be improved in the future is the researcher's use the instruments involved in any possible subsequent studies. For example, the researcher decided to use an abbreviated y-balance test for the data collection session (Shaffer et al., 2013). There were two reasons for the decision; firstly, there was a need to not over complicate the testing as it was not supposed to be difficult, and it was equally important that the baseline test was a fair representation of balance, rather than an estimation of how well a participant could achieve the task; secondly, the researcher was concerned about time constraints involved during the data collection. Kava (100ml) was served every 10 minutes, and every participant needed to be present for each serving. The concern with extending the Y-balance test include anterior left and right condition was that testing would take too much time. Upon reflection, the researcher considers that the right decision was made for this research as time proved to be quite a restrictive constraint. Subsequent studies could expand the Y-balance test, provided that the study design allowed time for the appropriate test trials to be completed.

Meanwhile, in the case of the force plate, time was not a constraint. The timing of the force plate testing was punctual and in hindsight, it may have been possible to include conditions other than the single, eyes-closed closed-stance condition included in the study – chosen based on results obtained in previous studies (Bauer et al., 2008; Miyata et al., 2015; Murray et al., 2019). There may be an opportunity for future researchers to consider further posture options. Furthermore, as time was not an issue, it would have been possible to include more force plate trials, rather than just the pre, mid and post-tests. The model was based on previous studies (Aporosa et al., 2020; Aporosa, 2017) and was maintained to attempt to standardise the process. However, perhaps more testing intervals; one each hour, would not have interfered with the kalapu where the kava session proceeded (Tecun 2017; Nabobo-Baba 2008; Aporosa et al., 2020). More data could be obtained, and perhaps more reliability and accuracy would be obtained, even with limited participant numbers (Bauer et al., 2008). The downside to that decision may be that the researcher would not be able to be a continuous member of the kava session. That would interrupt the 'set' and 'setting' of the kava session more than

is ideal (Aporosa et al., 2020; Nutt, 2012; Pollan, 2019). However, should the data present significant results, perhaps a concession could be made for the researcher not to be present at most of the kava session.

Conclusion

This study has reintroduced the subject of kava's effect on body balance into the scientific literature, as only the second study to investigate the role of traditionally influenced kava consumption in accounts of increased body sway, instability, or induced ataxia (Prescott et al., 1993).

The literature review outlined how kava mechanisms of action are not entirely clear, and that although the predominantly cited mechanism of action is GABAergic, there is substantial evidence to suggest other neurotransmitters are also involved. Furthermore, the current state of kava research does not apply to kava research conducted using traditionally influenced kava because the majority of the studies have been conducted using kava capsules, extracted kavalactones, or extremely low volumes of kava powder extracts (LaPorte et al., 2011; Ooi et al., 2018). Finally, Chapter 3 discussed the extent to which Prescott et al. (1993) have influenced the current evaluation of kava effects of human body balance. As it is the only experimental study involving human participants, Prescott et al. (1993) is cited by numerous studies and reviews which have argued that kava caused increased body sway (LaPorte et al., 2011; Ooi et al., 2018; Sarris et al., 2011; White, 2018), even though the researchers conducting the experiment used diluted kava which was adulterated by mixing orange juice with the aqueous kava extract (Prescott et al., 1993).

However, the study submitted to *Pacific Health Dialogue: The Journal of Pacific Research*, which constitutes Chapter 5, was justified in the literature review in Chapter 3, because while the previous studies which have suggested kava may cause increased body sway, hare somewhat supported by neurobiological assessments of kavalactone action at NMJ receptors, which demonstrate that kavalactones have the potential to cause both muscular sedation and partial muscular paralysis vis allosteric interactions with GABA and ACH networks respectively (Kautu et al., 2017; Singh, 2004; White, 2018). Evidence present in the kava literature combined with the fact that it is common for kava drinkers to drink large quantities of kava beverage and then drive home, and the anecdotal accounts of kava induced instability and ataxia (Perez & Holmes, 2005; Tarbah et al., 2013), mean that it is essential to update recent understandings of kava's effect on human body balance.

Analysis of the results found in the study in Chapter 5 did not support the hypothesis that traditionally influenced kava consumption will cause an increase in COP test scores, and a decrease in y-balance test scores because there was no statistically significant change in either the closed eyes, closed stance COP test condition, or the anterior y-balance test condition between baseline pre-tests and the post-tests. As a result, it remains unclear whether traditionally influenced kava has any significant impact on human body balance.

Future researchers should investigate the oft supported claims that kava consumption causes statistically significant increases in body sway (LaPorte et al., 2011; Prescott et al., 1993; Singh, 2004; White, 2018), and instability (Berry et al., 2019; Perez & Holmes, 2005; Tarbah et al., 2013; Tith & Lalwani, 2013; Turner, 1986), as the results of the current study – which involved higher volumes of more potent, unadulterated kava beverage than Prescott et al. (1993)- suggest that kava effects have been misrepresented or overestimated.

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Appendix A

Consent Form

Consent Form for Participants – Kava Drinkers

THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

CONSENT FORM

Project Title

Sway; The Effect of Kava on Standing Balance

A completed copy of this form should be retained by both the researcher and the participant.

Please complete the following checklist. Tick (✓) the appropriate box for each point.	**YES**
1. I have read the Participant Information Sheet (or it has been read to me) and I understand it.	
2. I have been given sufficient time to consider whether or not to participate in this study.	
3. I am satisfied with the answers I have been given regarding the study and I have been given a copy of this consent form and information sheet.	
4. I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time. I also understand that any data collected up to the point when I do withdraw, including Consent Forms, will be destroyed.	

5. I have the right to decline to participate in any part of the research activity.	
6. I understand that I am joining the study as a kava drinking participant, and that I feel comfortable drinking 6 x 100ml *bilo*/*ipu* per hour for 6 hours and that my balance will be tested on a force plate 3 times over the 6 hours.	
7. I understand that publications and presentations will result from this study and that my name will NOT appear in those publications or presentations. I also understanding that the researchers will make every effort to keep my identity confidential including using an allocated number instead of my name on the cognitive tests.	
8. I know who to contact if I have any questions about the study in general.	
9. I understand that I will be invited to a presentation of the study findings once completed. I also understand that my attendance at this presentation is not compulsory.	

Declaration by <u>participant</u>:

I agree to participate in this research project and I understand that I may withdraw at any time. If I have any concerns about this project, I may contact the Human Research Ethics Committee (HREC) that approved this study on: Phone: 0800 WAIKATO, Email: humanethics@waikato.ac.nz

Participant's name (Please print):

Signature: Date:

Declaration by member of <u>research team</u>:

I have given a verbal explanation of the research project to the participant, and have answered the participant's questions about it. I believe that the participant understands the study and has given informed consent to participate.

Researcher's name (Please print):

Signature: Date:

The lead researcher:

Harvey Aughton (Masters Student)

***Te Huataki Waiora*: Faculty of Health, Sport and Human Performance**

Ph: 07 838 4466 ext. 8282 or 021 838478

Email: ha102@students.waikato.ac.nz

The lead researcher's advisor:

Dr. Apo Aporosa

***Te Huataki Waiora*: Faculty of Health, Sport and Human Performance**

Ph: 07 838 4466 ext. 8282 or 021 838478

Email: apo.aporosa@waikato.ac.nz

Appendix B

Eligibility Screening Form

Sway: The Effect on Kava on Balance Eligibility and Screening Questionnaire

KAVA DRINKING PARTICIPANTS

THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

(PLEASE COMPLETE THIS FORM AND BRING IT WITH YOU TO THE TEST EVENT)

1. **General information**

Date of Birth: _____ Age: _____

Years of Education:_____ Marital Status:_____

Years in New Zealand:_____ Handedness: Right ☐ Left ☐

Occupation (eg. student, truck driver, lab. technician):_____

What is your ethnicity?:

Tongan☐ Samoan☐ niVanuatu☐ Rarotongan☐ Fijian☐ Māori☐ European☐

Other : _____(specify)

2. **Please indicate if you have ever received any of the following diagnoses:**

Concussion ☐Yes ☐No

Head Injury ☐Yes ☐No

Learning Disability ☐Yes ☐No

Depression ☐Yes ☐No

Anxiety Disorder ☐Yes ☐No

Personality Disorder ☐Yes ☐No

Psychotic Disorder ☐Yes ☐No

Have you been diagnosed with any other condition (e.g., stroke, multiple sclerosis) which may affect your performance?

_____

_____

_____

3. **Are you currently taking any prescription drugs** such as antibiotics, anti-anxiety (eg. *Benzodiazepine*), anti-depressant or sleep medication?
   ☐No ☐Yes: what? _____

   _____

4. **Is English your first language?** ☐Yes ☐No

   If **No**, what is your first language (specify):_____

(more questions over….)

5. **What is your kava drinking experience?** Tick what is appropriate:
   I drink kava about once per week: … ☐
   I drink kava about once per fortnight (2 weeks): ☐
   I drink kava about once per month: … ☐
   I drink kava occasionally, like a couple of times per year: ☐

6. **When you drink kava, what is 'usually' the situation or environment?** (eg. I go to a kava club/kalapu; I drink at home with a friend; I drink kava by myself; I drink small amounts to help me sleep):

   _____

   _____

   ____

7. **When you drink kava, how long do you 'usually' drink for?**: _____ hours/ ____ minutes.

8. What is you Height_____cm__What is your weight _____Kg

9. **Do you understand that you will be joining the study as a KAVA FASTING participant?** This means that:

- You will NOT have drunk any kava over the 4 days prior to the cognitive testing: ☐Yes ☐No
- That you WILL be familiar with kava drinking and have drunk kava at traditionally influenced kava sessions at least 20 times over the past 12 months. ☐Yes ☐No
- That you WILL drink kava at the cognitive testing event: ☐Yes ☐No
- The kava drinking session at the cognitive testing will be 6 hour long where you will be served 6 x 100ml cups of kava per hour. Do you feel you can drink this amount? ☐Yes ☐No
- That you will NOT be able to drink any alcohol in the 24 hours before cognitive testing. ☐Yes ☐No
- That you WILL have your balance tested on a force plate 3 times during the 6 hour kava session: ☐Yes ☐No
- That you are FREE TO LEAVE this study at any time including during the cognitive testing event: ☐Yes ☐No

---

The section is for the Research Staff: Final Eligibility check (**all should be yes**):

Over 18 years or age	…	…	☐Yes	☐No
No neurological or psychological conditions		…	☐Yes	☐No
Not taking contraindicated medication		…	☐Yes	☐No
Answered 'no' to question 4	…	…	☐Yes	☐No
Answered 'yes' to all questions in section 10		…	☐Yes	☐No
Inform participant if they meet the eligibility requirements			☐Yes	☐No

Appendix C

Information Sheet

Participant Information Sheet

THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

Project Title

Sway: The effect of kava on standing balance

Purpose

The aim of this research is to examine the effects of kava drinking on balance. This will be done by comparing kava drinkers with non-kava users. You are being invited to join the study as a FREQUENT KAVA DRINKER.

What is a FREQUENT KAVA DRINKER?

A FREQUENT KAVA DRINKER is a person who drinks kava regularly – 2 or more times per week – and has been doing so for at least 2 years. You know how to mix kava and understand what goes on at a typical kava session.

What would I have to do at this kava session?

You would be a participant at a 6 hour long kava session at The University of Waikato. There will be 6 servings of kava per hour. You will drink 100mls (approximately half an average sized *bilo* or *ipu*) of kava six times per hour for 6 hours. Three times during the kava session you will complete a test using a force plate that will assess your level of body sway. These test will measure your balance. Each test will take approximately 15 minutes.

What would I do when I was not being tested?

When not being tested, you would do what is normally done at kava session; relax, talk and listen to music. 'Kava chasers' – snacks (fruit, potato chips, nuts, etc.) – will be available together with water and juice, a toilet is nearby, and you can move about and stretch as you need.

When will the testing session be and what about getting there and home?

The testing session will start at 1pm on Friday 12th June You will be picked up from your home address by one of the research team in a University of Waikato minivan. You will be dropped home by the same researcher in the same minivan shortly after midnight.

Would I have to do any preparation for the testing?

Yes. The lead researcher, Harvey Aughton, or the supervisor, Dr. Apo Aporosa will phone you and explain the project to you and you can ask them any questions. If you still want to be involved in the kava session, they will send you a hand written questionnaire survey form (2 pages) to fill out. This will involve writing short answers (such as your name, address, age, ethnicity, occupation, brief medical history, kava drinking history, etc.) and ticking boxes. You may choose not to answer a question. You will bring the questionnaire survey with you to the kava session. You may ask the researchers about any questions including the ones you have chosen not to answer on the questionnaire survey. When you have received an answer to your question, you are free to answer or not answer the survey question.

Upon arrival at The University of Waikato kava session, you will be asked a few screening questions by the researchers (such as, have you read this information sheet and do you understand it, do you have any other questions, have you consumed alcohol in the past 24 hours, have you consumed caffeine or energy drinks in the past five hours, are you currently taking prescription medicines such as antibiotics, anti-anxiety, anti-depressant or sleep medication, etc.). These last questions are to assist test accuracy. Also you will be asked if you are still happy to be a part of the study and if so, you will sign a consent form.

Are there any things that could prevent me from being a part of this study?

Possibly yes. Here are the 'criteria' for you as a FREQUENT KAVA DRINKING research participant:

- You WILL be a regular kava drinker and routinely use kava 2 or more times each week, have done so for at least 2 years, and you feel you can drink 6 x 100mls bilo/ipu/cups per hour for 6 hours.
- You WILL be over the age of 18 years old.
- You will NOT be taking antibiotics, anti-anxiety (eg. *Benzodiazepine*), anti-depressant or sleep medication.
- You will NOT have consumed alcohol in the previous 24 hours prior to testing.

- You will NOT have consumed coffee, caffeinated drinks (including Coke) or energy drinks from midday before the test session or during the test session.
- You will NOT DRINK ANY KAVA IN THE 4 DAYS PRIOR (BEFORE) TESTING.

What happens at the end of the testing?

We will give you a meal and a small gift as a token of our appreciation for giving up your time and for completing the tests. We will then take you home. The researcher will contact you the next morning to ensure you are well. Over the following months the test results will be analysed and you will be invited to a presentation should you wish to know the test findings.

Is there anything else I should know about the test session?

Yes, that at any stage you can ask questions and/or withdraw as a research participant. More importantly, the researchers will continually remind you of this as we do not want you to feel trapped or uncomfortable. Additionally, to add to your comfort, you may want to eat a good meal 2 to 3 hours before we pick you up, a standard practice by many kava drinkers. There will be 'kava chasers' available during the kava session and testing. We will give you a meal at the end of the kava and testing session approximately 7 hours after you arrive. Finally, you may want to dress comfortably as there will be a period of about 10 minutes when you will be required to sit on the floor while we do a cultural presentation of kava. You will not be expected to respond to this. After this time you may sit on a chair or cushions.

What is kava and how safe is it?

Kava is a non-alcoholic drink made from the *piper mythisticum* plant. Kava has great cultural significance to the people of the Pacific and has been used by them for thousands of years for political, medicinal and social purposes. The effects of kava are soporific (sleep inducing) and relaxant with anti-anxiety/stress properties. These effects can make you feel a little lazy the following day similar to a lack of sleep. Researchers believe kava is safer than Paracetamol/Panadol pain medication (Rasmussen, 2005:6-7) with no reported hepatotoxicity (liver damage) among traditional kava users (Showman et al, 2014:58). Kava though can cause exfoliating dermopathy (a dryness and flacking of the skin) when consumed over many days at large volumes, although this subsides as kava consumption is reduced and has no lasting impacts. Kava is frequently consumed over many hours' at large volumes with this large amount of liquid in the stomach feeling uncomfortable to those with limited kava experience. If you are participating in the cognitive tests you will be familiar with kava's taste, effects (during and after) and feeling in the stomach.

What about my privacy and what will happen to the information collected during the cognitive tests?

As a research participant you will be allocated a number and that number will be used instead of your name on the computer tests. This means that no one other than the researchers can identify that it was you that did a particular test. The test data will be analysed and used to write a technical report, articles and presentations. No participants will be named in any publications or presentations. The researcher will keep your questionnaire/survey but will treat this with the strictest confidentiality and when not in use it will be locked away or password protected if held on a computer.

Declaration to participants

If you decide to take part in this research, you have the right to:

- Ask any further questions about the study during your participation;
- Refuse to answer any particular question;
- Withdraw from the study at any time;
- Be given a copy of the main points of the study findings when they become available.

This research has been approved by the University of Waikato Human Research Ethics Committee (HREC). Phone: 0800 WAIKATO, Email: humanethics@waikato.ac.nz

Who's responsible?

If you have any questions or concerns about the project, either now or in the future, please feel free to contact either:

The lead researcher:

Harvey Aughton (Masters Student)

***Te Huataki Waiora*: School of Health, Sport and Human Performance**

Ph: 07 838 4466 ext. 8282 or 021 838478

Email: ha102@students.waikato.ac.nz

The lead researcher's supervisor:

Dr. Apo Aporosa

***Te Huataki Waiora*: School of Health, Sport and Human Performance**

Ph: 07 838 4466 ext. 8282 or 021 838478

Email: apo.aporosa@waikato.ac.nz

Appendix D

Ethics Approval Letter

The University of Waikato
Private Bag 3105
Gate 1, Knighton Road
Hamilton, New Zealand

Human Research Ethics Committee
Julie Barbour
Telephone: +64 7 837 9336
Email:humanethics@waikato.ac.nz

THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

17 December 2019

Harvey Aughton
By email: ha102@waikato.students.ac.nz, harv.au@gmail.com

Dear Harvey,

HREC(Health)2019#79: Sway: The effects of kava on balance

Thank you for submitting your amended application HREC(Health)2019#79 for ethical approval.

We are now pleased to provide formal approval for your project where you will recruit approximately 18 regular kava drinkers to participate in a six hour traditional kava drinking session. Before, during and immediately after the kava session, participants will complete a self-assessment of perceived stability, followed by two balance tests on a force plate.

Please contact the committee by email (humanethics@waikato.ac.nz) if you wish to make changes to your project as it unfolds, quoting your application number with your future correspondence. Any minor changes or additions to the approved research activities can be handled outside the monthly application cycle.

We wish you all the best with your research.

Regards,

Julie Barbour PhD
Chairperson
University of Waikato Human Research Ethics Committee

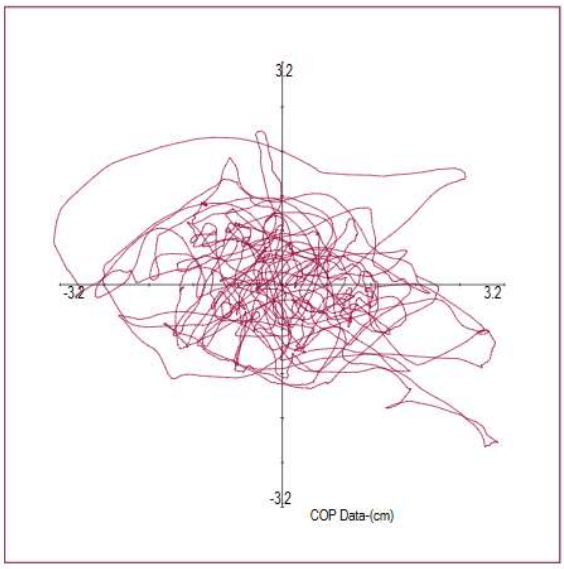
Appendix E

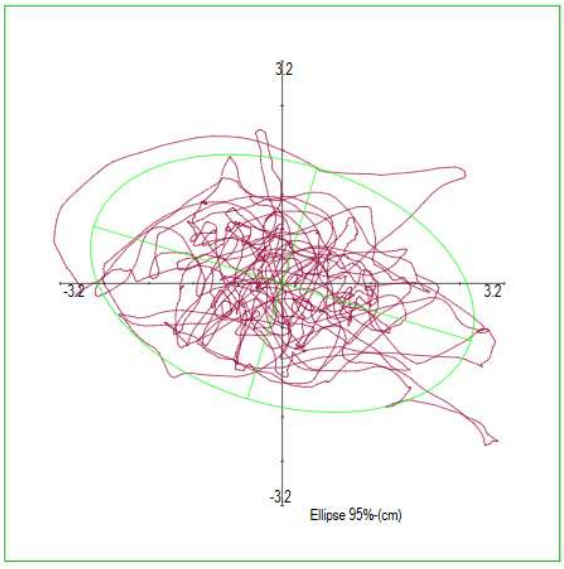
Centre of Pressure Plot Exemplar

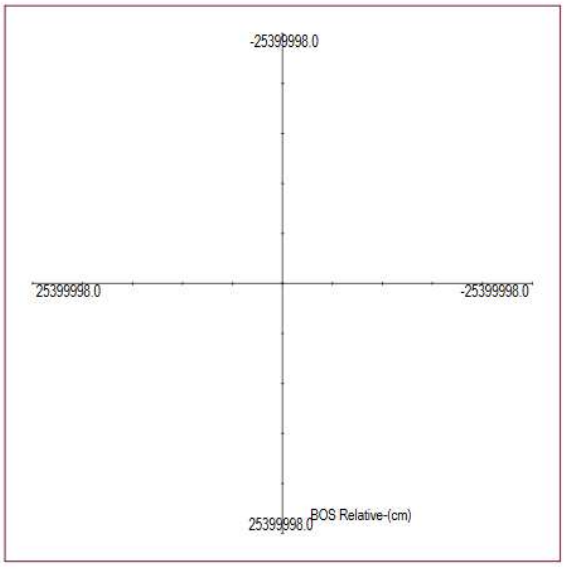
Subject Information

Number:	001
First Name:	
Middle Name:	
Last Name:	
Date:	06/11/20
Trial Type:	Closed Base
Trial Eyes:	Eyes Closed

Balance Test







Appendix F

Balance Clinic Group Data

Pre-Test

Path Lgth cm	V Avg cm/sec	Area95 cm sq
196.015	6.534	15.027
266.430	8.881	31.263
165.308	5.510	16.999
80.617	2.687	10.913
105.441	3.515	27.582
79.021	2.634	10.740
74.067	2.469	9.177
92.396	3.080	9.784
95.095	3.170	13.717
76.667	2.556	7.593
66.141	2.205	7.106
75.551	2.518	10.338
127.343	4.245	4.412
117.751	3.925	7.163
100.559	3.352	6.254
105.939	3.531	10.392
84.530	2.818	5.001
109.620	3.654	11.435
Summary statistics		
112.138	3.738	11.939
49.357	1.645	6.986
66.141	2.205	4.412
266.430	8.881	31.263
200.289	6.676	26.850

Mid-Test

Path Lgth cm	V Avg cm/sec	Area95 cm sq
147.608	4.920	20.889
161.897	5.397	17.564
206.458	6.882	26.828
83.805	2.794	13.219
73.387	2.446	19.786
69.872	2.329	6.807
83.014	2.767	11.017
57.839	1.928	4.388
75.976	2.533	8.870
68.449	2.282	8.105
69.211	2.307	7.161
63.491	2.116	8.297
63.491	2.116	8.297
102.253	3.408	8.480
92.252	3.075	8.565
89.425	2.981	6.653
120.283	4.009	11.529
131.475	4.382	15.093
137.042	4.568	25.035
Summary statistics		
99.854	3.328	12.452
39.412	1.314	6.416
57.839	1.928	4.388
206.458	6.882	26.828
148.619	4.954	22.439

Post-Test

Path Lgth cm	V Avg cm/sec	Area95 cm sq
196.993	6.566	68.852
141.763	4.725	34.954
170.710	5.690	32.612
77.515	2.584	14.541
80.765	2.692	20.559
71.291	2.376	14.464
75.106	2.504	10.116
58.773	1.959	4.767
65.763	2.192	4.899
46.995	1.567	5.167
61.258	2.042	10.619
56.115	1.870	6.512
81.602	2.720	7.649
100.570	3.352	8.738
89.604	2.987	5.845
134.566	4.486	13.092
131.920	4.397	14.748
92.236	3.075	11.879
Summary statistics		
96.308	3.210	16.112
40.862	1.362	15.324
46.995	1.567	4.767
196.993	6.566	68.852
149.998	5.000	64.085