

## The effects of plant extracts of *Medicago sativa* and *Trigonella foenum-graecum* on postprandial glucose levels in type 2 diabetic rats

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### S u m m a r y

The main purpose of this study was to determine if fenugreek (*Trigonella foenum graecum* L.) seeds extract (FSE) and lucerne (*Medicago sativa* L.) extract (LDE) modulate post-challenge carbohydrate metabolism in type 2 diabetes animal model.

Type 2 diabetes was induced in the Wistar rat neonates by intraperitoneal administration of streptozotocin. Experimental animals (60) were divided into following groups: normal rats treated with FSE or LDE, diabetic rats treated with FSE or LDE or glibenclamide or vehicle and given orally examined substances for 4 weeks. Glycaemia was controlled prior to the administration of test substances and at time points of 2 h and 4 h, thereafter at 1<sup>st</sup>, 7<sup>th</sup> and the last day of experiment. Insulin serum concentration was measured at time 0 at the same days.

The present study demonstrates the ability of FSE and LDE to decrease postprandial glycaemia in type 2 diabetic and non-diabetic rats. It seems that this effect mediated by enhancing insulin secretion is related only for LDE-treatment and observed only in n5-STZ rats.

**Key words:** fenugreek seeds, lucerne, postprandial glycaemia, type 2 diabetics rats

## INTRODUCTION

Type 2 diabetes mellitus is a complex syndrome involving deterioration of insulin secretion or decreased sensitivity of peripheral tissues to insulin (insulin resistance) or combination of both [1]. The incidence of this disease increased dramatically in last decades, first of all resulting from the increasing prevalence of obesity [2].

Patients with type 2 diabetes are at increased risk of cardiovascular disease (CVD), which may be partially attributed to a higher prevalence of traditional risk factors (obesity, hypertension, dyslipidaemia). However, factors closely related to diabetes may account for a substantial part of elevated cardiovascular risk [3]. Recently, there are growing evidences for postprandial (post-challenge) glucose as an independent and strong risk factor for CVD, especially myocardial infarction. The pathophysiology of this relationship is not clearly elucidated, but different mechanisms have been suggested (oxidant stress, activated inflammation with endothelial dysfunction, hypercoagulability) [4].

It is proposed that postprandial hyperglycaemia should be a target for treatment in people with diabetes to decrease the risk of CVD morbidity and mortality. Diet, exercise and various pharmacological agents can improve postprandial dysmetabolism. Among dietary changes the use of plant materials has been often considered. More than 400 plants have been documented worldwide as beneficial in the treatment of diabetes [5-7] and have been suggested as a rich, as yet unexplored source of potentially antidiabetic drugs.

Two plants from Leguminosae family: *Trigonella foenum graecum* L. (fenugreek) and *Medicago sativa* L. (lucerne) are known to exhibit hypoglycaemic activity both in animal and human studies [8-11]. Most of these studies were concentrated on the effects of plant extracts on fasting glucose levels and up-to-date no research has been carried out to evaluate the potential of these plants to decrease postprandial glucose level. Moreover, animal studies were conducted in most cases on alloxan- or streptozotocin-induced diabetes with metabolic abnormalities more typical for type 1 than type 2 diabetes in men. Dramatically increased worldwide prevalence of type 2 diabetes is a true challenge for modern medicine. Thus, dietary supplements that can modulate glucose homeostasis would be desirable.

The main purpose of this study was to determine if fenugreek seeds extract and lucerne extract modulate post-challenge carbohydrate metabolism in type 2 diabetes animal model.

## MATERIALS AND METHODS

### Plant material

The plant material was collected in The Garden of Medicinal Plants of the Research Institute of Medicinal, Poznań, Poland.

Dry aqueous extract of lucerne (LDE) – dried and powdered plant material was extracted with water (1:10) for 3 h at 85–90°C. After filtration the filtrate was lyophilised.

Dry 25% alcoholic extract of fenugreek seeds (FSE) – dried and powdered plant material was extracted with 25% ethanol/alcohol (1:10) for 3 h at 55–60°C. After filtration and ethanol/alcohol evaporation the water residue was lyophilised.

## Chemicals and drugs

Glibenclamide and streptozotocin were purchased in Sigma-Aldrich (Sigma-Aldrich Logistic GmbH).

## Animals

Type 2 diabetes was induced in the Wistar rat neonates (male and female) by intraperitoneal administration of streptozotocin (STZ): 80 mg/kg in 0.1 M citrate buffer (pH 5.0) 5 days after birth (n5-STZ rats) according to a previously described protocol [12]. Neonates from control group were injected with 0.1 M citrate buffer only. Newborn rat pups were littered with their mothers until weaning.

All experimental animals were housed in the Department of Pharmacology Animal House and allowed to acclimatize for 7 days in an environmentally controlled room at 22°C with alternating 12 h light/dark cycle. These animals were maintained on normal laboratory chow and water *ad libitum*. Fasted (deprived of food for at least 16 h) animals were allowed free excess to water. The experiments were designed and conducted in accordance with the ethical norms approved by local Ethics Committee guidelines (approval No 39/2004).

The animal model of diabetes used in our experiment was based on partial damage of pancreatic  $\beta$ -cells resulting in administration of STZ in rat neonates. This model of experimental type 2 diabetes provides partial deficits in insulin secretion, peripheral insulin resistance and, in consequence, mild hyperglycaemia [12].

## Experimental procedure

Experimental animals were divided into 6 groups as follows:  
group I (n=13): normal control rats treated with FSE (1 g/kg) in aqueous solution orally for 4 weeks;  
group II (n=10): normal control rats treated with LDE (1 g/kg) in aqueous solution orally for 4 weeks;  
group III (n=10): diabetic control rats (vehicle treated);  
group IV (n=10): diabetic rats treated with FSE (1 g/kg) in aqueous solution orally for 4 weeks;  
group V (n=7): diabetic rats treated with LDE (1 g/kg) in aqueous solution orally for 4 weeks;  
group VI (n=10): diabetic rats treated with glibenclamide (5 mg/kg), hypoglycaemic control substance, in aqueous solution orally for 4 weeks.

Ten-week-old rats were screened in type 2 diabetes model by oral glucose tolerance test (OGTT). For OGTT glucose (1 g/kg) was administered orally to rats that had been fasted for 18 h. Blood samples were collected at 0 and 1 h.

During the experiment blood samples were taken from the tail vein prior to the administration of test substances or the vehicle (fasting conditions) and at time points of 120 and 240 min. (2 and 4 h), thereafter (with free access to food) at 1<sup>st</sup>, 7<sup>th</sup> and the last (28<sup>th</sup>) day of experiment. Mean blood glucose (MBG) concentration was calculated on the base of daily glycaemic profile (0 h, 2 h, 4 h). Insulin serum concentration was measured at time 0 at the same days. Estimation of blood glucose was carried out by using dextrostix with Medisence glucometer (Precision QID Glucometer, 1.1-33.1 mmol/l R). Plasma insulin was assayed from frozen aliquots using enzyme immunoassay using a kit obtained from DRG (rat insulin ELISA, DRG Instruments GmbH, Germany). The sensitivity of the method was 0.07 µg/l. The insulinogenic index was calculated as the ratio of the concentration of glucose/insulin (glik/ins) concentration measured at the same time.

## Statistical analysis

All results were presented as mean values ( $\pm$  SD). Differences between the two groups were statistically analysed by means of Student's t-test (normal distribution) and considered significant by a difference at  $p \leq 0.05$ . The statistical significance of differences among the four groups was assessed using analysis of variance (ANOVA).

## RESULTS AND DISCUSSION

### Characteristic of n5-STZ-induced diabetic rats

Diabetic rats were confirmed by OGTT 10 weeks after STZ injection. Before treatment with plant materials glucose concentrations in the diabetic group were similar to those in the normal control group. However, the glucose levels at 120 min. in OGTT and glik/ins index were higher than those of corresponding normal rats, while fasting insulin levels and body weight were lower (tab. 1).

Table 1.

Characteristic of diabetic (n5-STZ) and control rats

	normal rats (n=20)	diabetic rats (n=38)
body weight [g]	276.5 $\pm$ 45.8	244.2 $\pm$ 39.6*
OGTT		
plasma glucose 0 h [mg/dl]	88.1 $\pm$ 8.9	88.3 $\pm$ 10.9
plasma glucose 1 h [mg/dl]	103.1 $\pm$ 8.8	199.7 $\pm$ 27.8*
plasma insulin [µg/l]	0.54 $\pm$ 0.49	0.38 $\pm$ 0.29
glik/ins	331.2 $\pm$ 191.2	399.0 $\pm$ 182.2

Each value represents mean  $\pm$  SD.

\*significantly different as compared to normal rats,  $p < 0.05$

### Effects of FSE and LDE on glucose and insulin serum levels in non-diabetic rats

Analysis of data from non-diabetic animals showed the fall only in postprandial glycaemia (2 h and 4 h after plant materials administration) after chronic, 4-week-treatment with both LDE and FSE (tab. 2 and 3). This effect was confirmed by analysis of MBG (tab. 3). Observation of fasting insulin levels did not show difference between days of experiment (tab. 4).

**Table 2.**

Effect of FSE and LDE treatment on fasting blood glucose level in normal and n5-STZ diabetic rats

group	fasting blood glucose level [mg/dl]	
	1 <sup>st</sup> day of study	28 <sup>th</sup> day of study
normal + FSE	114.2±7.9 <sup>b</sup>	97.6±12.9
normal + LDE	98.3±8.9 <sup>b</sup>	106.8±6.8
diabetic control	117.2±19.6	115.2±4.8 <sup>a</sup>
diabetic + FSE	119.7±7.7	84.0±7.9 <sup>bc</sup>
diabetic + LDE	93.3±10.3 <sup>b</sup>	95.8±5.7
diabetic + glibenclamide	96.9±14.8 <sup>b</sup>	79.1±18.7 <sup>bc</sup>

Each value represents mean ± SD.

\*statistically significant decrease in comparison to day 1 at p<0.05

<sup>a</sup>in vertical column differs from each other at p<0.05

<sup>b</sup>in vertical column differs from diabetic controls at p<0.05

<sup>c</sup>in vertical column differs from normal+LDE and normal+FSE at p<0.05

**Table 3.**

Effect of FSE and LDE treatment on postprandial glucose level in normal rats

group	postprandial glucose levels [mg/dl]		
	1 <sup>st</sup> day of study	7 <sup>th</sup> day of study	28 <sup>th</sup> day of study
		MBG [mg/dl]	
normal + FSE			
2 h	139.4±10.6	140.4±10.9	114.7±6.7 <sup>*</sup>
4 h	136.8±13.3	131.2±13.2	116.3±3.9 <sup>*</sup>
MBG	124.8±8.1	123.6±9.8	112.6±4.1 <sup>*</sup>
normal + LDE			
2 h	124.5±9.2	120.2±12.7	106.8±8.8 <sup>*</sup>
4 h	114.3±18.3	113.5±15.9	108.5±9.8 <sup>*</sup>
MBG	114.3±10.8	111.9±12.7	104.2±7.3 <sup>#</sup>

Each value represents mean ± SD.

\* statistically significant decrease in comparison to 1<sup>st</sup> and 7<sup>th</sup> day at p<0.05

# statistically significant decrease in comparison to 1<sup>st</sup> day at p<0.05

**Table 4**

Effect of FSE and LDE treatment on fasting insulin plasma level and glik/ins in normal and n5-STZ diabetic rats

group	fasting insulin plasma level [µg/l]		
	study day 1	study day 7	study day 28
		glik/ins index	
normal + FSE	0.71±0.7	0.41±0.11	0.51±0.1
	281.7±141.5	328.3±109.2	196.2±53.3

normal + LDE	0.54±0.49 332.0±242.4	0.59±0.49 218.7±102	0.64±0.46 209.9±237.8
diabetic control	0.26±0.11 457.4±180.3	0.36±0.15 403.1±140.1	0.33±0.15 480.4±37.0
diabetic + FSE	0.36±0.08 354.51±82.5	0.41±0.14 547.7±216.1	0.29±0.08 314.45±102.1
Diabetic + LDE	0.42±0.52 420.8±235.3	0.87±0.57 168.9±114.6*	1.4±1.18 146.4±156.6*
Diabetic + glibenclamide	0.39±0.3 379.7±30.1	0.63±0.52 301.2±271	0.58±0.34 235.6±149.2

Each value represents mean ± SD,

\*statistically significant decrease in comparison to day 1 at  $p < 0.05$

### Effects of FSE and LDE on glucose serum levels in rats with type 2 diabetes (n5-STZ)

Supplementation of aqueous extract of fenugreek seeds (FSE) to n5-STZ diabetic rats for 4 weeks resulted in significant decrease of fasting blood glucose (FBG) level. This effect was similar to that observed in glibenclamide-treated group. There was no reduction in FBG in lucerne- and vehicle-treated diabetic rats. However, it is noteworthy that n5-STZ diabetic rats showed near-normal, comparable to non-diabetic animals, fasting plasma glucose (tab. 2).

Subsequently, the effects of plant extracts on plasma glucose levels 2 and 4 hours after its oral administration were investigated and these time-points as a postprandial glycaemia (animals had free access to food after extracts administration) were considered. The administration of LDE resulted in time-dependent fall in postprandial plasma glucose. The glucose lowering effect of LDE at 2 h after extract administration was observed after chronic treatment (4-weeks) only as compared to the initial level (200.7±46.7mg/dl vs 139.0±37.9 mg/dl). On the contrary, glycaemia controlled 4 hours after LDE administration was significantly decreased after short-time (1-week) and long-time (4-week) supplementation with LDE (241.1±95.5 mg/dl vs 182.7±64.9 mg/dl and 165.2±47.1 mg/dl). However, the effect of chronic, 28-day-treatment with LDE was considerably more pronounced. These results are shown in fig. 1a.

Chronic, 4-week-treatment with FSE was associated with significant decrease in postprandial glycaemia controlled 4 h after plant material administration. Similar results, although of no statistical significance, were also observed at 2 h after administration of FSE (120.6±15.6 mg/dl vs 91.7±16.0 mg/dl). Short-term (1-week) administration of FSE in type 2 diabetic rats was not associated with the decrease in postprandial glucose levels (fig. 1b).

In vehicle-treated diabetic rats both fasting and postprandial plasma glucose were high and unchanged during the experiment (fig. 1c).

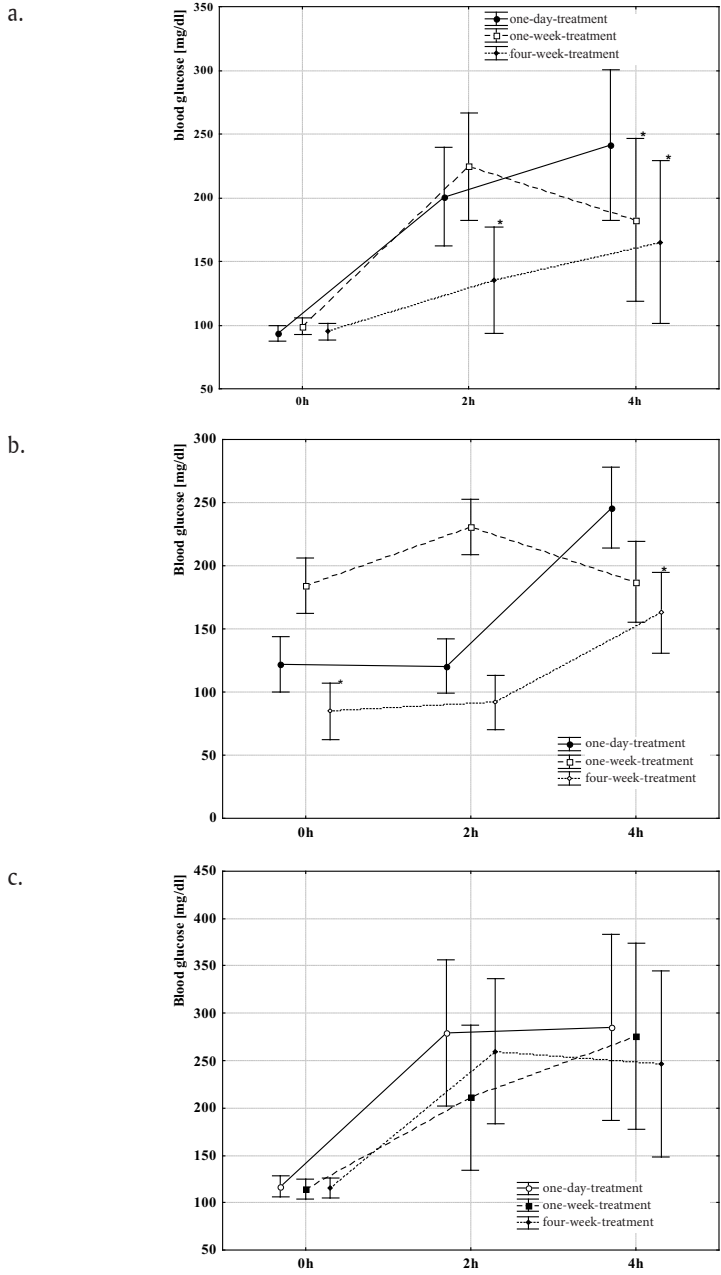


Figure 1.

- a. Effects of LDE on FBG and postprandial glycaemia in type 2 (n5-STZ) diabetic rats. Data are expressed as mean ± SD; \*statistically significant as compared to 1<sup>st</sup> day of treatment at p<0.05.
- b. Effects of FSE on FBG and postprandial glycaemia in type 2 (n5-STZ) diabetic rats. Data are expressed as mean ± SD; \*statistically significant as compared to 1<sup>st</sup> day of treatment at p<0.05.
- c. Fasting and postprandial glycaemia in type 2 (n5-STZ) non-treated diabetic rats.

Using mean paired differences, we compared chronic hypoglycaemic effects of LDE and FSE with standard hypoglycaemic substance, glibenclamide. The fall in plasma glucose concentrations at 2 h and 4 h in diabetic rats treated with LDE or FSE was higher (28<sup>th</sup> day – 1<sup>st</sup> day) than in the glibenclamide-treated group (fig. 2) and pronounced better in FSE-treated animals. This fall of glibenclamide in inhibition of postprandial plasma glucose rise in type 2 diabetic animals was previously reported by Ohnota et al. [13].

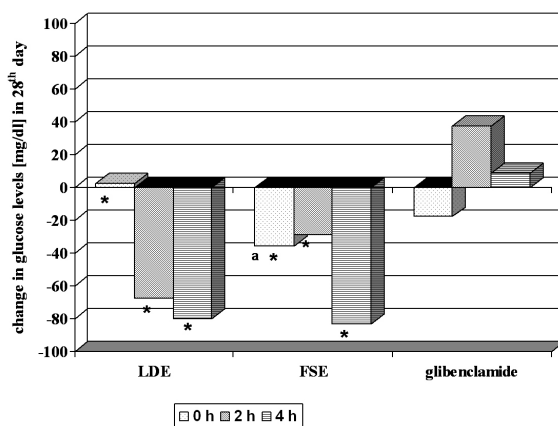


Figure 2.

Comparison of glucose levels with mean paired differences (28<sup>th</sup> day – 1<sup>st</sup> day) in diabetic LDE-treated and FSE-treated groups and glibenclamide-treated group.

\*statistically significant as compared to glibenclamide in treated and non-treated groups

<sup>a</sup>statistically significant as compared to LDE-treated group

The hypoglycaemic effectiveness of both investigated plant extracts was confirmed on the base of MBG analysis. As expected, the significant fall in mean blood concentration was noted after chronic treatment with both LDE and FSE and was similar to the effect exerted by glibenclamide (fig. 3).

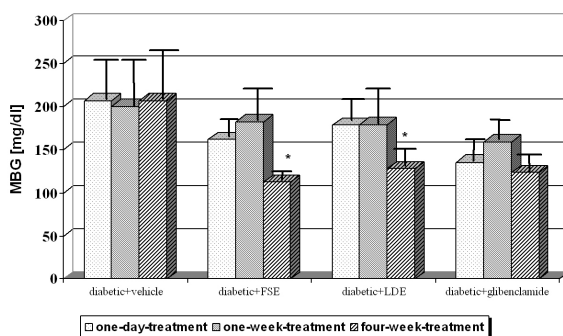


Figure 3.

Effects of FSE and LDE on MBG in type 2 (n5-STZ) diabetic rats. Data are expressed as mean  $\pm$  SD.

\*statistically significant as compared to 1<sup>st</sup> day of treatment at  $p < 0.05$



## Effects of FSE and LDE on insulin serum levels and insulin resistance (glik/ins index) in rats with type 2 diabetes (n5-STZ)

Subsequently, the effects of plants extracts on fasting plasma insulin levels in diabetic rats were investigated. Analysis of data from type 2 diabetic animals treated with LDE showed a tendency of increase in insulin plasma level and significant decrease in peripheral insulin resistance expressed as decrease in mean value of glik/ins index (tab. 4). These results clearly demonstrate insulin-releasing and antihyperglycaemic activity of lucerne extract in n5-STZ rats. Similar effect was observed by Gray et al. [14], who showed lucerne-related enhancement of glucose transport and metabolism in isolated skeletal-muscle preparation and dose-dependent stimulation of insulin *in vitro* in mechanism similar to sulphonylureas derivatives. It should be added that LDE-dependent carbohydrate metabolism normalizing action might be attributed to decrease of glucose absorption [15].

Chronic administration of FSE did not alter the insulin secretion and glik/ins index significantly (tab. 4). Previously, the hypoglycaemic effects of fenugreek have been attributed to several mechanisms. Sauvaire et al. [16] demonstrated *in vitro* 4-hydroxyisoleucine-related (4-hydroxyisoleucine amino acid from fenugreek seeds) increase in glucose-induced insulin release in human and rat pancreatic cells. In our study the clear association between the use of FSE and insulin secretion was not observed. It is highly probable that this effect was attenuated by relatively low fasting glucose levels. Thus, the fall in glucose concentration observed after chronic treatment with FSE may result rather from its antihyperglycaemic than insulinotropic activity. In study undertaken by Vijayakumar et al. [17] it was proved that FSE significantly improved glucose homeostasis in diabetic and in normal glucose-loaded mice by exerting a rapid, dose-dependent stimulatory effect on cellular glucose uptake in the liver and in the adipocytes. This effect was mediated by activating of the tyrosine phosphorylation of IRS-1 (Insulin Receptor Substrate 1) and the p85 subunit of PI-3K (Phosphatidylinositol 3-Kinase) and then by activating insulin signalling pathway. Antihyperglycaemic effect was also observed by Mohammad et al. [18] who proved that administration of *Trigonella* seed powder to alloxan-diabetic rats partially restored the altered (typical for diabetic state) expression of pyruvate kinase and phosphoenolpyruvate carboxykinase along with correction of muscle glucose transporter (GLUT4) distribution. Moreover, the improvement of glucose metabolism may be also mediated by inhibition of carbohydrate digestion and absorption [19]. It seems that fenugreek action reducing peripheral insulin resistance is more useful and adequate for patients with type 2 diabetes with dominating deterioration of insulin action on peripheral tissues.

## CONCLUSION

The present study demonstrates the ability of fenugreek seeds and lucerne extracts to decrease postprandial glycaemia in type 2 diabetic and non-diabetic rats.

It seems that this effect mediated by enhancing insulin secretion is related only for LDE-treatment and observed only in n5-STZ rats.

This antihyperglycaemic activity of fenugreek seeds and lucerne extracts may be useful for type 2 diabetes and especially important for patients with “pre-diabetic” state for diabetes prevention. These patients already manifest abnormalities of glucose metabolism and could benefit from a low risk, inexpensive, food-based intervention. Fenugreek and lucerne are dietary supplements that may hold promise in this regard but further researches are required.

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## REFERENCES

1. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus, provisional report of a WHO consultation. *Diabet Med* 1998; 15:539-53.
2. Wild S, Gorglic G, Gree A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care* 2004; 5:1047-53.
3. Koskinen P, Manttari M, Manninen V, Huttunen JK, Heinonen OP, Frick MH. Coronary heart disease incidence in NIDDM patients in Helsinki Heart Study. *Diabetes Care* 1992; 15:820-25.
4. O'Keefe JH, Bell DS. Postprandial hyperglycemia/hyperlipidemia (postprandial dysmetabolism) is a cardiovascular risk factor. *Am J Cardiol* 2007; 100:899-904.
5. Day C, Bailey CJ. Hypoglycaemic agent from traditional plant treatments for diabetes. *Int Ind Biotech* 1988; 8:5-8.
6. Roman-Ramos R, Flores-Saenz JL, Alarcón-Aguilar FJ. Anti-hyperglycemic effect of some edible plants. *J Ethnopharmacol* 1995; 48:25-32.
7. Li WL, Zheng HC, Bukuru J, De Kimpe L. Natural medicines used in traditional Chinese medical system for therapy of diabetes mellitus. *J Ethnopharmacol* 2004; 92:1-21.
8. Swantston-Flatt SK, Day C, Flatt PR, Gould BJ, Bailey CJ. Glycemic effect of traditional European plant treatments for diabetes. Studies in normal and streptozotocin diabetic mice. *Diabetes Res* 1989; 10:69-73.
9. Abdel-Barry JA, Abdel-Hassan I. A, Al-Hakiem. Hypoglycemic and antihyperglycemic effects of *Trigonella foenum graecum* leaf in normal and alloxan induced diabetic rats. *J Ethnopharmacol* 1997; 58:149-55.
10. Gray AM, Flatt PR. Pancreatic and extra-pancreatic effects of traditional anti-diabetic plant, *Medicago sativa* (lucerne). *Br J Nutr* 1997; 78:325-34.
11. Basch E, Ulbricht C, Kuo G, Szapary P, Smith M. Therapeutic applications of Fenugreek. *Altern Med Rev* 2003; 8:20-7.
12. Blondel O, Bailbe D, Portha B. Relation of insulin deficiency to impaired insulin action in NIDDM adult rats given streptozotocin as neonates. *Diabetes* 1989; 36:610-17.
13. Ohnota H, Kitamura T, Kinukawa M, Hamano S, Shibata N, Miyata H et al. A rapid-and short-acting hypoglycaemic agent KAD-1229 improves post-prandial hyperglycemia and diabetic complications in streptozotocin-induced non-insulin-dependent diabetes mellitus rats. *Jpn J Pharmacol* 1996; 71:315-23.
14. Gray AM, Flatt PR. Pancreatic and extra-pancreatic effects of the traditional anti-diabetic plant, *Medicago sativa* (Lucerne). *Brit J Nutr* 1997; 78:325-34.
15. Gallagher AM, Flatt PR, Duffy G, Abdel-Wahab YHA. The effects of traditional antidiabetic plants on *in vitro* glucose diffusion. *Nutr Res* 2003; 23:413-24.
16. Sauvaire Y, Petit P, Broca C, Manteghetti M, Baissac Y, Fernandez-Alvarez J, Gross R, Roye M, Leconte A, Gomis R, Ribes G. Hydroxyisoleucine: a novel amino acid potentiator of insulin secretion. *Diabetes* 1998; 47:206-10.

17. Vijayakumar MV, Singh S, Chhipa RR, Bhat MK. The hypoglycaemic activity of fenugreek seed extract is mediated through the stimulation of an insulin signalling pathway. *Brit J Pharmacol* 2005; 146:41-8.
18. Mohammad S, Taha A, Akhtar K, Bamezai RN, Baquer NZ. *In vivo* effect of *Trigonella foenum graecum* on the expression of pyruvate kinase, phosphoenolpyruvate carboxykinase and distribution of glucose transporter (GLUT4) in alloxan-diabetic rats. *Can J Physiol Pharmacol* 2006; 84:647-53.
19. Hannan JM, Ali L, Rokeya B, Khaleque J, Akhter M, Flatt PR, Abdel-Wahab YH. Soluble dietary fiber fraction of *Trigonella foenum-graecum* (fenugreek) seed improves glucose homeostasis in animal models of type 1 and type 2 diabetes by delaying carbohydrates digestion and absorption, and by enhancing insulin action. *Br J Nutr* 2007; 97:514-21.

## WPLYW WYCIĄGÓW ROŚLINNYCH *MEDICAGO SATIVA* I *TRIGONELLA FOENUM-GRAECUM* NA POPOSIŁKOWE STĘŻENIE GLUKOZY – BADANIA NA ZWIERZĘCYM MODELU CUKRZYCY TYPU 2

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### Streszczenie

Celem pracy było zbadanie wpływu wyciągów z nasion kozierdaki (*Trigonella foenum graecum* L., FSE) oraz ziela lucerny (*Medicago sativa* L., LDE) na poposiłkową glikemię u szczurów z cukrzycą typu 2.

Typ 2 cukrzycy (n5-STZ) indukowano u noworodków szczurzych poprzez dootrzewnowe podanie streptozotocyny. Badanie przeprowadzono, podając dożyłdkowo wyciągi roślinne (FSE lub LDE) oraz substancje kontrolne (glibenklamid lub *vehiculum*) przez 4 tygodnie zdrowym zwierzętom oraz szczurom z cukrzycą typu 2. Stężenie glukozy badano przed (na czczo) oraz po podaniu badanych substancji (2 i 4 godz. po podaniu) w 1., 7. i 28. dniu doświadczenia. W tych samych dniach badano także stężenie insuliny na czczo.

Wykazano, że stosowanie wyciągu z nasion kozierdaki oraz wyciągu z ziela lucerny jest związane z istotnym obniżeniem glikemii poposiłkowej w badanych grupach zwierząt zarówno z cukrzycą typu 2, jak i zdrowych. Wydaje się, iż efekt ten jest związany ze zwiększeniem sekrecji insuliny wyłącznie w odniesieniu do wyciągu z ziela lucerny.

**Słowa kluczowe:** kozierdaka, lucerna, glikemia poposiłkowa, cukrzyca typu 2 u szczurów