Scholars LITERATURE

La Prensa Medica Argentina

Research Article

DOI: https://doi.org/10.47275/0032-745X-199
Volume 106 Issue 3

# Activity of Exocrine Pancreatic Enzymes in Diabetic Female Rats

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## Abstract

The study designed to evaluate the activity of pancreatic exocrine enzymes in diabetic male rats induced by alloxan. The hyperglycemia was induced in forty-five rats after fasting of the animals for 24 hours by single intraperitoneal (i.p) injection of Alloxan 100 mg/kg B.W., three days after injection fasting blood glucose was measured when the concentration higher than 150 mg/dL, were considered as hyperglycemia/diabetes. A total of sixty adult male rats (45 diabetes and 15 non-diabetes) divided into two groups as follows. The first group serves as control groups (15 animals) will be single i.p injection with distilled water. the second group diabetic groups (45 animals from the first experiment) were subdivided into three subgroups as following (15 for each). Group (G1), Group (G2) and Group (G3) serve as 20, 40- and 60-days diabetic animals respectively. The blood samples collection were taken through cardiac puncture technique from each rat for each period days for measurement the following parameters: (Serum glucose, total protein, insulin, cholesterol, albumin, triglyceride, LDL-C, HDL-C, and VLDL-C) concentration, the rats pancreatic tissue were be taken for measured tissue pancreatic lipase, amylase, and trypsin concentration. The results demonstrate a significant increase in serum glucose concentration and a decrease in serum insulin and total protein in the diabetic group as compared with the control rats'group in all experimental days. The results showed a significant rise in serum total cholesterol concentration within the diabetic group when compared with the control group at day 20 and 60. Meanwhile, a significant increase in serum triglyceride and LDL concentration and a significant decrease in serum HDL concentration within the diabetic group when compared with the control rats group at day 20, 40 and 60. But the serum VLDL concentration depicted a significant increase in the group of diabetic when compared with the control rats group at day 40 and 60.

The value of pancreatic tissue protease activity clarified there was a significant decrease of protease action in the group of diabetic rats when compared with the control rats group on both day 20 and day 60. And a significant decrease in amylase activity in the diabetic groups when compared with the group of control rats in both day 20, 40 and day 60. While the results of pancreatic tissue lipase show there were non-significant changes within the diabetics group when compared with the control group. In conclusion, the exocrine pancreatic function is very frequently and severely altered in Diabetes mellitus male rats and the metabolic disorder effect of Diabetes mellitus was manifested by hyperlipidemic and hypoprotenimic.

**Keywords:** Pancreases; Enzyme; Diabetic; Rats

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**Citation:** Khodair AB, Al-Sharafi NMN (2020) Activity of Exocrine Pancreatic Enzymes in Diabetic Female Rats. Prensa Med Argent, Volume 106:3. 199. DOI: https://doi.org/10.47275/0032-745X-199.

**Received:** February 03, 2020; **Accepted:** February 17, 2020; **Published:** February 21, 2020

## Introduction

Type one of the Diabetes mellitus (T1DM) also known (insulin-dependent Diabetes mellitus), the response of auto-immune showing as a disease of a consequence autoimmune which demolished of beta cells of the pancreas slowly by the body's from own immune system causes destruction in the level of insulin secretion, in the increased in the status of diabetes kind one, the environmental and genetic predisposing factors are great serious, but the accurate connection is yet unknown [1]. The second type of the Diabetes mellitus (T2DM) known as non-insulin dependent Diabetes mellitus results from resistance insulin action and insufficient secretory pancreatic β cells. Also, Diabetes mellitus is a common disease that is foretold to rise in prevalence [2]. T2DM frequently co-habit simply by chance because their rise prevalence and shared risk factors, the obesity and aging is good risk factor for T2DM [3,4]. The Diabetes mellitus may be happen by the present carbohydrates and fat in daily food consumer that starch digestion in mammals is done by α-amylase and clinicians also amylase and α-glucosidase, decrease enzymes of the starch digestive or glucose carrier able to decrease glucose produce and absorption in the small intestine this decrease could help to control Diabetes mellitus [5]. Also, the main causes of Diabetes mellitus restricted by scientists it was genetic factors, environmental agent and another pathological state like autoimmune removal of the pancreatic beta β cells which excite and decrease of insulin and another anomaly which causes resistance to insulin effect appear to include in the development of the disease [6]. A diabetic patient may possess damage to many body systems especially blood vessels, nerve, heart and kidney and abnormality of lipoprotein metabolism [7]. The pancreatic enzymes also have role in the food digestion by producing fluid composed of digestive enzymes into the duodenum these enzymes make break down fat, protein and carbohydrate, approximately (1.5-3) L fluid are secreted every day. The enzyme breaks down proteins are trypsin and chymotrypsin, while the enzymes that digestion fats are lipase, lysophospholipase,

phospholipase and cholesterol esterase ,the carbohydrates and starch are broken down by amylase [8]. Also the cells found in the pancreas play important role in the keep blood sugar level these cells lie in the islet Langerhans are the alpha cells produce glucagon which make raise blood glucose level, beta cells produce insulin which inhibition blood glucose, delta cells secreted somatostatin which inhibition release insulin and glucagon and F cells produce pancreatic polypeptide which inhibition somatostatin [9]. When pancreatic enzyme insufficiency (decrease exocrine enzyme of the pancreas) this lead to cannot maintain normal digestion and cause inadequate digestion especially with fat this causing fat malabsorption which happen when intraduodenal lipase decrease below 5-10% of normal enzyme output this lead to pancreatic steatorrhea, reduce weight and reduce quality of the life [10,11]. Accordingly, the aim of the study and attempt to evaluate the pancreatic enzyme activity and its metabolic complication in diabetic males rats.

## Materials and Methods

Induced of diabetes: Hyperglycaemia was induced in 45 rats after exposure to the fasting of the experimental rats for 24 h by intraperitoneal injection of alloxan for bodyweight 100 mg/kg. Alloxan can of producing hypoglycemia at fatal level which leads to death as a result of large amount pancreatic secretion of insulin to avoid hypoglycemia the rats have then kept of glucose at 5% solution during 24 hours, 3 days after injection, threat with fasting blood glucose higher than 150 mg/dL, were considered as hyperglycemic/diabetes [12]. A total of sixty adult male rats (45 diabetes and 15 non-diabetes) divided into two groups as follows. The first group serves as control groups (15 animals) will be single i.p injection with distilled water. the second group diabetic groups (45 animals from the first experiment) were subdivided into three subgroups as following (15 for each). Group (G1), Group (G2) and Group (G3) serve as 20, 40 and 60 days diabetic animals respectively. The blood samples collection were taken through cardiac puncture technique from each rat for each period days for measurement the following parameters: Serum glucose, insulin, total protein, albumin, triglyceride, cholesterol, HDL-C, LDL-C, and VLDL-C concentration, using semi-automatic chemistry analyzer Belgium using kit Cyan com./Belgium) while insulin used Immunoenzymometric assay The rats pancreatic tissue were be taken for measured tissue pancreatic lipase, amylase and trypsin concentration.rats pancreatic tissue were be taken for measured tissue pancreatic Protease, lipase and amylase concentration. According to rats enzymatic ELISA kit (Rat PRSS1/Protease, Serine, 1/Catalog No: E-EL-R0799); Rat PL/Pancreatic Lipase/Catalog No: E-EL-R2441) and Rat AMY2/Amylase Alpha 2, Pancreatic/Catalog No: E-EL-R2545) respectively. Tissue homogenates: It recommended to getting detailed references from the literature before analyzing different tissue types. For general information, hemolysed blood may be affected by the result, so the tissue should be minced into small pieces and rinsed in ice-cold PBS (0.01M, PH=7.4) to remove excess blood thoroughly. Tissue pieces should be weighed and then homogenized in PBS (tissue weight (g): PBS (ML) volume=1:9) with a glass homogenizer on ice. To further break down the cell, you can sonicate the suspension with an ultrasonic cell disrupter or subject it to freeze-thaw cycles. The homogenates are then centrifuged for 5 min at 5000×g so get the supernatant. Cell culture supernatant or other biological fluids: Centrifuge samples for 20 minutes at 1000×g at 2-8℃. Collected the supernatant to carry out the assay.

### Statistic Analysis

Statistical analysis of the experimental results was conducted according to SPSS version 13.00. The data were expressed as mean±standard errors (SE) and P value<0.05 was considered statistically significant LSD was carried out to test the significant level among means of treatment.

## Results And Discussion

### Rat serum glucose concentration detection

In table 1, the result clarified there was and a significant (P<0.05) increase in serum glucose concentration in a diabetic group as compared with a control group in all experimental days. Also, the results show non-significant (P>0.05) changes within diabetics when compared with the same group in all experimental days.

### Rat serum pancreatic insulin concentration detection

In table 2 illustrates the results of serum insulin concentrations of diabetic and control groups. There was a significant (P<0.05) decrease in serum insulin concentration in the diabetic group as compared with the control group in all experimental days. Also, the results show non-significant (P>0.05) changes within diabetics and control groups when compared with the same group in all experimental days.

### Rat serum total protein concentrations detection

The mean value of serum total protein concentrations of diabetic and control groups illustrated in the table (Table 3). The results showed a significant (P>0.05) decrease in serum total protein concentrations within diabetics groups as compared with significant control groups in days 20, 40 and 60. Also, the results show non-significant (P>0.05) changes within the diabetics group when compared with the same group in all experimental days.

### Rat serum albumin concentrations detection

The results in the table of serum albumin concentration the results show there were non-significant (P>0.05) changes within the diabetics group when compared with the control group (Table 4).

**Table 1:** Effect of Diabetes mellitus on serum glucose (mg/dL).

| Day/Groups | 20 | 40 | 60 |
|---|---|---|---|
| D | 425.81±25.68 aB | 405.81±14.59 aB | 384.87±12.93 aB |
| C | 177.38±46.28 aA | 133.82±6.45aA | 125.55±8.07 aA |
| Sig. (2-tailed) | 0.002 | 0 | 0 |

**Table 2:** Effect of Diabetes mellitus on serum insulin (µU/ml).

| Day/Groups | 20 | 40 | 60 |
|---|---|---|---|
| D | 4.11±0.172 aA | 4.35±0.410 aA | 4.67±0.418 aA |
| C | 13.74±0.625 aB | 14.79±0.737 abB | 15.48±0.708 bB |
| Sig. (2-tailed) | 0.000 | 0.000 | 0.000 |

**Table 3:** Effect of Diabetes mellitus on serum protein (mg/dL).

| Day/Groups | 20 | 40 | 60 |
|---|---|---|---|
| D | 8.92 ±0.77 aB | 10.02±0.39aB | 7.438±0.69 aB |
| C | 13.14 ±0.38 aA | 12.27±0.63aA | 12.91±1.05 aA |
| Sig. (2-tailed) | 0.001 | 0.017 | 0.002 |

**Table 4:** Effect of Diabetes mellitus on serum albumin (mg/dl).

| Day/Groups | 20 | 40 | 60 |
|---|---|---|---|
| D | 2.83±0.27 aA | 2.57±0.18 aA | 2.87±0.28 aA |
| C | 3.24±0.28 aA | 3.09±0.23 aA | 3.57±0.56 aA |
| Sig. (2-tailed) | 0.327 | 0.12 | 0.305 |

## Rat serum lipid profile detection

The below table illustrates the mean value of serum total cholesterol, triglyceride, HDL, LDL and VLDL concentrations respectively (Table 5). The results showed a significant (P<0.05) increase in serum total cholesterol concentration within a diabetic group when compared with the control group at day 20 and 60 while non-significant (P<0.05) changes in after 40 of day experiment. Meanwhile, a significant (P<0.05) increase in serum triglyceride and LDL concentration and significant (P<0.05) decrease in serum HDL concentration within the diabetic group when compared with the control group at day 20, 40 and 60. But the results of serum VLDL concentration depicted a significant (P<0.05) increase in the diabetic group when compared with the control group at day 40 and 60.

## Rat tissue pancreatic protease concentration detection

In table 6 illustrate the mean value of pancreatic protease activity of control and diabetic groups the results clarified there was a significant (P<0.05) decrease of protease activity in diabetic group when compared with control group in both days 20 and day 60 while non-significant (P>0.05) changes between diabetic group and control group in day40. As while as, non-significant (P>0.05) changes in diabetics group when compared with each other in all experimental days.

## Rat tissue pancreatic amylase concentration detection

In table 7 illustrates the effects of diabetic on rats pancreatic amylase activity of control and diabetic groups for 60 days. The results depicted that a significant (P<0.05) decrease of amylase activity in the diabetic group when compared with the control group in both day 20, 40 and day 60.

**Table 5:** Effect of Diabetes mellitus on serum lipid profile.

| Effect of Diabetes mellitus | Day/Groups | 20 | 40 | 60 |
|---|---|---|---|---|
| **Serum cholesterol** | D | 121.72±2.30 bB | 97.62±4.93 aA | 109.94±5.90 abB |
| | C | 77.07±3.96 aA | 73.59±4.85 aA | 67.46±5.00 aA |
| | Sig. (2-tailed) | 0 | 0.008 | 0.001 |
| **Serum triglyceride** | D | 38.51±4.71aA | 83.95±6.45bB | 142.54±8.08 cB |
| | C | 33.68±1.53aA | 28.78±2.05 a A | 37.52±5.67 aA |
| | Sig. (2-tailed) | 0. 359 | 0 | 0 |
| **Serum HDL-C** | D | 15.06±0.23 bA | 13.23±0.39 aA | 12.37±0.36 aA |
| | C | 18.15±0.19 aB | 18.66±0.42 aB | 18.05±0.21 aB |
| | Sig. (2-tailed) | 0 | 0 | 0 |
| **Serum VLDL-C** | D | 7.70±0.94 aA | 16.79±1.29 bB | 28.50±1.61 cB |
| | C | 6.73±0.30 aA | 5.75±0.41aA | 7.50±1.13 aA |
| | Sig. (2-tailed) | 0.359 | 0 | 0 |
| **Serum LDL-C** | D | 98.95±2.77 b A | 67.59±4.30 aA | 69.05±6.04 aA |
| | C | 52.17±3.76 aB | 49.17±4.92 aB | 41.90±6.05 aB |
| | Sig. (2-tailed) | 0 | 0.023 | 0.013 |

**Table 6:** Effect of dibetes mellitus on pancreatic protease activity (ng/ml).

| Day/Groups | 20 | 40 | 60 |
|---|---|---|---|
| D | 0.231±0.004 aA | 0.249±0.0079 aA | 0.234±0.0072 aA |
| C | 0.279±0.004 aB | 0.265±0.0052 aA | 0.271±0.0075 aA |
| Sig. (2-tailed) | 0 | 0.145 | 0.008 |

**Table 7:** Effect of dibetes mellitus on pancreatic amylase activity (ng/ml).

| Day/Groups | 20 | 40 | 60 |
|---|---|---|---|
| D | 5.71 ±0.24 aA | 6.67±0.26 aA | 6.68±0.42 aA |
| C | 9.49±0.34 aB | 9.56±0.54 aB | 9.69±0.43 a B |
| Sig. (2-tailed) | 0 | 0.001 | 0 |

## Rat tissue pancreatic lipase concentration detection

In table 8 illustrates the mean value of pancreatic lipase of control and diabetic groups the results show there were non-significant (P>0.05) changes within the diabetics group when compared with the control group. Also, non-significant (P>0.05) changes between the diabetic group and the control group when compared with each other.

**Table 8:** Effect of Diabetes mellitus on pancreatic lipase activity (pg/mL).

| Day/Groups | 20 | 40 | 60 |
|---|---|---|---|
| D | 235.58±0.03 aA | 235.70±0.044 aA | 235.68±0.073 aA |
| C | 235.59±0.03 aA | 235.65±0.086 aA | 235.62±0.067 aA |
| Sig. (2-tailed) | 0.778 | 0.61 | 0.508 |

Where: μ±SE; C=Control groups; D=Diabetic groups; Capital letters donate differences between groups, P<0.05; Small letters donate differences within groups>0.05.

Insulin responsible for regulates glucose levels in the blood, supply glucose to the cells of bodies to absorb and use glucose, when glucose unable to enter the cells of the body cannot be used as a source of energy for the body's activities and cause elevated blood glucose level [13]. Reduce or resistance of insulin in the body caused in decreases in tissue uptake of glucose that result reduction in glucose concentration in intracellular and increase extracellular, reduce glucose in cell causes increase pathway of glycogenolysis and gluconeogenesis that result increase fats breakdown, this make diabetic ketoacidosis and decrease protein manufacture and gamma globulins which causing many effects include cachexia, frequent water intake, whereas increase glucose extracellular cause hyperglycemic coma and osmotic dieresis and increase digestion of protein [14], the results of research on the human including indirect role tests are shown, on average 51% (26-74%) of patients for non-insulin-dependent Diabetes mellitus and 32% (28-36%) of patients with non-insulin-dependent Diabetes mellitus showed abnormal exocrine activity [15]. Insoluble animal collagen is said to suffer a series of changes in post-maturity like resistance to collagenase digestion resistance to collagenase was also noticed to be strongly marked in collagen from individuals with Diabetes mellitus, our data indicate that there's also a rise in collagen with age and diabetes glycosylation [16].

Insulin reduces breakdown triglyceride in adipose tissue by decrease an intracellular lipase that hydrolyzes triglyceride, so that insulin increase anabolism lipid and fat-sparing influence, therefore, insulin drive more cells to use carbohydrates for energy expenditure instead of fatty acids, also Insulin have profound influence on lipoprotein lipase activity, acetyl-CoA carboxylase and synthesis of fatty acid, insulin promote uptake of amino acids by intracellular and stimulated protein synthesis, also its stimulate the permeability of more cells to ( k, mg, and p) ions [17]. Metabolism of lipids especially in type two Diabetes mellitus is affected by several factors involve a degree of glucose control and insulin resistance is the principal pathophysiological mechanism of diabetic dyslipidemia [18].

In medical diagnosis, the amylase in blood serum is measured which when higher than normal average concentration in can act as a signal for one of many medical disease and conditions, that may be macroamylasemia, perforated peptic ulcer, acute pancreatic inflammation, mesenteric ischemia, strangulation, ileus, macroamylasemia, and mumps. through other bodies, fluids such as peritoneal fluid or urine can measure the levels of amylase in animals [19].

Pancreatic exocrine insufficiency is a common occurrence in type 1 and type 2 Diabetes mellitus there are also frequent changes in exocrine pancreatic morphology in diabetic patients, many hypotheses attempt to explain these findings involving insulin deficiency as a trophic factor for exocrine tissue, alteration in the production and action of other islet hormones, and autoimmune against prevalent endocrine and exocrine antigens, Diabetes mellitus may also result from underlying pancreatic illnesses (e.g. chronic pancreatitis), as a result of diabetic neuropathy, another pathophysiological idea proposes the functional and morphological changes, decades earlier pancreatic study organizations showed that pancreatic exocrine deficiency is found in a significant proportion of patients with Diabetes mellitus, these early trials were conducted by direct pancreatic activity exams (e.g: secretin-pancreozymin test) [20]. The concentrations of serum amylase and lipase are used as pancreatitis diagnostic markers, many of these other one causes may have abdominal symptoms such as pancreatitis, however, a host of other triggers that can lead to a substantial increase in these two blood parameters [21]. Many non-pancreatic intraabdominal pathologies can be cause increase level serum amylase and lipase, the cause of increased lipase in diabetic ketoacidosis is not known it may be pathogenic factors that involve decrease the renal clearance of lipase, cellular stress, pancreatic hypoperfusion and non-pancreatic produce of these enzymes [22]. Another theory is that endocrine pancreatic insulin in diabetes that "spillover" into exocrine pancreas with pancreatic enzymes released into the blood [22]. The exocrine acinar cells of the pancreas secretion induce many forms of enzymes involving amylase and lipase that allow the role of amylase in the digestion of food particles to break down starch into maltose malt triose and reduce dextrins in the digestion. Lipase comes from the pancreas, moves through the intestine and also cleaves triglycerides into fatty acids and monoglycerides [23]. For many years, estimation of the level of serum amylase indicates diffuse pancreatic damage because of advanced pancreatic disease, but many studies seem to correlate this low level of serum amylase with metabolic syndrome and Diabetes mellitus [24]. Ata N, et al. (2015) examining the effect of increased blood glucose on pancreatic exocrine functions and reported that serum amylase and lipase levels rise significantly with glycemic control regulation, but still lower than command [25-27].

In conclusion, the exocrine pancreatic function is very frequently and severely altered in Diabetes mellitus male rats and the metabolic disorder effect of Diabetes mellitus was manifested by hyperlipidemic and hypoprotenimic.

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