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Research Article

## Histopathological Comparison of Placentas of Women with High and Normal BMI During Pregnancy

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### Abstract

**Objective:** High body mass index (BMI) during pregnancy has been associated with adverse outcomes for the mother, fetus, and placenta. The effects of these adverse outcomes on the placenta in pregnant women with high BMI have not yet been histomorphologically characterized. Clarifying the effects of high BMI on placental function and determining necessary measures to protect maternal and fetal health is important. The aim of this study is to evaluate the possible structural and functional changes occurring in the placentas of women with normal weight and those with high BMI during pregnancy using hematoxylin-eosin (H&E) staining, and to compare their placentas histopathologically.

**Materials and Methods:** Placental tissues were obtained from 45 normal, healthy pregnant patients with a BMI <30 kg/m<sup>2</sup> and 45 obese pregnant women with a BMI ≥30 kg/m<sup>2</sup>. All samples underwent routine histological processing and were stained with H&E. Placental sections were examined under a light microscope for villous architecture, syncytiotrophoblast integrity, stromal changes, fibrin deposition, vascular congestion, and syncytial node formation.

**Results:** In H&E staining, while the villus structures in the control group placentas were regular and well-preserved, the high BMI group showed impaired villus integrity, fibrinoid deposition, congestion, stromal edema, and a significant increase in syncytial nodes.

**Conclusion:** High BMI during pregnancy is associated with significant placental histomorphological changes detectable by routine H&E staining. These changes suggest that maternal weight gain damages placental tissue through hypoxic stress and inflammatory responses.

**Keywords:** High body mass index, gestational weight gain, maternal obesity, placenta, histopathology.

## INTRODUCTION

Pregnancy involves active maternal changes to ensure growth and development in both mother and fetus. Obesity arises as a result of an imbalance between energy intake and expenditure<sup>1</sup>. Obesity is one of the fastest-growing global health problems among women in sexual marriage<sup>2</sup>. Excessive weight gain during pregnancy can cause various adverse effects on the health of both mother and fetus. Maternal obesity increases the risk of adverse health outcomes in both mothers and fetuses. Complications include preeclampsia, gestational diabetes mellitus (GDM), and gestational hypertension<sup>3</sup>. The placenta is a complex organ with a vital role in embryo implantation and maintenance of pregnancy. The placenta transports maternal nutrients to the fetus and regulates the fetal nutrition environment<sup>4,5</sup>. More specifically, the human placenta is "hemochorial," and its placental characteristics are selected by its direct contact with maternal blood<sup>6</sup>. Placental function is disrupted by a range of maternal metabolic signals, many of which are affected by maternal obesity<sup>7,8</sup>. Thus, it is suggested that maternal obesity can lead to significant changes in

placental function, demonstrating that maternal obesity causes adverse fetal changes. Histopathological changes in the placentas of pregnant women with high BMI are associated with adverse reproductive problems and a risk to healthy fetal development. Some of these include: placental growth and hypertrophy, changes in villous structure, increased fibrin fragments, abnormalities in placental vessels, and placental hypoxia<sup>9,10</sup>.

The aim of this study is to compare placentas from pregnant women with high BMI to those from pregnant women with normal BMI, and to investigate possible structural and functional changes in placental tissue. The findings aim to clarify the effects of excessive weight gain in pregnant women on placental function and to identify necessary measures to protect maternal and fetal health.

## MATERIALS AND METHODS

### Study Population and Tissue Collection

This study was conducted using placental tissues from 45 normal, healthy pregnant women with a BMI <30 and 45 obese pregnant women with a BMI ≥30 who had given birth, all admitted to the Obstetrics and Gynecology Clinic

of Dicle University Faculty of Medicine. Patients aged between 18 and 40 years, those with other pregnancy complications, systemic disorders, or chronic diseases were excluded from the study.

Small pieces of placental tissue were taken from the pregnant women included in the study for histological examination; these tissue samples were not viable, had been severed from the body, and were not suitable for pathology analysis.

### Histological Processing and Hematoxylin-Eosin Staining

Placental tissue samples obtained from pregnant women were excised to a suitable size for histological processing, then fixed in 10% formalin. After fixation, the tissues were passed through tap water, increasing alcohol series, and xylene stages for paraffin tissue processing, and the tissues were embedded in paraffin blocks. Following tissue processing, placental tissue sections obtained from paraffin blocks were placed in a bain-marie at 37°C. To dissolve excess paraffin on the slide, the sections were kept in an incubator at 58-62°C for 6 hours. The sections were deparaffinized in xylene for 3 x 15 minutes, then dehydrated by passing them through decreasing alcohol series (100%, 96%, 90%, 70%, 50% ethyl alcohol) for 10

minutes each. Afterwards, the sections, which were kept in distilled water for 5 minutes, were stained with H&E. Following staining, sections were rapidly passed through increasing alcohol series (80%, 90%, 96% ethyl alcohol series) and held in absolute alcohol for 2 minutes. Finally, the sections were held in xylene for 3x15 minutes, and Entellan was dropped onto the tissue and covered with a coverslip. The prepared slides were evaluated and images were taken using a Zeiss Imager A2 photomicroscope.

## RESULTS

### Hematoxylin-Eosin Findings

Control Group (Figure 1): In the placental section of the control group, the chorionic villus structures are morphologically well preserved. The cyto- and syncytiotrophoblast cell layers are regular, and there is no significant atypia in the nuclei. Congestive changes in the villous stroma are minimal. Fibrinoid deposition is limited and within physiological limits. Syncytial nodes are rarely observed, indicating normal placental maturation. Intervillous spaces show a homogeneous distribution; no inflammatory cell infiltration was detected.

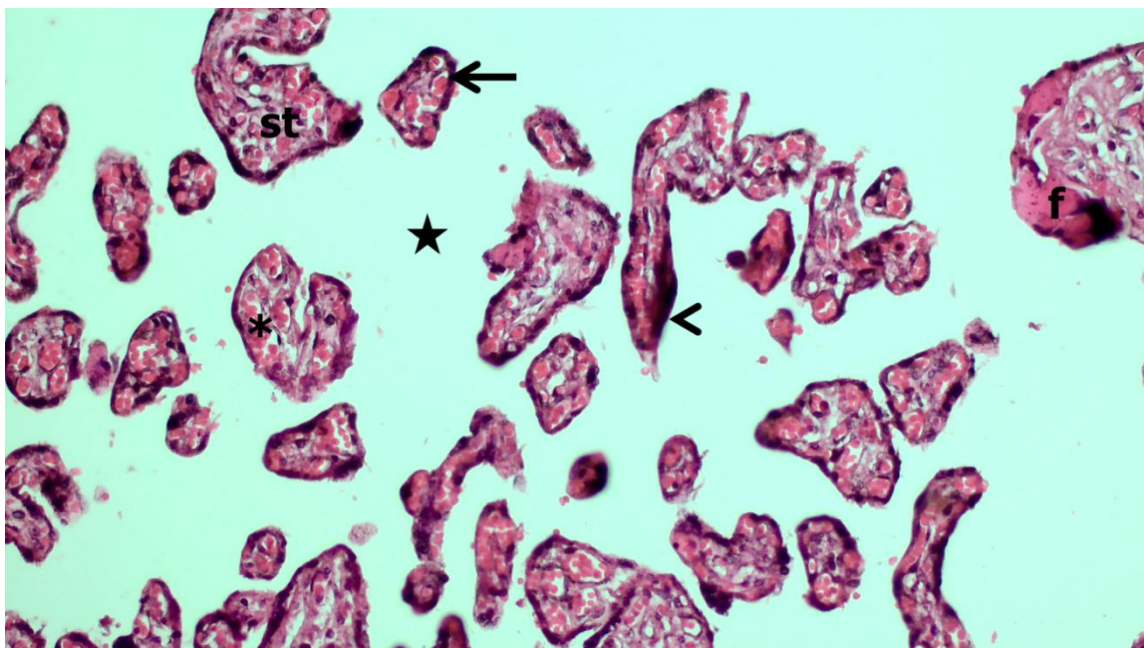


Figure 1: Placental tissue sample from the control group stained with H&E. Arrow: chorionic villus, arrowhead: syncytial node, st: stroma, asterisk: villous capillary, f: fibrin deposition, asterisk: intervillous space, Scale bar: 50  $\mu$ m, magnification: x20

High BMI Group (Figure 2): In placental sections from pregnant women who gained excessive weight, the villous structures are significantly disrupted, appearing misshapen and with irregular contours in places. Thickening and hyperplasia of syncytiotrophoblasts are noteworthy. Intense congestion and edema are present in the stroma. Syncytial nodes are significantly increased, which may be indicative of hypoxic stress or premature villous maturation. Fibrinoid deposition is more

widespread than in the control group and surrounds the villous surfaces in places. Inflammatory cell infiltration and erythrocyte extravasation are observed in the intervillous space. Overall, a disruption of placental tissue integrity and an increased predisposition to inflammation and fibrosis are observed (Figure 2). Histopathological findings were more intense in this group compared to the control group.

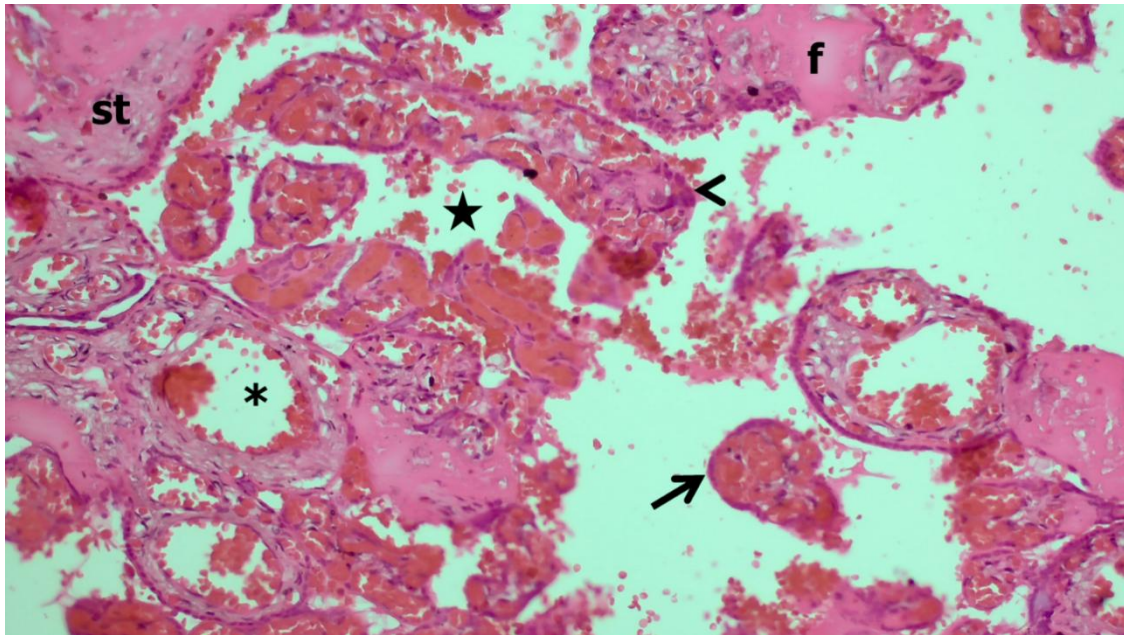


Figure 2: Placental tissue sample from the high BMI group stained with H&E. Arrow: chorionic villus, arrowhead: syncytial node, st: stroma, asterisk: villous capillary, f: fibrin deposition, asterisk: intervillous space, Scale bar: 50  $\mu$ m, magnification: x20

## DISCUSSION

Excessive weight gain during pregnancy can have various negative effects on maternal and fetal health. The placenta, as a critical organ facilitating the exchange of substances between mother and fetus, is essential for the healthy progression of pregnancy. This study histopathologically demonstrated the structural and functional changes detected in the placentas of pregnant women with high BMI and those with normal BMI after hematoxylin-eosin staining and light microscopy.

Previous studies have made claims regarding changes in pregnant women with high BMI. These include placental growth and hypertrophy, changes in villous structure, increased fibrin deposition, abnormalities in placental vessels, placental hypoxia, and fibrinoid necrosis<sup>9-12</sup>.

In this study, while the villous structures of the placentas in the control group were regular and well-preserved in hematoxylin-eosin staining, the villous integrity was disrupted in the high BMI group, and a significant increase in fibrinoid deposition, congestion, stromal edema, and syncytial knots was observed. Villous disorganization and stromal degeneration can reduce the available effective surface area for maternal-fetal exchange, while vascular congestion suggests impaired uteroplacental blood flow<sup>13</sup>. Increased fibrin deposition is often interpreted as an indicator of placental damage and hypoxic stress and has been associated with decreased placental efficiency<sup>14</sup>. These changes suggest that maternal weight gain damages placental tissue through hypoxic stress and inflammatory responses. The potential impact of these adverse placental changes on the fetus carried by pregnant women with high BMI is another topic that can be discussed.

These findings aim to clarify the effects of excessive weight gain during pregnancy on placental functions,

thereby contributing to the identification of necessary measures to protect maternal and fetal health.

## CONCLUSION

High BMI during pregnancy can have negative effects not only on maternal health but also on placental structure and function. In this study, impaired villous integrity, fibrinoid accumulation, congestion, stromal edema, and a significant increase in syncytial knots were observed in the placenta of pregnant women with high BMI. The findings aim to clarify the effects of high BMI on placental functions during pregnancy and contribute to the identification of necessary measures to protect maternal and fetal health. It may also guide new studies on which fetal health problems high BMI may cause in pregnant women.

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**Ethical Approval:** This study was approved by the Dicle University Faculty of Medicine Non-Interventional Clinical Research Ethics Committee (Approval date: 14 May 2025; Approval number: 2025/198). All procedures were conducted in accordance with the ethical standards of the institutional and national research committees and with the principles of the Declaration of Helsinki.

**Informed Consent:** Informed consent was obtained from all participants prior to inclusion in the study.

**Conflict of Interest:** The author declares that there is no conflict of interest regarding the publication of this thesis.

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## REFERENCES

1. Bouchard C., "Can obesity be prevented?" *Nutr Rev.* 1996;54(4 Pt 2):125-130. <https://doi.org/10.1111/j.1753-4887.1996.tb03907.x> PMID:8700439
2. Poston L, Caleyachetty R, Cnattingius S, ve diğerleri. Gebelik öncesi ve maternal obezite: epidemiyoloji ve sağlık sonuçları. *Lancet Diabetes Endocrinol* 2016;4(12):1025-1036. [https://doi.org/10.1016/S2213-8587\(16\)30217-0](https://doi.org/10.1016/S2213-8587(16)30217-0) PMID:27743975
3. Jeve, YB; Konje, JC; Doshani, A. Obez Kadınlarda Plasental Disfonksiyon ve Doğum Öncesi Gözetim Stratejileri. *En İyi Uygulama Araştırmaları Klinik Obstetrik ve Jinekoloji.* 2015;29:350-364.
4. Sadovsky Y, Jansson T. Bölüm 39 - Plasenta ve Plasental Taşıma Fonksiyonu. İçinde: Plant TM, Zeleznik AJ, Albertini DF, Goodman RL, Herbison AE, McCarthy MM, Muglia LJ, Richards JS, editörler. *Knobil ve Neill'in Üreme Fizyolojisi.* 4. San Diego: Academic Press; 2015:1741-1782.
5. Wallace JM, Bhattacharya S, Horgan GW. Aberdeen'de tekil doğumlar için plasenta ağırlığına ilişkin gebelik yaşı, cinsiyet ve pariteye özgü persentil çizelgeleri. *UK Placenta.* 2013;34(3):269-274. <https://doi.org/10.1016/j.placenta.2012.12.007> PMID:23332414
6. Évain-Brion, D.; Malassine, A.; Frydman, R. *Le Placenta Humain*; Ed. Médicales Internationales: Cachan, Fransa, 2010.
7. Vaughan OR, Rosario FJ, Powell TL, Jansson T. Plasental Amino Asit Taşımalarının ve Fetal Büyümenin Düzenlenmesi. *Prog Mol Biol Transl Sci.* 2017;145:217-51. <https://doi.org/10.1016/bs.pmbts.2016.12.008> PMID:28110752
8. Jansson T, Powell TL. Plasental besin alımlamasının gelişimsel programlamadaki rolü. *Clin Obstet Gynecol.* 2013. Eylül;56(3):591-601. <https://doi.org/10.1097/GRF.0b013e3182993a2e> PMID:23703224 PMID:PMC3732521
9. Kelly AC, Powell TL, Jansson T. Placental function in maternal obesity. *Clin Sci (Lond).* 2020 Apr 30;134(8):961-984. <https://doi.org/10.1042/CS20190266> PMID:32313958 PMID:PMC8820171
10. Santos ED, Hernández MH, Sérazin V, Vialard F, Dieudonné MN. Human Placental Adaptive Changes in Response to Maternal Obesity: Sex Specificities. *Int J Mol Sci.* 2023 Jun 5;24(11):9770. <https://doi.org/10.3390/ijms24119770> PMID:37298720 PMID:PMC10253453
11. Brouwers L, Franx A, Vogelvang TE, Houben ML, van Rijn BB, Nikkels PG. Association of Maternal Prepregnancy Body Mass Index With Placental Histopathological Characteristics in Uncomplicated Term Pregnancies. *Pediatr Dev Pathol.* 2019 Jan-Feb;22(1):45-52. <https://doi.org/10.1177/1093526618785838> PMID:29969058 PMID:PMC6604681
12. Kristiansen O, Roland MC, Zucknick M, Reine TM, Kolset SO, Henriksen T, Lekva T, Michelsen T. Maternal body mass index and placental weight: a role for fetal insulin, maternal insulin and leptin. *J Endocrinol Invest.* 2022 Nov;45(11):2105-2121. <https://doi.org/10.1007/s40618-022-01842-2> PMID:35781790 PMID:PMC9525437
13. Burton GJ, Jauniaux E. Oxidative stress. *Best Pract Res Clin Obstet Gynaecol.* 2011;25(3):287-299. <https://doi.org/10.1016/j.bpobgyn.2010.10.016> PMID:21130690 PMID:PMC3101336
14. Friis CM, Roland MC, Godang K, Voldner N, Bollerslev J, Henriksen T. Adiposity-related inflammation: effects of pregnancy. *Obesity (Silver Spring).* 2013;21(1):E124-E130. <https://doi.org/10.1002/oby.20120> PMID:23505192