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ANTIPROLIFERATIVE PROPERTIES OF *MUSA PARADISIACA CV (AWAK)* ETHYL ACETATE TOWARDS PRIMARY MOUSE EMBRYONIC FIBROBLAST CELL (3T3-L1)

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TABLE OF CONTENTS

ABSTRACT

Diabetes mellitus is a universal disease that affecting millions of people worldwide. Currently, the common agents used to control this disease are hypoglycemic drugs and insulin. Apparently, the use of these agents has many prominent side effects and complications. Search for a better alternative is a very appealing. The present study aims to investigate the potential of banana soft pith ethyl acetate extract of *Musa paradisiaca* as safer natural hypoglycaemic agent. Primary mouse embryonic fibroblast cell (3T3-L1) was used in this study. Quantitative study of cell viability and qualitative study of morphological changes were examined. Both MTT 3-(4,5-dimethylthiazol-2-yl)-2,5-dimethyl tetrazolium bromide dye assay and acridine orange/propidium iodide (AO/PI) staining indicated a general decrease in the cell viability percentage and number of cell observed under confocal microscope towards all concentrations, except for 250μg/ml of the extract which minute increment in cell viability percentage. The above findings suggest that the banana soft pith ethyl acetate extract of *Musa paradisiaca* may exert potential hypoglycaemic action through lipolysis which required further investigations in the future.

CHAPTER 1

INTRODUCTION

1.1 Background

Insulin resistance is defined as decreasing in rate of glucose able to be elicited by a given concentration of insulin compared to the normal range (Scheen, 2005). It is accepted as a major cause of type 2 diabetes mellitus (T2DM). Insulin resistance may be caused by several factors. It is well appreciated role in diabetes, obesity, and metabolic syndrome. Obesity is the single most important contributor to insulin resistance (Tahrani *et al.*, 2010). Glucose toxicity which result from chronic hyperglycemia and increases in free fatty acid uptake by β cell can directly induced insulin resistance. Other than that, insulin resistance may be caused by smoking (Woynillowicz *et al.*, 2012 & Melnik, 2009), use of hormonal contraceptives, androgens, glucocorticoids, beta-adrenergic blockers, thiazide diuretics, intake of food with high glycaemic index, and reduced physical activity (Melnik, 2009).

The first response to insulin resistance is compensatory hyperinsulinemia. If β cells still can compensate with adequate insulin secretion to overcome the resistance, glucose metabolism remain normal. Many data suggest that insulin resistance is the result of defects in insulin action at the cellular level. Insulin