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Influence of Diabetes on Endometrial Gland Function and Early Embryonic Nutrition in Rats

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Abstract
This study examined how streptozotocin-induced diabetes alters the carbohydrate composition of endometrial gland secretions during early pregnancy in rats. Four groups of adult female Wistar Albino rats weighing 250 to 300 g, with eight animals per group, were studied: diabetic pregnant, diabetic non-pregnant, non-diabetic pregnant, and non-diabetic non-pregnant control. Lectin histochemistry using Peanut Agglutinin (PNA) specific for β-galactose, Wheat Germ Agglutinin (WGA) for β-N-acetylglucosamine and sialic acid, Concanavalin A (Con A) for α-mannose, α-glucose and α-N-acetylglucosamine, and Soy Bean Agglutinin (SBA) for α-β N-acetylgalactosamine was performed on uterine tissue collected on days five and seven of pregnancy or after cervical stimulation. Diabetic pregnant rats showed positive binding for Con A, PNA and SBA on day five with diminished WGA staining. By day seven this group maintained Con A and PNA positivity while SBA remained positive and WGA stayed weak. Non-diabetic pregnant rats exhibited Con A, WGA and SBA binding on day five but lacked PNA staining; day seven samples showed only Con A and weak SBA positivity. Control animals displayed inconsistent lectin binding across time points. Conventional histochemical stains including Periodic Acid-Schiff, Alcian Blue at pH 1.0 and 2.5, and aldehyde fuchsin produced no detectable staining in any group. Diabetes fundamentally alters the glycosylation profile of endometrial gland secretions, particularly inducing β-galactose expression detected by PNA exclusively in diabetic animals. These changes may compromise histiotrophic nutrition during the critical pre-implantation and early post-implantation periods.

Keywords: Diabetes mellitus; Endometrial glands; Lectin histochemistry; Early pregnancy; Rat model; Uterine secretions.

1. Introduction

The field of glycobiology has emerged as pivotal in studies related to cellular communication, differentiation, and tissue function through glycoproteins with oligosaccharides attached. The carbohydrates are responsible for controlling the process of protein folding, stability, intracellular transport, ligand binding, and cell-cell interaction. Carbohydrate biosynthesis that takes place in the endoplasmic reticulum and Golgi bodies is crucial for maintaining normal physiological function. Changes in glycosylation patterns have been shown to play critical roles in various diseases such as metabolic diseases like diabetes mellitus. Lectins are carbohydrate-binding proteins with unique specificity for sugars and thus help characterize the presence of carbohydrates in tissues.

Properly glycosylated proteins and secretions are extremely important in the female reproductive system. Glycoproteins are involved in many physiological processes during reproduction from gamete recognition to fertilization, implantation, and embryogenesis. Zona pellucida glycoprotein subunits ZP1, ZP2, and ZP3 are responsible for oocyte-sperm recognition and fertilization, and ZP3 functions as a receptor for sperm. After fertilization, conceptuses require a nourishing uterus for the development process before the establishment of placentation. This stage of conceptus development, referred to as histiotrophic nutrition, involves the secretion of a large variety of substances by endometrial glands, including glycoproteins, lipids, growth factors, cytokines, and transport proteins (Hempstock et al., 2004; Filant & Spencer, 2014). Experimentally, researchers have been able to prove that uterine glands are absolutely necessary for implantation and embryo development (Filant & Spencer, 2013; Dhakal et al., 2021).

Diabetes mellitus refers to a persistent disorder marked by high blood glucose levels, insufficient or ineffective insulin, and disruptions of metabolism in cells. Diabetic women tend to have decreased fertility and pregnancy problems as well as poor implantation and negative effects on the baby (Andlib et al., 2024). Animal experiments in diabetic females have confirmed that there is reproductive dysfunction as well as negative impacts on fetal development (Giavini et al., 1986; Kiss et al., 2009; Damasceno et al., 2014). The administration of streptozotocin (STZ) to rats in order to induce diabetes mellitus has become one of the most popular experimental animal models in order to research the pathophysiological mechanisms of diabetes due to its ability to cause destruction of pancreatic β-cells and maintain hyperglycemia, similar to that seen in type 1 diabetes mellitus (Akbarzadeh et al.,

2007). This animal model has found wide application for the assessment of reproductive problems, abnormal fetal growth, and developmental disorders caused by maternal diabetes (Damasceno et al., 2014; Wang et al., 2018).

Diabetes causes serious disruption to the uterine milieu, which might negatively affect the process of embryo development even prior to the onset of normal placentation. Earlier research has shown increased embryonic resorption, growth delay, congenital abnormalities, and reduced fetal survival rate among diabetic pregnancies (Giavini et al., 1986; Giavini et al., 1993; Damasceno et al., 2014). Histological studies revealed such tissue-specific lesions in the diabetic uterus as thickened basement membrane, atrophic and/or dystrophic epithelium, fatty cell accumulation, disturbed vascularization, and endometrial regression. These changes indicate that diabetes could hinder the secretory function of the uterine glands and, thus, result in poor histiotrophic nutrition of the developing embryo. Nevertheless, although the impact of diabetes on the morphology of uterine structures has already been well-documented, relatively little is known about how diabetes affects the biochemical structure and glycosylation pattern of glandular secretions.

Glycosylation being extremely susceptible to changes in the metabolic state of cells, chronic hyperglycemia might affect protein glycosylation processes leading to impaired secretion of proteins with altered carbohydrate content that might be crucial for the development and subsequent attachment of the embryo. Recently, evidence has been provided showing that metabolic disorders such as diabetes and obesity negatively influence reproductive organs in connection with oxidative stress, inflammation, disrupted cellular communication, and altered secretory functions (Andlib et al., 2024; Pavlović et al., 2025). However, the effect of diabetes on the carbohydrate content of the endometrial glands during early pregnancy has not been sufficiently studied yet.

Lectin histochemistry is an effective method of studying carbohydrate content in the cells since it is possible to detect alterations in carbohydrate chains using it. The information obtained from the analysis of carbohydrate chains will help us discover functional alterations in secretory function which are not seen using regular histological methods.

Thus, the current research intends to examine the impact of diabetes resulting from streptozotocin administration on endometrial glands secretion activity and early embryo nutrition in pregnant rats through lectin histochemistry. Using lectin histochemistry, the current investigation will compare the glycosylation patterns of endometrial glands secretions of diabetic and normal rats in order to determine if any reproductive disorder linked to diabetes arises due to the change in biochemical environment during early pregnancy.

2. Literature Review

Diabetes during pregnancy is an important metabolic disease which has a negative effect on fertility and pregnancy. The experimental rat model using streptozotocin (STZ)-induced diabetes is a widely studied approach to evaluate the effects of high blood sugar levels on reproductive organs since it shows a variety of similarities to human diabetic pregnancies such as oxidative stress, insulin deficiency, embryonic loss, and fetal anomalies (Akbarzadeh et al., 2007; Damasceno et al., 2014). While previous researches were mainly concerned about fetal abnormalities and placenta dysfunction, new research findings have shown that diabetes can affect the early stages of pregnancy by compromising the uterine environment necessary for embryo survival and implantation.

Preimplantation development of the conceptus is possible only due to the secretions produced by the endometrial glands. They produce a combination of nutrients, growth factors, cytokines, and other substances that are necessary for the growth and development of the conceptus during this period (Hempstock et al., 2004). In recent years, the endometrial glands are seen not as a passive source of such secretions but rather as regulators of receptivity, implantation, and decidualization processes (Filant & Spencer, 2014). Several experiments have shown that problems with gland development or functioning negatively affect these processes (Filant & Spencer, 2013; Dhakal et al., 2021).

Metabolic derangements resulting from diabetes might affect endometrial glands through various pathways like oxidative stress, inflammation, vasculature change, and hormonal imbalances. Several structural changes in the uterus affected by diabetes have been documented in pathologic studies such as loss of integrity of the epithelium, decreased glandular functioning, and inadequate endometrial development, which can result in a decrease in histotrophic secretion needed for early pregnancy (Falkay et al., 2007; Amira et al., 2011). Also, the presence of diabetes has been linked to embryonic resorption, poor embryonic development, and low fetal survival rate due to adverse effects on the maternal–embryonic interface prior to placenta development (Giavini et al., 1986; Kiss et al., 2009).

The latest research has shown that diabetes and other metabolic diseases affect female fertility through a similar pathological mechanism involving mitochondrial dysfunction, inflammation, insulin resistance, and disruptions in reproductive hormonal signaling (Andlib et al., 2024; Pavlović et al., 2025). Nevertheless, there are very few studies focusing on the effect of diabetes on endometrial glands and their contribution to the nutrition of preimplantation and early post-implantation embryos. Thus, the importance of dysfunctional endometrial glands in the pathology of embryopathic changes during diabetes is currently unknown. Further research into the impact of diabetes on uterine glands can help us better understand this problem.

Notwithstanding the existing knowledge that diabetes leads to pathological changes in endometrial structures, affecting the development of the embryo, there is no research that explores systematically how the pathological conditions affect the composition of carbohydrates in the secretory products of endometrial glands during the early

stages of pregnancy. The existing body of research only explores changes at the protein level as well as the structural pathologies without any information on the patterns of glycosylation. Moreover, the time-dependent nature of the changes was not explored by previous research either.

3. Methodology

Experimental design. Thirty-two adult female Wistar Albino rats weighing 250 to 300 g were divided into four groups with eight animals each. Group one (A) comprised diabetic pregnant rats, group two (B) diabetic non-pregnant rats, group three (C) non-diabetic pregnant rats, and group four (D) non-diabetic non-pregnant controls (Figure 1). Animals were housed four per cage under standard conditions at 21 to 23 °C with a 12-hour light-dark cycle and had free access to standard chow and tap water.

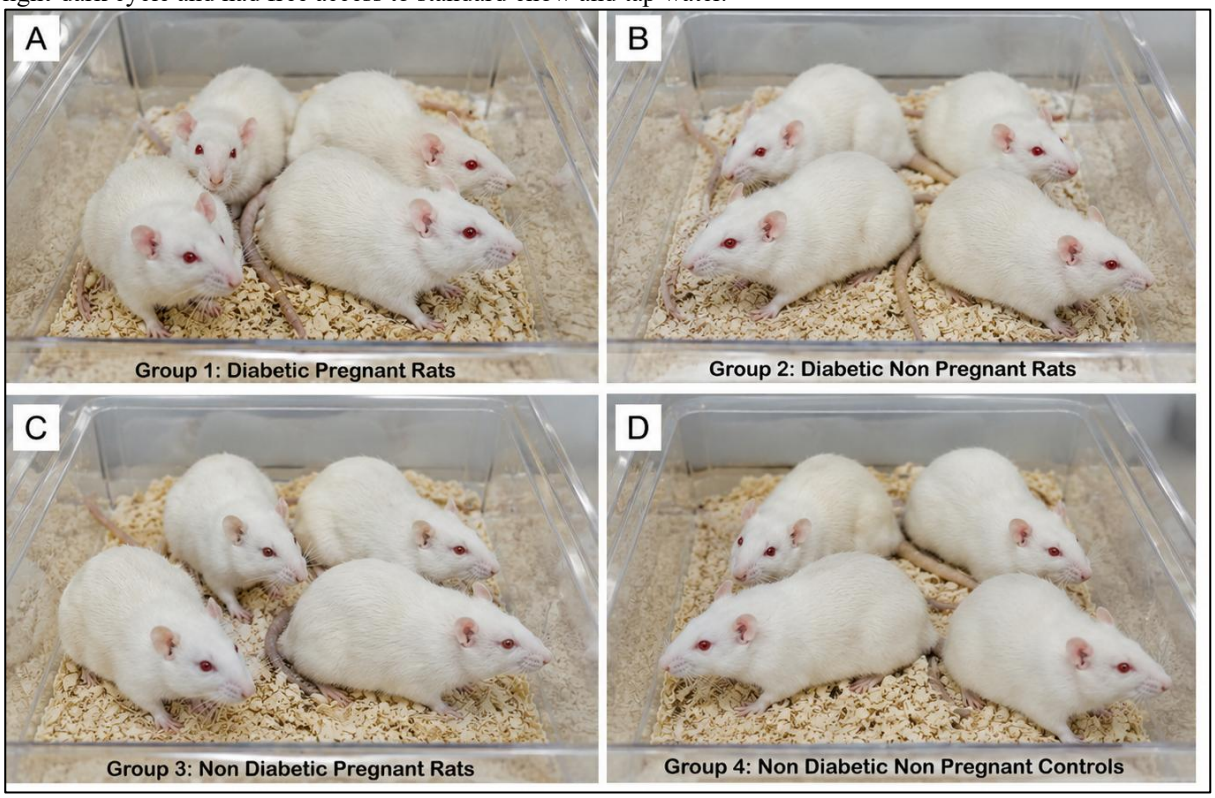

Figure 1. Experimental grouping of adult female Wistar Albino rats used in the study.

Oestrus cycle monitoring and mating. Vaginal smears were collected daily, fixed with methanol for one minute, stained with Giemsa for 30 minutes and examined under light microscopy. Females identified as being in oestrus were placed with males at a ratio of three to four females per male overnight during the dark cycle. The presence of spermatozoa in vaginal smears the following morning confirmed mating, which was designated as day one of pregnancy. For non-pregnant groups, females were stimulated with a glass rod to mimic mating conditions, and tissue collection was performed five and seven days after stimulation.

Diabetes induction. Diabetes was induced in groups one and two by intraperitoneal injection of freshly dissolved streptozotocin at a dose of 45 mg/kg in physiological saline. Blood glucose levels were measured from tail vein samples 48 hours after injection, and animals with glucose exceeding 200 mg/dL were considered diabetic.

Tissue collection and processing. On days five and seven of pregnancy for pregnant groups or after cervical stimulation for non-pregnant groups, rats were anaesthetised with a ketamine-xylazine mixture at 80 to 12 mg/kg. Uterine horns were removed following a midline incision, and animals were sacrificed by cervical dislocation. All procedures complied with the institutional animal ethics committee guidelines. Tissues were fixed in buffered formalin prepared by dissolving 4 g of sodium dihydrogen phosphate monohydrate and 6.5 g of disodium hydrogen phosphate dihydrate in 900 mL of distilled water with 100 mL of 37% formaldehyde. Fixed tissues were processed through graded alcohols and xylene, embedded in paraffin and sectioned at 5 μm thickness.

Histological and histochemical procedures. Crossman's modified triple staining technique was used to visualise general uterine architecture. Conventional histochemical stains included Periodic Acid-Schiff for neutral mucins, Alcian Blue at pH 2.5 for acid mucopolysaccharides, Alcian Blue at pH 1.0 for sulphated mucopolysaccharides, and aldehyde fuchsin for sulphomucins or sulphated proteoglycans.

Lectin histochemistry. For biotinylated lectins including PNA, WGA and Con A obtained from Sigma Aldrich, sections were deparaffinised, rehydrated and treated with hydrogen peroxide in 80% ethanol for 30 minutes to block endogenous peroxidase activity. After washing three times with phosphate-buffered saline at pH 7.2, sections were incubated with serum block solution for ten minutes. Lectins were diluted 1:50 in phosphate-buffered saline containing 1% bovine serum albumin, applied to sections for 60 minutes, followed by biotinylated secondary

antibody for 20 minutes, enzyme conjugate for ten minutes and AEC chromogen for ten minutes. Counterstaining used Mayer's haematoxylin. For peroxidase-labelled SBA, the same protocol was followed without the secondary antibody step. Staining intensity was scored as negative, weak positive, positive or strong positive.

4. Results

General histological findings. Crossman's triple staining revealed structural differences between groups. In diabetic pregnant rats on day five, endometrial glands appeared histologically normal. By day seven, however, generalised uterine disruption was evident, particularly affecting connective tissue architecture. Diabetic non-pregnant day five specimens showed glandular structural disorganisation despite otherwise normal uterine appearance. By day seven this group developed pronounced connective tissue degeneration. Non-diabetic pregnant rats and controls maintained normal histology at both time points. Stromal cell dissociation, basal membrane thickening, pyknotic nuclei in glandular epithelium, vacuolisation and intracellular lipid accumulation were observed only in diabetic animals.

Conventional histochemistry. No positive staining resulted from Periodic Acid-Schiff, the Periodic Acid-Schiff and Alcian Blue combination, Alcian Blue at pH 2.5, Alcian Blue at pH 1.0, or aldehyde fuchsin procedures in any experimental group.

Lectin histochemistry. Table 1 presents the experimental groups and their diabetic and pregnancy status. Table 2 summarises the semi-quantitative lectin staining scores for endometrial glands across groups and time points.

Table 1. Experimental groups and conditions

Group	Designation	Diabetes (STZ)	Pregnancy	Day of tissue collection
1	Diabetic pregnant	Yes	Yes	5 and 7
2	Diabetic non-pregnant	Yes	No	5 and 7 (post-stimulation)
3	Non-diabetic pregnant	No	Yes	5 and 7
4	Control	No	No	5 and 7 (post-stimulation)

Table 2. Lectin histochemistry results for endometrial glands

Group	Day	Con A	PNA	WGA	SBA
Diabetic pregnant	5	+	+	+/-	+
Diabetic pregnant	7	+	+	+/-	+
Diabetic non-pregnant	5	+/-	+	–	++
Diabetic non-pregnant	7	+/-	+	–	+
Non-diabetic pregnant	5	+	–	+	+
Non-diabetic pregnant	7	+	–	–	+/-
Control	5	+/-	–	–	–
Control	7	+	–	+/-	+

Scoring: (–) negative, (+/–) weak positive, (+) positive, (++) strong positive.

In diabetic pregnant rats on day five, endometrial glands showed positive PNA staining localised to apical cytoplasm, positive SBA, weak positive WGA and positive Con A. On day seven, Con A showed intracytoplasmic positivity, PNA stained both luminal contents and some intracellular compartments, SBA labelled apical cytoplasm, and WGA remained weak positive.

Diabetic non-pregnant day five samples exhibited weak luminal Con A staining, strong PNA positivity in both lumen and glands, strong SBA binding in glands, and negative WGA in glands despite positive luminal staining. By day seven, Con A binding was weak, PNA remained positive in lumen and gland lumens, SBA-stained glands positively but not the lumen, and WGA showed no glandular staining with weak luminal positivity.

Non-diabetic pregnant day five specimens showed positive Con A in glandular secretions, negative PNA, and positive WGA and SBA. On day seven, Con A remained positive, PNA stayed negative, SBA showed weak positivity, and WGA was negative.

Control day five samples exhibited weak Con A positivity inconsistently across specimens, negative PNA in glands with positive luminal staining, weak SBA in glands with positive lumen, and negative WGA in glands with weak luminal positivity. On day seven, Con A-stained glands positively with strong luminal binding, PNA was negative in both compartments, SBA showed positivity in glands and lumen, and WGA remained negative.

Figure 1 summarises the key lectin binding patterns on day five of pregnancy or post-stimulation. Panel A shows diabetic pregnant rats with PNA-positive staining in glandular epithelium. Panel B shows diabetic non-pregnant rats with strong PNA positivity in glands and uterine lumen. Panel C shows non-diabetic pregnant rats with negative PNA binding. Panel D shows control animals with negative PNA binding but positive Con A in the lumen. Scale bars are 20 µm in panels A and C and 100 µm in panels B and D. PNA positivity appears exclusively in diabetic groups regardless of pregnancy status, while non-diabetic animals show no PNA binding. In control animals, weak Con A staining in the lumen but not in glands indicates that α-mannose and glucose residues are present in luminal fluid but not concentrated in glandular epithelium at this time point.

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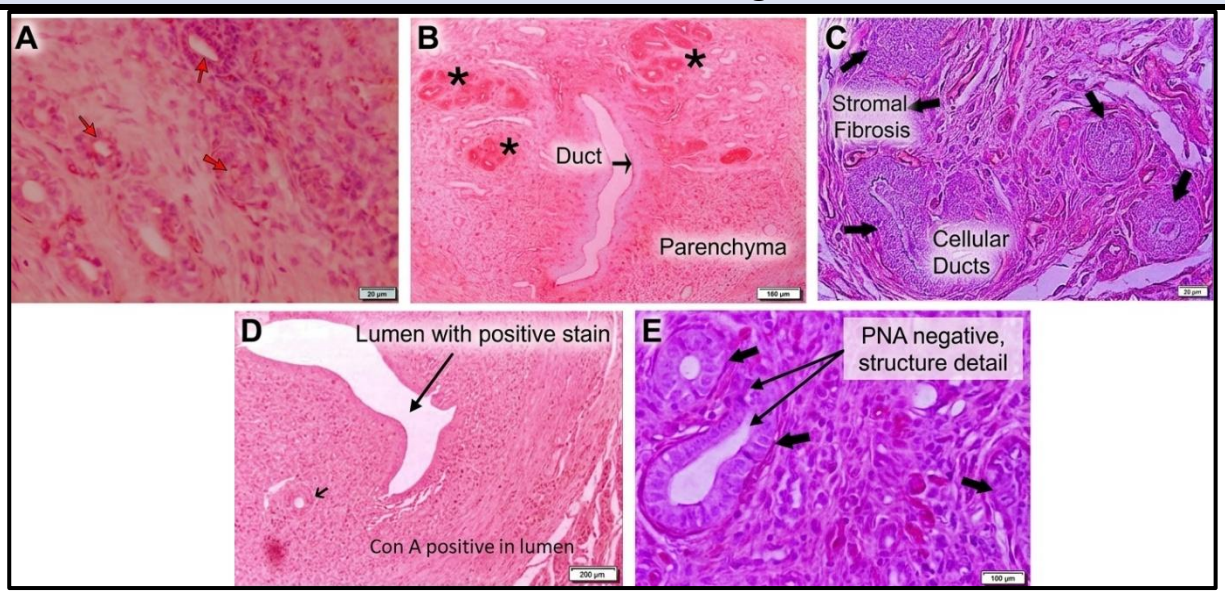

Figure 1. Representative PNA lectin histochemistry of endometrial glands on day 5. (A) Diabetic pregnant rat (Group 1): positive staining in glandular epithelium (arrows). (B) Diabetic non-pregnant rat (Group 2): strong PNA positivity in glands (stars) and uterine lumen (arrow). (C) Non-diabetic pregnant rat (Group 3): no PNA binding (arrows). (D& E) Control rat (Group 4): no PNA binding in glands or lumen. Scale bars = 20 µm (A, C) and 100 µm (B, D& E).

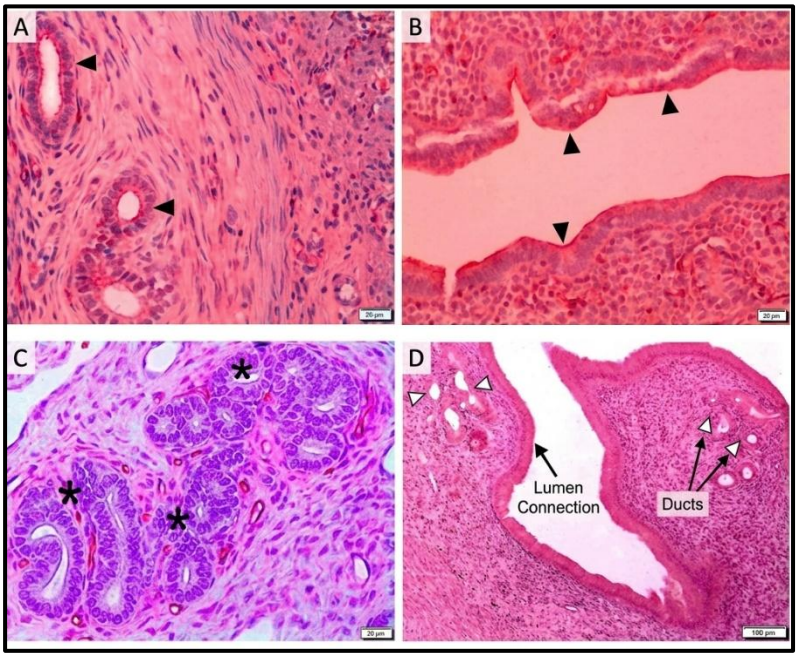

Figure 2. Representative lectin histochemistry of endometrial glands on day 7. (A) Diabetic pregnant rat (Group 1): persistent PNA positivity in glands (arrows). (B) Diabetic non-pregnant rat (Group 2): PNA binding in glands (star) and uterine lumen (thick arrow). (C) Non-diabetic pregnant rat (Group 3): negative PNA binding in glands (stars). (D) Control rat (Group 4): positive SBA staining in glands (arrowhead) and lumen (arrow). Scale bars = 20 µm (A, C) and 100 µm (B, D).

Figure 2 shows the corresponding patterns on day seven. Panel A shows diabetic pregnant rats with persistent PNA positivity in glands. Panel B shows diabetic non-pregnant rats with PNA binding in glands and uterine lumen. Panel C shows non-diabetic pregnant rats with negative PNA but positive Con A. Panel D shows control animals with positive SBA in glands and lumen. Scale bars are 20 µm in panels A and C and 100 µm in panels B and D. By day seven, diabetic animals continue to show strong PNA positivity, confirming that diabetes-associated β-galactose exposure is stable over the early post-implantation period. Non-diabetic pregnant rats remain PNA-negative but retain Con A binding, indicating that high-mannose type N-glycans are preserved in normal pregnancy. Control animals show SBA positivity on day seven, suggesting that α-β N-acetylgalactosamine expression appears later in the cycle even without pregnancy.

5. Discussion

The main conclusion that can be drawn from the results of this experiment is that diabetes leads to an evident alteration in the lectin binding pattern of rat endometrial gland cells, mainly due to the development of PNA positivity, which corresponds to β-galactose expression. Unlike all other groups not suffering from diabetes, this alteration in cell glycosylation took place on day 5 and day 7 of pregnancy and in non-pregnant diabetic rats. As a result, it can be concluded that the described phenomenon is the result of lasting effects of diabetes on cell functioning and metabolism, while not the temporary effect caused by the hormonal changes during pregnancy.

Positive PNA labeling occurred only in diabetic rats during both experimental stages. Since the lack of any PNA staining was noted in all control groups, it can be speculated that diabetes triggers the biosynthesis of β-galactosylated glycoconjugates or causes changes in glycosylation processes that uncover galactose residues previously covered by sialic acid. There is evidence that hyperglycemia can modify the activity of glycosyltransferases and affect proper processing of glycoproteins in multiple tissues, resulting in an altered distribution pattern of glycoconjugates. Regarding the reproductive tract, diabetes-induced disturbances of glycobiology might affect uterine receptivity, embryo-maternal interaction, and glandular secretion (Andlib et al., 2024). The decline in WGA binding in diabetic rats further indicates the possible role of diabetes in promoting desialylation of glycoproteins and exposure of their β-galactose residues for further recognition by PNA.

Con A-binding exhibited the least variability between groups. Majority of the samples showed positive or weakly positive reaction irrespective of whether they were from diabetic or non-diabetic females, and whether they were from pregnant females or not. This lesser variability indicates the conservation of high mannose or hybrid type N-glycans in the endometrial secretions despite the metabolic derangement caused by the disease state of diabetes mellitus. The preservation of this Con A binding property indicates that these N-glycans are conserved as they play an important role in forming the basic structure of the glycoprotein that is relatively immune to the effects of diabetes compared to terminal carbohydrate structures.

In contrast, WGA binding was seen to be more dynamic. There were positive staining results in non-diabetic pregnant rats on day 5 of gestation, while this became less on day 7; meanwhile, diabetic rats had weak or negative staining during the duration of the experiment. Due to the fact that WGA mainly reacts to sialic acid and N-acetylglucosamine, these observations indicate that diabetes affects the process of sialylation in endometrial gland secretions. Sialylation is crucial for proper development since sialylated glycoproteins form part of the histiotrophe and have an effect on their structural integrity, protection from proteolytic enzymes, and receptor-ligand interactions with maternal tissues (Hempstock et al., 2004). This will have a negative effect on the quality of secretions from glands and affect the nutrition of the embryo during the important peri-implantation stage.

The binding pattern of soybean agglutinin (SBA) was quite variable between different experimental groups at various time points, implying an independent influence and possible interaction between diabetes and pregnancy on the expression of glycoconjugates containing N-acetylgalactosamine. Day 5 SBA positivity was quite strong in diabetic non-pregnant rats but not as intense in pregnant rats, indicating that pregnancy-induced hormonal alterations can affect glycosylation pathways even in the presence of diabetes.

In this regard, the absence of histochemical positivity does not mean that the glands do not contain any secretory materials rich in carbohydrates. Rather, the positive responses to lectin histochemistry confirm the presence of certain types of glycoconjugates that cannot be detected through regular histochemical procedures. The implication from this is that neutral mucins, acidic mucopolysaccharides, sulfated mucopolysaccharides, and sulfomucins could be either present in trace amounts or have special characteristics that make them difficult to detect via histochemistry.

The biological importance of these changes in glycosylation can be considered significant as well. While the placental circulation is still absent, the developing fetus is entirely reliant on histiotrophic nourishment coming from secretions of endometrial glands. In addition to nutrients, the secretions contain a number of growth factors, cytokines, and adhesion molecules necessary for successful implantation and subsequent development of the blastocyst (Hempstock et al., 2004; Filant & Spencer, 2014). Experimental research shows that without endometrial glands, the implantation of the blastocyst, decidualization, and its survival cannot take place (Filant & Spencer, 2013; Dhakal et al., 2021). Thus, modifications of the glycosylation pattern of glandular secretions may have a direct impact on the biochemical milieu of the developing conceptus.

The augmented presentation of β-galactosyl moieties together with decreased sialylation noted in the diabetic animals may lead to marked changes in the biological characteristics of endometrial glycoproteins. Modulation of the glycosylation pattern may result in protein instability, modulation of receptor interactions, modulation of protein transport, and cellular signaling. As such, the diabetic secretory endometrium may fail to promote normal embryonic development or may modify the process of embryo-maternal interaction during implantation. This may be considered an important mechanism for the higher incidences of embryonic death, impaired fetal growth, implantation failure, and congenital malformations in diabetic pregnancies (Giavini et al., 1986; Giavini et al., 1993; Kiss et al., 2009; Damasceno et al., 2014). Hence, the current results provide further support to existing studies that indicate impairment of endometrial gland secretion as another mode through which diabetes can adversely affect reproduction.

Several weaknesses are apparent in the current investigation. A total sample size of eight animals per experimental group was adequate for conducting a qualitative histochemical analysis; however, further quantitative analysis of

the results could not be accomplished owing to this relatively low number. In addition, diabetes was induced via streptozotocin treatment, which is a widely used procedure for producing extensive damage to β cells resulting in sustained hyperglycemia similar to type 1 diabetes mellitus (Akbarzadeh et al., 2007). While many investigators have used this approach for studying gestation in diabetic women (Kiss et al., 2009; Damasceno et al., 2014), such an animal model does not fully reflect the characteristics of gestational diabetes or even mild pre-existing diabetes observed in patients in real life. In addition, lectin histochemistry can detect only specific carbohydrates but not the particular glycoproteins with which these glycans are associated nor their biological activity.

In summary, the current results show that diabetes mellitus is capable of modifying the pattern of glycosylation of endometrial gland secretion products during the early stage of pregnancy. The presence of PNA-binding β-galactose molecules along with the decreased level of WGA binding glycans shows changes in carbohydrate metabolism which can affect negatively the histiotrophic nourishment of the embryo. Considering the role of uterine glands in implantation and survival of the embryo, it is possible to assume that the changes induced by diabetes mellitus can contribute to reproductive disturbances.

6. Conclusion

Insulin dependent diabetes mellitus in rats, which results from streptozotocin administration, causes a change in the histochemistry of lectins found in glandular epithelial secretions within the endometrium during early pregnancy. The major alteration observed is a positivity for PNA staining with respect to β galactose only in the diabetic animals along with a reduction in WGA binding for sialic acid and β N acetylglucosamine when compared to the non-diabetic animals. This indicates the formation of desialylated and galactose bearing glycoproteins under diabetic conditions, in order to provide nourishment to the developing embryo. No carbohydrates could be detected through conventional histochemical stains such as PAS, Alcian Blue, and aldehyde fuchsin.

These findings add to knowledge about the pathophysiology of diabetic embryopathy through the identification of an alteration in the nutritive environment of the pre-implantation and early post-implantation embryo. Further research is needed to determine what glycoproteins are secreted from the diabetic rat uterus carrying the modified carbohydrate structures and whether normalization of maternal blood sugar levels will reverse the pattern of lectin binding. In cases where the glycosylation alterations could be reversed through glycaemic normalization, these could become valuable histological indicators of successful diabetic management during pregnancy. Image quantification is recommended for subsequent analysis. Protein identification of the PNA-binding proteins from the diabetic rat uterus would provide the answer as to what proteins have the β-galactose modifications.

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