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

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ABSTRACT

Type 2 diabetes mellitus (T2DM) and its complications have become a major public health concern in many countries. Around the world, diabetes prevalence in adults aged 20 and over 4 percent in 1995 and it is expected to increase to 5.4 percent in 2030 with the developing countries have a slightly higher prevalence than developed countries. In this study, th cytotoxic effect of banana soft pith (BSP) (*Musa paradisiaca*) hexane extract was evaluated on primary mouse embryonic fibroblast cell (3T3-L1). This study found that hexane extract of BSP (HA) markedly inhibit the proliferation of 3T3-L1 adipocytes at high concentration. The cell population was visually assessed using laser scanning confocal microscopy following AO/PI staining which shows consistent result with cytotoxicity study with prominent reduction of viable cell. As a conclusion, this study suggested that BSP extract has potential therapeutic benefit towards T2DM that require further investigation.

CHAPTER 1

INTRODUCTION

1.1 Background

Insulin resistance can be defined as a reduction in the rate of glucose disposal elicited by a given insulin concentration compared to the normal range. It is a physiological condition in which cells are no longer responsive towards normal action of insulin. Insulin resistance may be caused by several general factors.

To understand the role of these factors in the onset of insulin resistance one has to consider insulin signalling pathway. Insulin binds to its cell surface receptor, a protein tyrosine kinase, resulting in the activation of the receptor tyrosine residue via autophosphorylation. Activation of insulin receptor tyrosine kinase will leads to phosphorylation of insulin receptor subtrates 1 (IRS-1) and other proteins that are capable to induce tyrosine phosphorylation cascade. Tyrosine phosphorylated IRS-1 activates phosphatidylinositol-3-kinase (PI-3K) pathway which signals through Akt (protein kinase B) to mediate the translocation of the glucose transporter GLUT4 from an intracellular pool to the plasma membrane which is crucial for insulin stimulated glucose transport (Serrano *et al.*, 2009). The PI-3K pathway also stimulate protein synthesis, glycogen synthesis and lipogenesis.

Insulin resistance may be resulted from insulin receptor inactivation due to increased level of free fatty acids (FFA). Elevated fatty acids metabolism products activate