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## Evaluation of the anti-diabetic potential of the herb *Hymenocardia acida*

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**Introduction:** Insulin resistance (IR) is a condition in which insulin target tissues show reduced response to the hormone. IR in skeletal muscle and liver in particular are major contributors to the development of type 2 diabetes (T2D). Several oral anti-diabetic drugs have been used with some success over the years to reduce IR and thus blood glucose. However, given their variable efficacy between individuals and the occurrence of occasional side effects, the search for alternative therapeutics continues. Many conventional medicines were originally derived from plant materials, while herbal remedies remain popular in the western world and are commonly the only affordable form of medication available in the developing world. A number of plant products are used by traditional healers in West Africa for the treatment of diabetes in people, including derivatives of *Hymenocardia acida*. However, objective study of the efficacy of extracts of this herb has been very limited to date

**Aims:** In this project, we aim to evaluate the potential for extracts of *H. acida* and other West African herbs to overcome IR using *in vitro* and *in vivo* models.

**Methods and Results:** Rat skeletal muscle (L6Glut4myc) myoblasts and H4IIE hepatocytes underwent viability testing using resazurin with a range of concentrations of MeOH, CCl<sub>4</sub> and hexane extracts of 3 plant products. The majority of these extracts did not reduce viability below the 80% of control threshold. Subsequently, differentiated myotubes and hepatocytes were palmitic acid (PA) treated for 16h to generate IR and then incubated with the plant extracts at 3 concentrations across a 100-fold range, below any concentration that impaired viability. Changes in insulin sensitivity were then assessed using radiolabelled 2-deoxyglucose uptake or glucose production in myotubes/hepatocytes respectively  $\pm$  insulin. Preliminary findings suggest that *H. acida* extracts ameliorate the defect in insulin-stimulated glucose uptake in insulin resistant myotubes.

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