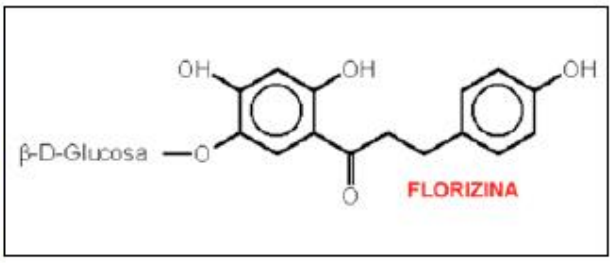
EQUIGLUC™

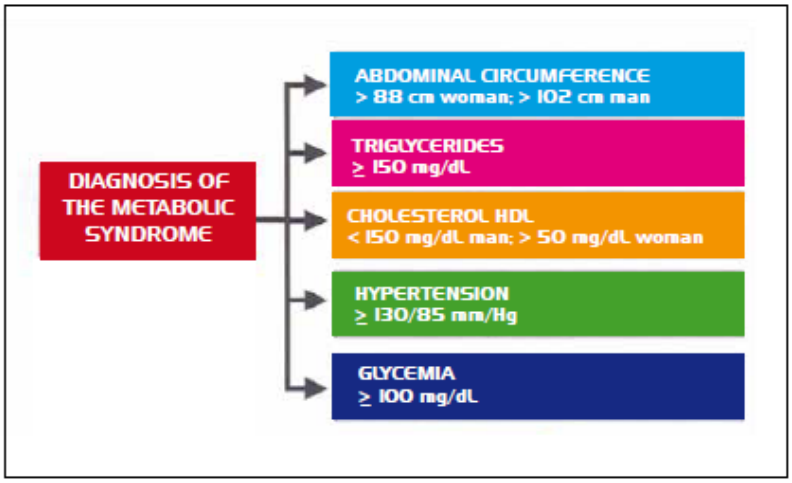
FLORIZINA

Apple Dry Extract
50% phlorizin



Nutratrade s.r.l.

EQUIGLUC™



PHLORIZIN: apple glucosidethat is able to limit PHYSIOLOGICALLY GLUCOSE post-prandial REABSORBTION

BIBLIOGRAPHY

Insulin Sensitivity-Enhancing Activity of Phlorizin Is Associated with Lipopolysaccharide Decrease and Gut Microbiota Changes in Obese and Type 2 Diabetes (db/db) Mice.

Mei X[1,2], Zhang X[1], Wang Z[2], Gao Z[1], Liu G[1], Hu H[3], Zou L[2], Li X[1]..

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Phlorizin Supplementation Attenuates Obesity, Inflammation, and Hyperglycemia in Diet-Induced Obese Mice Fed a High-Fat Diet.

Shin SK[1], Cho SJ[2,3], Jung UJ[4], Ryu R[5,6], Choi MS[7,8].

Physiol Res. 2016 Jun 20;65(2):239-50. Epub 2015 Oct 8.

Reduction in the amplitude of shortening and Ca(2+) transient by phlorizin and quercetin-3-O-glucoside in ventricular myocytes from streptozotocin-induced diabetic rats.

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Abstract

Phlorizin exists in a number of fruits and foods and exhibits many bioactivities. The mechanism of its antidiabetic effect has been known as it can competitively inhibit sodium-glucose symporters (SGLTs). However, phlorizin has a wide range of two-phase metabolism in systemic circulation and shows poor oral bioavailability. An alternative mechanism may involve gut microbiota in intestine. Sixteen obese mice with type 2 diabetes (db/db) and eight age-matched control mice (db/+) were divided into three groups: diabetic group treated with phlorizin (DMT group), vehicle-treated diabetic group (DM group), and normal control group (CC group). Phlorizin was given in normal saline solution by intragastric administration for 10 weeks. After the last treatment course, body weight, energy intake, serum lipopolysaccharides (LPS), insulin resistance, and fecal short-chain fatty acids (SCFAs) were compared. 16S rRNA gene denaturing gradient gel electrophoresis (DGGE) and quantitative PCR were used to determine the changes in microbiome composition. Coadministration of phlorizin significantly prevented metabolic syndrome by decreasing weight gain, energy intake, serum lipopolysaccharides, and insulin resistance, and the fecal level of total SCFAs was dramatically increased, especially butyric acid. DGGE and quantitative PCR demonstrated that phlorizin coadministration increased the gut microbial diversity and the growth of Akkermansia muciniphila and Prevotella. Meanwhile, the gut microbiota structure of db/db mice after phlorizin treatment was improved and approached the normal group. The mechanism of the hypoglycemic action of phlorizin is associated with LPS decrease and gut microbiota changes; briefly, it acts in the intestine to modify gut microbial community structure, resulting in lower LPS load in the host and higher SCFAs producing beneficial bacteria.

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Phlorizin Supplementation Attenuates Obesity, Inflammation, and Hyperglycemia in Diet-Induced Obese Mice Fed a High-Fat Diet.

Shin SK[1], Cho SJ[2,3], Jung UJ[4], Ryu R[5,6], Choi MS[7,8].

Abstract

Obesity, along with its related complications, is a serious health problem worldwide. Many studies reported the anti-diabetic effect of phlorizin, while little is known about its anti-obesity effect. We investigated the beneficial effects of phlorizin on obesity and its complications, including diabetes and inflammation in obese animal. Male C57BL/6J mice were divided into three groups and fed their respective experimental diets for 16 weeks: a normal diet (ND, 5% fat, w/w), high-fat diet (HFD, 20% fat, w/w), or HFD supplemented

with phlorizin (PH, 0.02%, w/w). The findings revealed that the PH group had significantly decreased visceral and total white adipose tissue (WAT) weights, and adipocyte size compared to the HFD. Plasma and hepatic lipids profiles also improved in the PH group. The decreased levels of hepatic lipids in PH were associated with decreased activities of enzymes involved in hepatic lipogenesis, cholesterol synthesis and esterification. The PH also suppressed plasma pro-inflammatory adipokines levels such as leptin, adipsin, tumor necrosis factor-α, monocyte chemoattractant protein-1, interferon-γ, and interleukin-6, and prevented HFD-induced collagen accumulation in the liver and WAT. Furthermore, the PH supplementation also decreased plasma glucose, insulin, glucagon, and homeostasis model assessment of insulin resistance levels. In conclusion, phlorizin is beneficial for preventing diet-induced obesity, hepatic steatosis, inflammation, and fibrosis, as well as insulin resistance.

Physiol Res. 2016 Jun 20;65(2):239-50. Epub 2015 Oct 8.

Reduction in the amplitude of shortening and Ca(2+) transient by phlorizin and quercetin-3-O-glucoside in ventricular myocytes from streptozotocin-induced diabetic rats.

Hamouda NN[1], Qureshi MA, Alkaabi JM, Oz M, Howarth FC.

Abstract

Diabetes mellitus is the leading cause of cardiovascular morbidity and mortality. Phlorizin (PHLOR) and quercetin-3-O-glucoside (QUER-3-G) are two natural compounds reported to have antidiabetic properties by inhibiting sodium/glucose transporters. Their effects on ventricular myocyte shortening and intracellular Ca(2+) in streptozotocin (STZ)-induced diabetic rats were investigated. Video edge detection and fluorescence photometry were used to measure ventricular myocyte shortening and intracellular Ca(2+), respectively. Blood glucose in STZ rats was 4-fold higher (469.64+/-22.23 mg/dl, n=14) than in Controls (104.06+/-3.36 mg/dl, n=16). The amplitude of shortening was reduced by PHLOR in STZ (84.76+/-2.91 %, n=20) and Control (83.72+/-2.65 %, n=23) myocytes, and by QUER-3-G in STZ (79.12+/-2.28 %, n=20) and Control (76.69+/-1.92 %, n=30) myocytes. The amplitude of intracellular Ca(2+) was also reduced by PHLOR in STZ (82.37+/-3.16 %, n=16) and Control (73.94+/-5.22 %, n=21) myocytes, and by QUER-3-G in STZ (73.62+/-5.83 %, n=18) and Control (78.32+/-3.54 %, n=41) myocytes. Myofilament sensitivity to Ca(2+) was not significantly altered by PHLOR; however, it was reduced by QUER-3-G modestly in STZ myocytes and significantly in Controls. PHLOR and QUER-3-G did not significantly alter sarcoplasmic reticulum Ca(2+) in STZ or Control myocytes. Altered mechanisms of Ca(2+) transport partly underlie PHLOR and QUER-3-G negative inotropic effects in ventricular myocytes from STZ and Control rats.