RESEARCH ARTICLE

Hesperidin & Apigenin Attenuates Diabetes & Lipid Levels in Experimentally Induced Diabetes Mellitus in Wistar Rats

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ABSTRACT

The aim of the present investigation is to study the antidiabetic potential of Hesperidin and Apigenin in Diabetic rats. Wistar Albino rats of either sex (150 to 200 g) were purchased from the CPCSEA approved vendor New Delhi. A total of three animals were used, which received a single oral dose in 500 mg/kg, body weight of different flavonoids. Doses equivalent to 25 mg and 50 mg per kilogram body weight were calculated, and suspended in 1% w/v Tween 80 solutions for the experiment. For OGTT, Different doses of both flavonoids i.e. Hesperidin & Apigenin were administered 60 min prior to oral glucose load (2.0 g/kg). Diabetes was induced in overnight fasted rats by Streptozotocin (60 mg/kg), 15 min. after the i.p. administration of Nicotinamide (120mg/kg). Streptozotocin was dissolved in citrate buffer (0.1 M, pH 4.5) & nicotinamide was dissolved in normal saline. During the study period of 21 days, animals was weighed at 0, 7, 14 and 21 day and effect of vehicle, standard drug and all solvent fractions on body weight was determined. Blood samples was collected by cardiac puncture and retro-orbital plexus method into EDTA sprinkled tubes and centrifuged at 3000 rpm for 20 min. Serum was separated as supernatant and stored at -20°C until analysis performed. The biochemical parameters, namely hemoglobin, urea, creatinine, total protein, total cholesterol, triglycerides, HDL determined. The other biochemical tests were performed using kits from Erba Diagnostics. Hesperidin and Apigenin significantly prevented the increase in blood glucose levels after 60 min. of glucose administration at the doses of 25 and 50 mg/kg. After 21 days blood samples were collected by retro-orbital plexus under mild anesthesia. Diabetic control group with no drug treatment showed no significant difference in the fasting serum glucose level after 21 days treatment as compared to the initial day treatment. Hesperidin & Apigenin (25 mg/kg & 50 mg/kg) treated diabetic rat showed a significant ($p< 0.05$) increase in body weight. The diabetic rat treated with glibenclaimide (10mg/kg) showed gradual and consistent increase in body weight. Treated diabetic rat with Hesperidin & Apigenin showed significant fall in total cholesterol, triglycerides, HDL-C, LDL-C and VLDL-C level at dose of 25 and 50 mg/kg. The Hesperidin and Apigenin had the antidiabetic activity with effects on lipids and other kidney markers.

Keywords: Hesperidin, Apigenin, Diabetes & Lipid Levels, Experimentally Induced Diabetes Mellitus, Wistar Rats

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INTRODUCTION

The term diabetes mellitus depicts a metabolic issue of different etiology portrayed by progressing hyperglycaemia with aggravations of starch, fat and protein processing coming about in view of disfigurements in insulin release, insulin action, or both.[1] The effects of diabetes mellitus integrate long stretch mischief, brokenness and frustration of various organs. Glucose is principal for giving energy to regular body capacities. In diabetes the blood glucose levels are extended on account of relative or out and out absence of insulin. Insulin is a substance. A Compound is a substance released by one of the organs in our body. This organ is organized in midriff and is known as pancreas. Insulin goes probably as a gatekeeper that licenses part of glucose into the cell.[2]

Assuming how much insulin is strange or the capability of insulin is to blame overabundance of glucose gathers in the body with unsafe consequences for the cells of different organs. Diabetes is a metabolic problem wherein body can't deal with glucose for its energy prerequisites. There are a few classes of oral hypoglycemic medications that apply antidiabetic impacts through various systems, in particular sulfonylureas, biguanides, α-glucosidase inhibitors, thiazolidinediones, and non-sulfonylureas secretagogues. Oral sulfonylureas, for example, glimepiride and glyburide, act to diminish

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glucose, fundamentally by hoisting insulin discharge from islets of Langerhans. This system works just within the sight of insulin.[3,4]

The α-glucosidase inhibitors, for example, acarbose and miglitol, hinder specific compounds answerable for the breakdown of starches in the small digestive system. Likewise, reversibly restrains both pancreatic α-amylase and α-glucosidase compounds by restricting to the carb restricting district and by impeding their hydrolysis into monosaccharides, which prompts a more slow retention along with a decrease in postprandial glucose levels.[4,5] Thiazolidinediones, for example, pioglitazone and rosiglitazone, have been accounted for to expand β-cell capability by bringing down free unsaturated fat levels that at last lead to β-cell demise.[6] The point of the current examination is to concentrate on the counter diabetic movement of chosen flavonoids for example Hesperidin and Apigenin in diabetic complexity.

MATERIAL AND METHODS

Experimental Animals

Wistar albino skinned rodents of one or the other sex (150 to 200 g) were bought from the CPCSEA endorsed seller New Delhi. They were kept up with under standard research facility conditions at 25 ± 2°C, relative mugginess (50 ± 15%) and typical photoperiod (12-hour light-dull cycle) were utilized for the trial. Business pellet diet (MFD, by Nav Maharashtra Chakan Oil Plants ltd., New Delhi, India) and water were given not obligatory over the span of study.

Selection of Dose

Intense oral harmfulness test was completed by the OECD rule No. 423. Wistar Pale skinned person Rodents were saved for the time being fasting preceding medication organization. A sum of three creatures was utilized, which got a solitary oral portion in 500 mg/kg, body weight of various flavonoids. The creatures were noticed for a time of 24 hr for the progressions in conduct, extreme touchiness responses and so on. Mortality, if, still up in the air over a time of about fourteen days.

Preparation of Doses

Doses equivalent to 25 mg and 50 mg per kilogram body weight were calculated, and suspended in 1% w/v Tween 80 solutions for the experiment.

Standard Oral tolerance test

Glucose opposition checks how well our body's phones ready to hold glucose load that is the essential wellspring of energy. The oral glucose strength test, or OGTT, is essential for illustrating metabolic problems, the normal progression from prediabetes to type 2 diabetes, and cardiovascular and metabolic prescriptions.

The two flavonoids, Hesperidin and Apigenin, were administered in varying amounts 60 minutes before the oral glucose load (2.0 g/kg). Animals were erratically allotted into following social occasions of six animals each. Group-I served as control and group-II treated with standard drug moreover group-III to VI were treated with bioactive flavonoids. The blood tests were accumulated from each social event not some time before glucose association (0 min) and at different time interval after glucose association. Blood glucose levels were evaluated using Glucometer.[7]

STZ Induced diabetic model

Streptozotocin is a normally happening synthetic that is especially harmful to the insulin-creating β-cells of the pancreas in warm blooded creatures.[8] It was at first separated in the last piece of the 1950s as an enemy of disease.[9]

In 2-month-old wistar rodents, a part of 200-230 mg/kg b.w. nicotinamide, given intraperitoneally 15 min before streptozotocin association (60 mg/kg i.v) yields a constraint of animals with 40% reduction of pancreatic-cell mass and moderate stable non-fasting hyperglycaemia (150-180 mg/dl),[10] yet also factors fortifying β-cell improvement. Diabetes was activated in for the time being went without rodents by Streptozotocin (60 mg/kg), 15 min. after the i.p. association of Nicotinamide. Streptozotocin was separated in citrate support and nicotinamide was deteriorated in standard saline. The treatment schedule was same as described in oral glucose tolerance test.

Treatment

Tests were acted in rodents that had been abstained for the time being (denied of nourishment for no less than 12 h except for permitted free admittance to water). The medication arrangement and vehicle was presented orally once everyday for 21 days. The impact of vehicle, flavonoids and standard medication on blood glucose, body not set in stone in that frame of mind at 0, 7, 14, multi day after oral organization. Blood test was gathered by retro-orbital plexus and heart cut of the multitude of creatures at 21st day under gentle sedation and biochemical boundaries was assessed utilizing the demonstrative unit (ERBA Symptomatic Mannheim, Germany) in Autoanalyser.

Evaluation of Physiological parameters

Blood tests was accumulated through cardiovascular cut and retro-orbital plexus procedure into EDTA sprinkled tubes and centrifuged at 3000 rpm for 20 min. Serum was separated as supernatant and taken care of at - 20°C until examination performed.

Biochemical analysis

The biochemical boundaries, specifically hemoglobin, urea, creatinine, all out protein, complete cholesterol, fatty, not set in stone. The other biochemical tests were performed utilizing packs from Erba Diagnostics.

Statistical analysis

All experiments were performed in triplicate using a minimum of three replicates. Values are expressed as mean ± S.E.M. Data were analyzed by one-way analysis of variance (ANOVA) followed by Dunnet's test. The values are ***P<0.0001, *P<0.0005, were considered significant, **P<0.001 when compared against control.

RESULTS AND DISCUSSION

Acute toxicity studies

The rodents were abstained for 3 h prior to dosing and given not obligatory water as it were. After culmination f study, there was no deadly side effects were found in rodent and mortality. Subsequently 1/tenth and 1/fifth portion for example 25 and 50 mg/kg were considered as a protected portion for the whole review.

Standard Oral tolerance test

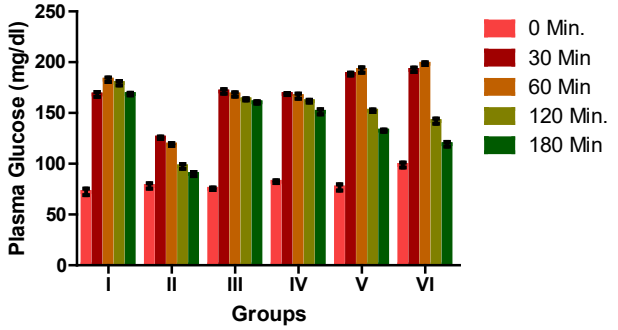
Various dosages of Hesperidin and Apigenin were managed 60 min preceding oral glucose load (2.0 g/kg). The OGTT is generally used to assess a creature's capacity to control blood glucose. Inside 30 min from beginning of the glucose resistance test, blood glucose fixation practically dramatically increased from its underlying degree of control. This hyperglycemia was kept up with until 60 min and afterward started to diminish. Hesperidin and Apigenin essentially forestalled the expansion in blood glucose levels after 60 min. of glucose organization at the dosages of 25 and 50 mg/kg **(Figure 1)**.

Effect of flavonoids on Blood glucose level

Following 21 days blood tests were gathered by retro-orbital plexus under gentle sedation. Diabetic benchmark group with no medication treatment showed no huge distinction in the fasting serum glucose level following 21 days treatment when contrasted with the underlying day treatment. Anyway treated diabetic gatherings showed steady and predictable fall in serum glucose level as displayed in **Figure 2** reliable fall in serum glucose level was seen in flavonoids treated bunches from 21 days dosing and esteems were viewed as huge when contrasted and untreated diabetic benchmark group.

Impacts of Hesperidin and Apigenin on Body Weight in Diabetic creatures

The impact of Hesperidin and Apigenin (25 mg/kg and 50 mg/kg) and glibenclaimide (10 mg/kg) on body weight in streptozocin nicotinamide prompted diabetic rodents is portrayed in **figure 3**. Diabetic benchmark group with no medication treatment showed no massive distinction in body weight following 21 days treatment when contrasted with the underlying day treatment. The reduction in body weight of diabetic rodent compares to fat catabolism and protein wastage and furthermore might be because of less accessibility of glucose for energy usage. Notwithstanding, Hesperidin and Apigenin (25 mg/kg and 50 mg/kg) treated diabetic rodent showed a huge ($p< 0.05$) expansion in body weight. The diabetic rodent treated with glibenclaimide (10mg/kg) showed steady and reliable expansion in body weight.



All values represent means ± S.D of the mean (n=6), *p<0.05 vs diabetic control group

Figure 1: Effect of bioactive flavonoids on glucose level in OGTT

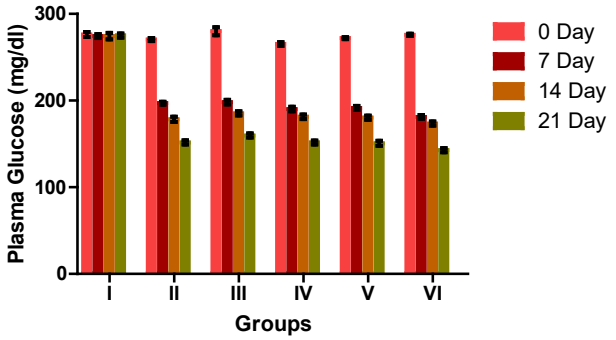


Figure 2: Effect of bioactive flavonoids on glucose level in STZ induced diabetic rats

Effects of Hesperidin & Apigenin on Lipid Levels in Diabetic animals

Following 21 days treatment, treated diabetic rodent with Hesperidin and Apigenin showed huge fall in absolute cholesterol, fatty substances, HDL-C, LDL-C and VLDL-C level at portion of 25 and 50 mg/kg as portrayed in **Table 1**. The standard medication glibenclaimide (10mg/kg) likewise showed fall in all out cholesterol, fatty substances, HDL-C, LDL-C and VLDL-C level when contrasted with control bunch.

Effects of Hesperidin & Apigenin on Urea, Creatinine & Protein Levels in Diabetic animals

The impact of Hesperidin and Apigenin (25 mg/kg and 50 mg/kg) and glibenclaimide (10 mg/kg) on urea, creatinine and protein in streptozotocin-nicotinamide prompted diabetic rodents is displayed in **Table 2**. The standard medication, Hesperidin and Apigenin showed a portion related decrease in serum centralization of urea and creatinine however switch impact on the serum convergence of all out protein.

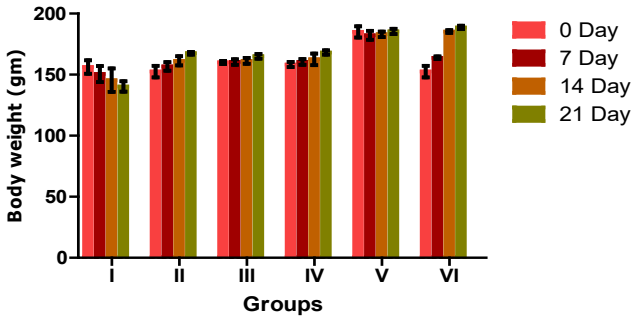


Figure 3: Effect of hesperidin and apigenin on body weight in STZ induced diabetic rats

Table 1: The effect of Hesperidin & Apigenin on serum cholesterol, triglycerides, HDL-C, VLDL-C and LDL-C level in diabetic rats

Groups/Treatment	*Total cholesterol (mg/dl)*	*Triglycerides (mg/dl)*	*HDL-C (mg/dl)*	*LDL-C (mg/dl)*	*VLDL-C (mg/dl)*
Group-I	257.2 ±1.23	298.4 ± 2.23	42.1± 1.27	156.2±1.12	58.7±1.33
Group-II	173.4±1.34***	182.2 ± 2.31***	49.3±2.52**	101.1±1.18**	40.5±1.34*
Group-III	220.3± 1.44**	240.5 ± 1.35**	45.4±1.13**	129.4±1.33**	53.7 ± 2.22*
Group-IV	211.6 ±1.26**	230.7 ± 1.66**	46.3±1.82**	112.2±2.46**	49.6±1.56*
Group-V	202.3 ± 2.36**	212.3 ± 2.81***	46.2±1.04**	103.8±2.67**	46.4±1.78*
Group-VI	191.7±2.67***	201.5 ± 3.46***	47.5±1.74**	101.3±1.43***	42.2±1.11*

All values represent means ± S.D of the mean (n=6), *p<0.05, vs diabetic control group

Table 2: The effect of Hesperidin & Apigenin on Urea, Creatinine & Protein in diabetic rats

Groups/ Treatment	*Urea (mg/dl)*	*Creatinine (mg/dl)*	*Protein (mg/dl)*
Group-I	56.2 ± 1.34	1.71 ± 2.22	3.89 ± 0.44
Group-II	41.3 ± 1.22**	1.44 ± 2.56*	8.22 ± 0.46***
Group-III	50.2 ± 2.46**	1.72 ± 1.14*	4.78 ± 0.22**
Group-IV	47.6 ± 1.74**	1.63 ± 1.78*	6.11 ± 0.66**
Group-V	45.8 ± 2.12**	1.57 ± 2.21*	7.56 ± 1.45**
Group-VI	41.4 ± 2.55***	1.58 ± 2.36*	7.44 ± 3.69**

All values represent means ± S.D of the mean (n=6), *p<0.05, vs diabetic control group

Plant-based slims down are turning out to be more famous with the up and coming age of Europeans, likewise because of the biological viewpoints. Hence, future results of clinical preliminaries ought to show the ideal techniques for bringing restorative plants into the pharmacological treatment of diabetes mellitus and the streamlining of portions and types of plants in blended treatments to stay away from unwanted secondary effects.

Oxidative pressure is a very much perceived factor which includes in pathogenesis of diabetes and its confusions. In addition, movement of microvascular and cardiovascular entanglements of diabetes are well associate with oxidative pressure and receptive oxygen species (ROS). As such; expansion in ROS is a demonstrated system during hyperglycemia which is trailed by enactment of pathways bringing about a few harms in various organs. Oxidation may likewise be related with waterfall prompted by hyperglycemia. In hyperglycemia; cell passing is the outcome of NFκB enactment with hindrance in the guideline of ROS. In view of the kind of cell included, diabetic patients will confront confusions in different organs like liver, kidneys, lung and eyes.

Predictable with this view, our information gives additional proof that there is presence of oxidative pressure with a modification in cell reinforcement protein exercises and expanded lipid peroxidation trial creatures. Diabetic nephropathy (DN) is one of the most significant microvascular inconveniences of diabetes and a significant reason for end stage renal sickness.[11] Numerous pathways have been associated with pathogenesis of DN including oxidative pressure,[12] initiation of protein kinase C,[13] expanded creation of cutting edge glycation finished results (AGE),[14] and polyol hexosamine pathway motion.[15] The outrageous creation of responsive oxygen species (ROS) has been recommended as a typical outcome prompting heightened oxidative harm at the degree of lipid peroxidation[16] and top in DN in relationship with diabetes.[17] Consequently, any treatment that can settle oxygen digestion and manage oxidative pressure can lessen and postpone the improvement of DN.[18-22]

The oral organization of the two flavonoids at a portion from 25 up to 50 mg/kg brought about no clinical indications of intense poisonousness, nor did they impact the body weight of the Swiss pale skinned person rodents. The OGTT is generally used to assess a creature's capacity to control blood glucose. Hesperidin and Apigenin fundamentally forestalled the expansion in blood glucose levels after 60 min. of glucose organization at the portions of 25 and 50 mg/kg. Glibenclamide likewise hindered the expansion in blood glucose levels after 30 min.

The impact of 21 days treatment of Hesperidin and Apigenin and control oral organization on serum glucose level in streptozotocin-nicotinamide (STZ+NIC) actuated diabetic rodents was assessed. Diabetic benchmark group with no medication treatment showed no tremendous distinction in the fasting serum glucose level following 21 days treatment when contrasted with the underlying day treatment. Anyway treated diabetic gatherings showed progressive and steady fall in serum glucose level. A reliable fall in serum glucose level was seen in Hesperidin and Apigenin treated bunches from 21 days dosing and esteems were viewed as huge when contrasted and untreated diabetic benchmark group.

The impact of Hesperidin and Apigenin (25 mg/kg and 50 mg/kg) and glibenclaimide (10 mg/kg) on body weight in streptozocin nicotinamide actuated diabetic rodents was noticed. Diabetic benchmark group with no medication treatment showed no massive contrast in body weight following 21 days treatment when contrasted with the underlying day treatment. The reduction in body weight of Hesperidin and Apigenin treated diabetic rodent compares to fat catabolism and protein wastage and furthermore might be because of less accessibility of glucose for energy usage.

The impact of Hesperidin and Apigenin and glibenclaimide

on serum cholesterol, fatty substances, HDL-C, VLDL-C and LDL-C level in streptozotocin-nicotinamide prompted diabetic rodents was noticed. Following 21 days treatment, treated diabetic rodent with Hesperidin and Apigenin showed huge fall in absolute cholesterol, fatty substances, HDL-C, LDL-C and VLDL-C level at portion of 50 mg/kg. The standard medication glibenclaimide (10mg/kg) additionally showed fall in all out cholesterol, fatty oils, HDL-C, LDL-C and VLDL-C level when contrasted with control bunch.

The impact of Hesperidin and Apigenin and glibenclaimide on urea, creatinine and protein level in streptozotocin-nicotinamide actuated diabetic rodents was noticed. The standard medication, Hesperidin and Apigenin showed a portion related decrease in serum centralization of urea and creatinine however switch impact on the serum convergence of all out protein.

CONCLUSION

The Hesperidin and Apigenin had the antidiabetic action with impacts on lipids and other kidney markers. In the nutshell, we can say that Hesperidin and Apigenin could be an important way to deal with work on the helpful viability, to lessen portion and improvement in measurements routine. Further examinations should be approved in human workers to guarantee for their antidiabetic action.

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