INTRODUCTION

The magnitude and duration of postprandial hyperglycaemia due to hydrolysis of carbohydrates in the small intestine are major risk factors of macro- and microvascular complications in diabetes. Indeed, diabetes management strategies may involve several strategies that suppress postprandial glucose peaks. Reports from our laboratory indicate that Syzygium aromaticum-derived oleanolic acid (OA) inhibits the inhibition of the carbohydrate hydrolyzing enzymes in the small intestine 

OBJECTIVES

The objectives of this study were to:

1. evaluate the influence of OA on postprandial blood glucose concentrations in normal and STZ-induced diabetic rats
2. examine the effects of OA on the hydrolysis of sucrose and starch.

EXPERIMENTAL DESIGN

The effects of OA on postprandial blood glucose changes were assessed in non-diabetic- and STZ-induced diabetic male Sprague-Dawley rats while the effects on intestinal carbohydrate-hydrolyzing enzymes were investigated in vitro.

IN VIVO STUDIES

Oral glucose tolerance (OGT) responses

OGT responses were monitored in non-diabetic and STZ-induced diabetic rats loaded with monosaccharide, disaccharide and polysaccharide. Oral glucose tolerance assays were carried out in 6 rats (n=6 in each group). Values are presented as mean ± SEM.

RESULTS AND DISCUSSION

By comparison with animals loaded with carbohydrates alone, co-administration of OA with glucose, sucrose and starch significantly reduced the peak blood glucose values of separate groups of non-diabetic and STZ-induced diabetic rats (Figure 1). There is a concentration-dependent reduction in the Cmax values (Table 1). The suppression of the postprandial glucose spikes response by OA to carbohydrate loads was associated with a reduction of the area under the blood glucose-time curve (AUC) of non-diabetic and diabetic animals (Table 1). The in vivo half-maximal inhibitory concentrations (IC50) of OA on sucrose, α-amylase and α-glucosidase were calculated using blood glucose concentrations following oral loading of control and OA treated non-diabetic rats. Values are presented as means, and vertical bars indicate SEM of means (n=6 in each group).

CONCLUSION

These results suggest that OA suppresses postprandial hyperglycaemia perhaps via the inhibition of the carbohydrate-hydrolyzing enzymes in the small intestine.

REFERENCES