

## Effect of ethyl acetate leaf fraction of *Gongronema Latifolium* on alloxan induced diabetes rats

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Diabetes mellitus is a metabolic disorder caused by insufficient or absent production of the hormone insulin by the pancreas. The effect of ethyl acetate fraction of *Gongronema latifolium* on body weight, blood glucose level and lipid profile was evaluated in alloxan-induced diabetic rats using standard methods. The result revealed that there is a significant increase in body weight of Groups 2(15.17%), Group 3(12.05%) and group 4(14.05%) after 14 days treatment when compared with untreated diabetic rats (-5.61%). The effect of treatments on blood glucose level of alloxan-induced diabetic rats shows a significant increase ( $P < 0.05$ ) of percentage change of blood glucose in group 2(43.05%), 3(23.50%) and 4(43.36%) when compared with group 1(8.87%) and also a significant increase ( $P < 0.05$ ) in groups 2 and 4 when compared to group 3 on the 14th day of treatment. The results of the determined serum lipid profile indicates a significant decrease ( $P < 0.05$ ) in the levels of serum TC, TG, LDL-C and VLDL-C and a significant increase ( $P < 0.05$ ) in the level of serum HDL-C of group 2(TC=3.01  $\pm$  0.12mg/dl, TG=2.31  $\pm$  0.14mg/dl, LDL-C=0.83  $\pm$  0.28mg/dl, VLDL-C=0.46  $\pm$  0.34mg/dl and HDL-C=1.72  $\pm$  0.29mg/dl), Group 3(TC=4.56  $\pm$  0.96mg/dl, TG=2.52  $\pm$  0.24mg/dl, LDL-C, 1.13  $\pm$  0.09mg/dl VLDL-C=0.95  $\pm$  0.16mg/dl and HDL-C=1.27  $\pm$  0.10mg/dl) and Group 4(TC=3.56  $\pm$  0.64mg/dl, TG=2.36  $\pm$  0.16, LDL-C=0.09  $\pm$  0.14mg/dl, VLDL-C=0.67  $\pm$  0.32mg/dl and HDL-C=1.47  $\pm$  0.15mg/dl) when compared with the group 1(TC=9.92  $\pm$  1.02mg/dl, TG=3.18  $\pm$  0.26mg/dl, LDL-C=7.74  $\pm$  1.86mg/dl, VLDL-C=1.11  $\pm$  0.12mg/dl and HDL-C=1.08  $\pm$  0.11mg/dl). Consequently, treatment with the fractions and standard drug reverse the alloxan effect. This study thereby indicates that ethyl acetate leaf fraction of *Gongronema latifolium* may exert a hypoglycemic effect and could improve the lipid profile concentrations in diabetic rats.

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## Cynarin inhibits melanogenesis through ERK signaling pathway on $\alpha$ -MSH-induced melanin biosynthesis

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Cynarin is composed of plant such as artichoke, sunflower seed and burdock. These materials have compound such as chlorogenic acid (Caffeoylquinic Acid), caffeic acid, cynarin (Dicaffeoylquinic Acid), quercitrin (Quercetin Rhamnoside), arctiin, quercetin, luteolin. They provide special bio-physiological effect: chlorogenic acid, caffeic acid, quercitrin and luteolin as anti-melanogenesis factor; arctiin as inflammatory regulator (MAPK related factor) or protector from UVB-induced stress and so on. However, it has not been reported yet for melanogenesis biosynthesis of cynarin as component of these plants. Therefore, we also investigated melanogenesis of cynarin via signaling pathway. Melanogenesis-related protein as MITF, tyrosinase were decreased by cynarin via ERK signaling pathway in  $\alpha$ -MSH-induced human melanoma cells (MNT-1). Cynarin has also represented inhibition of melanin content and intracellular tyrosinase activity. Cynarin is being provided at anti-melanogenesis or anti-pigmentation factors because of its investigated anti-melanogenesis mechanism in  $\alpha$ -MSH-induced human melanoma cell and are well worth enough.

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