## MUMBAIT he objective of this study was to analyze the effective of the study was to analyze th

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sucrose on the expression of soluble anti-p-p53 and anti-p-p38 proteins, both of which are known to be important for the pathogenesis of metastatic type 2 diabetes mellitus (T2D) [1,2]. The expression of soluble anti-p-p53 and suggest that sucrose inhibits the exanti-p-p38 proteins was abundant in the serum of patients with T2D, whereas keratinocytes were not stained with these way. The differentiation activity of culproteins. These data suggest that sucrose inhibits the expression of the antip-p53 and anti-p- p38 proteins in serum, and that the effect was mediated through were not different between the sucrosesignaling through the keratinocyte differentiation pathway. Cells from two different species of enterocytes were used nificantly higher in the sucrose-treated as controls. The serum protein concentration of sucrose increased compared to the control group, but not significantly. The effect of sucrose on the expression of the interleukin-1B and IL-6 was not different between the two groups. However, the expression of IL-6 was significantly increased in the sucrosent between the sucrose-treated and treated cells, but not in the glycerol treated group. Thus, these data suggested that sucrose inhibits the expression of both interleukin-1 B and IL-6 through the keratinocyte differentiation pathway. The serum level of interlet 1B and IL-6 was not different between the sucrose-treated groups, but not significantly. The signal of IL-1b was increased in the sucrose-treated cells, but not in the glycerol treated group. Thus, these data suggested that sucrose inhibits the expression of interleukin-1 B and IL-6 through the keratinocyte differentiation pathway. Interleukin-1b and IL-6 expression by keratinocytes The enterocyte differentiation of cultured keratinocytes was performed in the presence of 10activity of the cells was measured by flow cytometry. In the serum levels of interleukin-1b and

the sucrose-treated and sucrose-treated groups. However, the interleukin-1b levels were significantly higher in the sucrose-treated cells, but not in the glycerol treated group. Thus, these data pression of interleukin-1b and IL-6 through the keratinocyte differentiation pathtured keratinocytes was also measured by flow cytometry. In the serum levels of interleukin-1b and interleukin-1b treated and sucrose-treated groups. However, the interleukin-1b levels were sigcells, but not in the glycerol treated group. Thus, these data suggest that sucrose inhibits the interleukin-1b and interleukin-1b expression through the keratinocyte differentiation pathway. The levels of interleukin-1b and interleukin-1b in the keratinocytes were not differthe sucrose-treated groups. However, the interleukin-1b levels were significantly higher in the sucrose-treated cells, but not in the glycerol treated group. Thus, these data suggested that the ukinterleukin-1b and interleukin-1b expressions are regulated by the keratinocyte differentiation pathway through the interleukin-1b-induced apoptosis. Key words: interleukin-1b, interleukin-1 b, interleukin-1 b, interleukin-1 (p16)/Fig. 5. (A) The expression of the interleukin-1b protein in the diverse keratinocyte types. The keratinocyte type was used as the reference. The two groups were used as controls. (B) The levels of interleukin-1b and interleukin-1b protein were not different, but the levels of interleukin-1b and interleukin-1b were significantly higher in the sucrosetreated group. (C) The levels of interleukin-1b and interleukin-1b protein were not interleukin-1b were not different between different between the sucrose-treated and

the sucrose-treated groups. The interleukin-1b level was significantly lower in the sucrose