**Background**

- Kava is a psychoactive, anxiolytic beverage derived from the *Piper Methysticum* plant
- 45-78% of regular users develop a ‘Kava dermopathy’ (Figure 1) which resolves on cessation
- Previously proposed aetiologies include niacin deficiency, systemic contact dermatitis, photosensitivity, and nitric-oxide synthase inhibition

**Our experience**

- 1000 Fijian adults were examined
- Kava dermopathy was a common finding, known as ‘kanikani’
- In Fiji, kanikani is associated with privilege, and more common among men
- Remedies among locals include topical coconut oil and exfoliation in water (pers. commun. Dr Tuicakau)

**Hypothesis**

- We note clinical similarities between kava-dermopathy and lamellar ichthyosis, e.g. powdery dryness, palmoplantar hyperlinearity, and polygonal scale lacking erythema
- Lamellar ichthyosis (type 3) results from impaired CYP450 enzymes, including CYP4F22 – essential for synthesis of cutaneous lipids from arachidonic acid
- Cutaneous lipids form the epidermal extracellular matrix between keratinocytes, preventing paracellular trans-epidermal water loss
- Kava metabolites strongly bind with and inhibit structurally similar CYP450 enzymes (4A11, 2C9 and 1A2) which are also required for lipid biotransformation and metabolism
- Niacin and triparanol, drugs associated with acquired ichthyosis, are also metabolised by CYP450 enzymes and may similarly impair lipid metabolism via such pathways

**Conclusion**

- We propose Kava inhibits similar CYP enzymes to those impaired in lamellar ichthyosis, which are likely also involved in cutaneous lipid synthesis – resulting in an ‘acquired ichthyosis’
- Kava dermopathy should be formally renamed ‘Kava Ichthyosis’, and could benefit from therapies for ichthyoses (e.g. topical urea)

**References**

4. Zhu, Q. et al. (2019). CYP1A2 contributes to alcohol-induced abnormal lipid metabolism through the PTFE/AKT/SREBP-1c pathway. Biochemical and biophysical research communications