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The Impact of Parity on Insulin Sensitivity: A Case-Control Study Using HOMA-IR Analysis

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تأثير الحمل على حساسية الأنسولين: دراسة حالة – ضابطة باستخدام تحليل *HOMA-IR*

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Abstract:
This study, conducted in 2024 at Misrata Medical Center, aimed to investigate the impact of parity on insulin sensitivity using the HOMA-IR analysis. The research included 102 participants, with ages ranging from 18 to 50 years. They were divided into two groups: a control group of 46 nulliparous women (women with no deliveries) and a patient group of 56 multiparous women (women with more than three deliveries). A significant positive correlation was identified between parity and HOMA-IR ($r = 0.489^{**}$, $p < 0.01$), indicating that higher parity is associated with increased insulin resistance. Nulliparous women exhibited the lowest HOMA index values, while multiparous women, particularly those with five or more deliveries, showed the highest values. Statistical analysis confirmed significant differences in insulin resistance among parity groups, emphasizing the need for further studies to explore underlying mechanisms and clinical implications.

Keywords: Parity, Insulin sensitivity, Insulin resistance, HOMA-IR analysis.

الملخص
تم إجراء هذه الدراسة عام 2024 في مركز مصراتة الطبي للكشف عن تأثير عدد مرات الولادة على حساسية الأنسولين باستخدام تحليل *HOMA-IR*. شملت الدراسة 102 مشاركة، وتراوحت أعمارهن بين 18 سنة و50 سنة. تم تقسيمهن إلى مجموعتين: المجموعة الضابطة المكونة من 46 امرأة لم يكن لديهن مرات ولادة، ومجموعة المرضى المكونة من 56 امرأة متعددات الولادة (لديهن أكثر من ثلاث ولادات). تم تحديد ارتباط إيجابي معنوي بين عدد مرات الولادة ومؤشر *HOMA-IR* ($r = 0.489^{**}$، $p < 0.01$)، مما يشير إلى أن ارتفاع عدد مرات الولادة يرتبط بزيادة مقاومة الإنسولين. أظهرت النساء اللواتي لم يلدن أقل قيم للمؤشر، في حين سجلت النساء متعددات الولادة، وخاصة من لديهن خمس ولادات أو أكثر، أعلى القيم. أكدت التحليلات الإحصائية وجود فروق ذات دلالة إحصائية في مقاومة الإنسولين بين مجموعات الولادة، مما يشدد على ضرورة إجراء دراسات إضافية لاستكشاف الآليات والدلالات السريرية لهذه العلاقة.

الكلمات المفتاحية: عدد الولادات، حساسية الأنسولين، مقاومة الأنسولين، تحليل *HOMA-IR* .

Introduction

Insulin resistance and the metabolic abnormalities associated with it have been linked to metabolic syndrome, type 2 diabetes mellitus, and cardiovascular disease in both adults and the elderly. Recently, metabolic syndrome has gained recognition in children and adolescents. The rise in childhood obesity worldwide is particularly concerning due to its strong association with insulin resistance [1].

The concept of "insulin resistance" emerged with the introduction of insulin therapy for diabetes more than 50 years ago. Clinical observations at that time indicated the existence of two distinct groups of diabetic patients, which roughly aligned with the contemporary classifications of type 1 and type 2 diabetes, differentiated by their responses to exogenously administered insulin. The term "insulin resistance" was introduced to describe patients exhibiting a significantly increased requirement for insulin, defined as needing over 200 units per day, often in association with antibodies produced by the insulin preparations available at that time, such as bovine and porcine insulin. As research progressed, particularly with the advent of radioimmunoassay techniques in the 1960s, it became possible to distinguish type 1 diabetic patients, characterized by absolute insulin deficiency, from type 2 patients, who typically present with normal or elevated insulin levels.

This led to the recognition of individuals with normal glucose levels but relatively high insulin levels. Further investigations conducted in the 1970s and 1980s, utilizing in vivo metabolic techniques to measure glucose uptake during insulin infusions and ex vivo analyses of tissues from insulin-resistant patients, conclusively demonstrated that insulin resistance is a result of impaired insulin action in peripheral tissues such as adipose tissue, muscle, and liver. Currently, insulin resistance is defined as a clinical state characterized by a diminished biological response to normal or elevated levels of insulin, resulting in impaired glucose uptake and metabolism in target tissues [2,3].

In 1927, the phenomenon of insulin resistance associated with obesity was first documented through the case of a young man who presented with significant adiposity and severe diabetes. Despite adhering to a caloric restriction diet aimed at managing his condition, he demonstrated minimal to no response to subcutaneous injections of insulin, even at dosages as high as 160 units per day [4]. This case was particularly striking because the patient's obesity profile markedly differed from the commonly reported cases of severely undernourished individuals suffering from diabetes at that time. Consequently, his condition was not classified as pancreatic diabetes; instead, his glucosuria was attributed to an alternative underlying mechanism [4,5]. The formal recognition of insulin resistance as a contributing factor to diabetes did not occur until several years later.

The pivotal work of Sir Harold Himsworth played a crucial role in this advancement. Himsworth systematically evaluated two distinct groups of patients following the administration of a fixed oral glucose load, assessing their insulin responses both before and after insulin administration. His methodology involved objective measurements of urinary glucose concentrations, allowing for a quantifiable and standardized approach to evaluating insulin and glucose responses. Himsworth's findings were groundbreaking, leading him to propose a classification of diabetes into two distinct phenotypes: the "typical diabetes" characterized by insulin deficiency, usually observed in young, lean individuals with normal blood pressure; and a second group exhibiting insulin resistance, often older, with excess adiposity and comorbidities such as hypertension and arteriosclerosis.

The significant differences in urinary glucose responses between these two groups provided compelling evidence for the existence of insulin resistance and its role in the pathophysiology of diabetes. This critical recognition not only enhanced the understanding of the metabolic mechanisms underlying diabetes but also laid the groundwork for future research into insulin dynamics. Himsworth's classification was officially accepted by the Royal College of Physicians of London in 1939, marking a significant milestone in the medical community's comprehension of the complexities of diabetes. The introduction of the term "insulin sensitivity" represented a monumental shift in the understanding of glucose metabolism, highlighting the dual pathways through which diabetes could manifest. Thus, the early observations of insulin resistance paved the way for a deeper exploration of metabolic disorders and their multifaceted nature [4,6,7].

After the presentation to the Royal College of Physicians in London, attention shifted towards developing insulin bioassays to support clinical phenotypes and observations indicating two different mechanisms causing diabetes. Early methods relied on insulin's ability to lower glucose in vivo or in vitro. Alongside evaluating responses to the oral glucose tolerance test, standard curves were created to quantify insulin sensitivity and indirectly estimate plasma insulin concentrations that could lead to hypoglycemia in rats or rabbits, glucose uptake in excised rat hemidiaphragms, or oxidation rates of [1-14C] glucose in the rat epididymal fat pad. It was recognized that individuals with diabetes ranged from insulin-sensitive to insulin-insensitive, with the same carbohydrate load resulting in higher blood glucose levels. However, precise quantification of "insulin insensitivity" was needed, independent of indirect glucose response measurements or ex vivo standard curves. Critical next steps included the

development of accurate glucose and insulin assays, essential for a better understanding of insulin resistance mechanisms [7].

The Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) is a commonly utilized tool in large epidemiological studies and clinical practice for estimating insulin resistance [8].

Insulin resistance changes over time during pregnancy, and in the last half of the pregnancy, insulin resistance increases considerably and can become severe, especially in women with gestational diabetes and type 2 diabetes. Numerous factors such as placental hormones, obesity, inactivity, an unhealthy diet, and genetic and epigenetic contributions influence insulin resistance in pregnancy, but the causal mechanisms are complex and still not completely elucidated [9].

Some studies have indicated that there has been a significant increase over the past few decades in the number of reproductive-age women who are either overweight or obese. Overweight and obese women are at an increased risk of experiencing decreased insulin sensitivity compared to their lean or average-weight counterparts. The interplay between obesity and reduced insulin sensitivity further elevates the long-term risk of developing metabolic syndrome and its associated complications, including diabetes, hypertension, hyperlipidemia, and cardiovascular disorders.

Due to the metabolic changes that occur during normal pregnancy, particularly the notable 60% reduction in insulin sensitivity, overweight and obese women are at a heightened risk of metabolic dysregulation during pregnancy [10].

The number of pregnancies may influence insulin sensitivity, as indicated by a population-based study of 1,186 women aged 40 and older. Women diagnosed with diabetes before age 40 or with insulin-dependent diabetes were excluded. Based on WHO criteria, 714 had normal glucose tolerance, 326 had impaired glucose tolerance, and 146 had non-insulin-dependent diabetes mellitus (NIDDM).

After adjusting for age, obesity, and family history of diabetes, increased parity was significantly associated with a higher risk of both NIDDM (odds ratio 1.16) and impaired glucose tolerance (odds ratio 1.10) per pregnancy. This association persisted even when accounting for obesity, indicating that the increased risk is not explained by this factor [11]

It was conducted a study involving 1,880 Caucasian women to explore the relationship between parity and peripheral insulin sensitivity index (ISIOGTT) or gestational diabetes mellitus (GDM). Participants underwent a 100-g, 3-hour oral glucose tolerance test (OGTT) during the 24th to 28th weeks of gestation. The findings indicated that higher parity was linked to decreased ISIOGTT and increased CP/FPG in women with more than three pregnancies. GDM was diagnosed in 124 women (6.58%), demonstrating a linear relationship with parity ($P = 0.0034$), significantly influenced by age. However, these associations became non-significant after adjusting for age, pregestational body mass index (BMI), and weight gain. The study concluded that parity is not directly associated with declines in insulin sensitivity or increases in CP/FPG; rather, these relationships are mediated by progressive aging and weight gain before or during pregnancy, especially with longer intervals between pregnancies [12]

It was conducted another study in Korea involving 4,098 postmenopausal women using nationally representative data from the Korea National Health and Nutrition Examination Survey (2010–2012). This cross-sectional study utilized multivariate logistic regression to evaluate the relationship between parity and insulin resistance syndrome, adjusting for potential confounding variables. The findings revealed that the prevalence of metabolic syndrome significantly increased with higher parity [13].

In another study conducted in Colombia to investigate the relationship between insulin resistance and parity, which included 1,795 participants, significant associations were found between these factors. The data of participants were analyzed using multiple statistical models to assess the impact of the number of births on insulin resistance levels. The results indicated that an increased number of births was associated with higher insulin resistance, suggesting potential effects on women's health postpartum [14].

Understanding the impact of parity on insulin sensitivity is crucial in the field of endocrinology and metabolic health. Parity, or the number of times a woman has given birth, can have significant long-term effects on insulin sensitivity, which in turn affects the risk of developing conditions such as Type 2 diabetes and cardiovascular diseases. Despite the growing body of research on insulin resistance and related metabolic disorders, the specific effects of parity on insulin sensitivity have not been extensively studied.

This research aims to fill this gap by examining the relationship between parity and insulin sensitivity using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). By employing a case-control study design, this study seeks to provide robust evidence on how the number of pregnancies can influence metabolic health. The findings from this research could have significant implications for

clinical practice, including the development of tailored interventions and management strategies for women at different stages of their reproductive lives.

Furthermore, this study contributes to a broader understanding of how reproductive factors influence metabolic health, which is essential for improving women's health outcomes globally. The insights gained from this research could inform public health policies and lead to more effective prevention and treatment strategies for insulin resistance and its associated conditions.

Material and methods

This study was conducted at Misrata Medical Center from September to December 2024 to assess the impact of parity on insulin sensitivity using HOMA-IR analysis. The study was analytical in a case-control design, including 102 samples of women aged between 15 and 60 years, with 56 cases and 46 controls. The control group consisted of women with no prior births, while the case group included women with more than three births. Pregnant women and those with diabetes were excluded, as well as individuals with a body mass index (BMI) greater than 30 cm

Exclusion Criteria

1. Pregnant women
2. Women with diabetes
3. Individuals with a body mass index (BMI) greater than 30 cm (obesity)
4. Patients with kidney disease
5. Women with severe obesity

Results and discussion

Using the SPSS statistical software, we examined the impact of parity on insulin resistance between two groups of women. The first group, the control group, consisted of 46 nulliparous women (women who have never given birth). The second group consisted of 56 multiparous women (women with more than three childbirths). The HOMA (Homeostatic Model Assessment) index was used to evaluate insulin resistance in both groups. The participants' mean age is 33.67 years, with values spanning from 18 to 50 years, and a standard deviation (SD) of 10.42, indicating moderate variability. Their average height is 1.58 m, within a range of 1.43 m to 1.80 m, with minimal variation reflected by an SD of 0.08. Waist circumference averages at 106.07 cm, ranging from 90 cm to 127 cm, with a moderate SD of 9.64. Meanwhile, the mean BMI stands at 37.90, varying between 27.55 and 64.18, and an SD of 6.65 highlights notable differences in BMI among participants.

Table 1: Descriptive Statistics of Anthropometric and Demographic Data for Female Participants.

Parameter	Mean	Maximum	Minimum	Standard Deviation
Age (years)	33.67	50	18	10.42
Height (m)	1.58	1.80	1.43	0.08
Waist (cm)	106.07	127	90	9.64
Body Mass Index	37.90	64.18	27.55	6.65

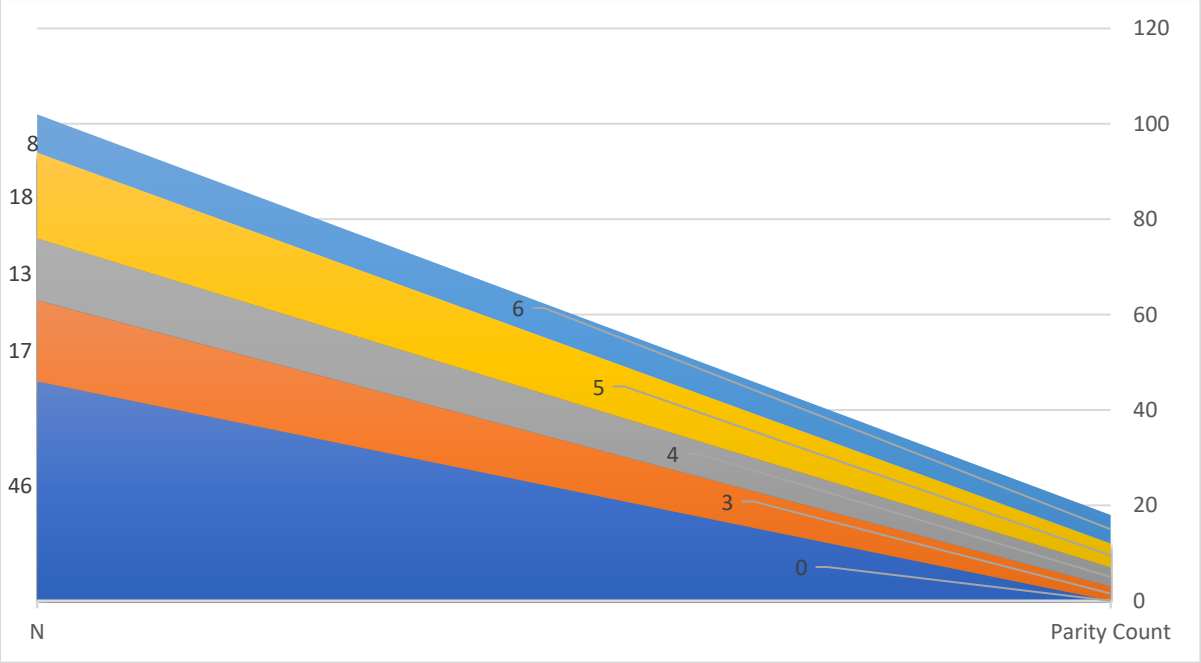

Figure 1: Sample Size in Relation to Parity

The mean HOMA index varied across parity groups, with Group control (women who have never given birth) having the lowest value (2.3545), indicating minimal insulin resistance, while Group 5 (The group of women with a number of 5 children) exhibited the highest value (4.5372), reflecting significant insulin resistance. Variability analysis, based on standard deviations and confidence intervals, highlights that Group 5 (The group of women with a number of 5 children) demonstrated the widest range, with a maximum HOMA value of 12.60. These results suggest a potential relationship between parity and increased insulin resistance, as higher parity groups tended to show elevated HOMA index values. where the numbers indicate groups such as 3, 4, 5, and 6, referring to the parity or the number of deliveries in the cases under study

Table 2: Descriptive Statistics of HOMA Index Across Parity Groups (Updated).

Case/control	Parity Count	N	Mean	Standard Deviation	Standard Error	95% Confidence Interval for Mean (Lower Bound)	95% Confidence Interval for Mean (Upper Bound)	Minimum	Maximum
Control	0	46	2.1451	1.41322	0.20826	1.7269	2.5633	0.90	6.98
case	3	17	4.4035	1.66576	0.40401	3.5471	5.2600	2.10	6.90
	4	13	4.2892	1.26880	0.35190	3.5225	5.0560	1.25	5.80
	5	18	4.5372	2.31301	0.54518	3.3870	5.6875	2.10	12.60
	6	8	4.0513	2.10669	0.74483	2.2900	5.8125	1.20	6.20
Total		102	3.3612	2.01508	0.19952	2.9654	3.7570	0.90	12.60

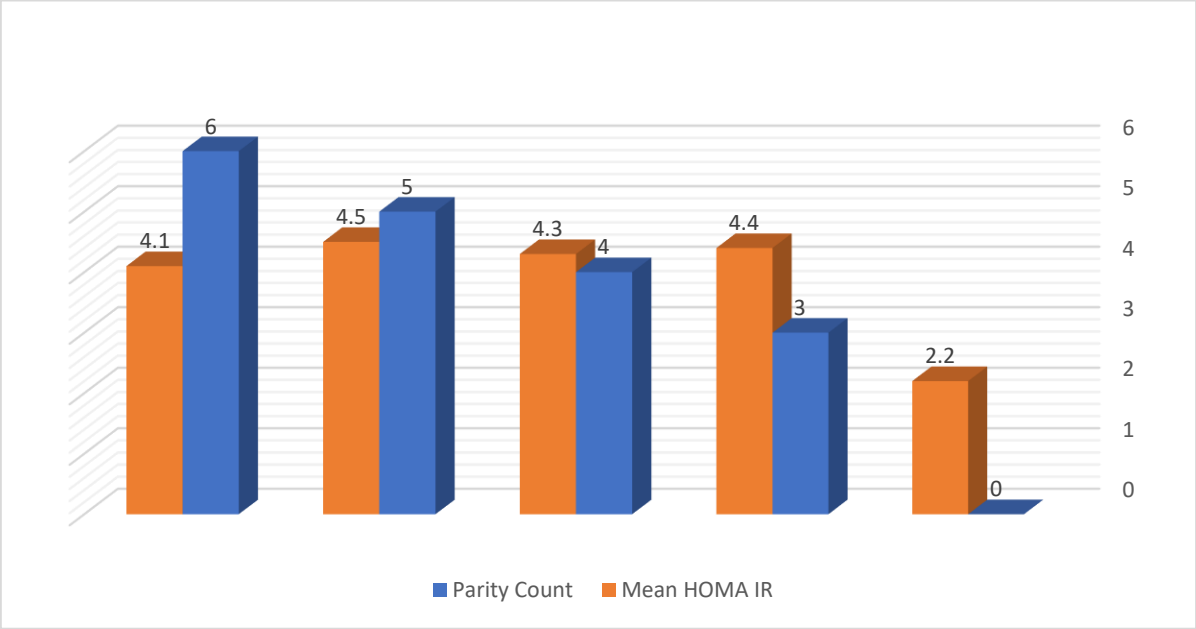

Figure 2: Comparison of the Mean HOMA-IR Analysis and Parity

A one-way ANOVA was conducted to examine the differences in insulin resistance between the two groups. The results indicate a significant variation in insulin resistance between the groups. The high F-ratio (9.066) and the highly significant p-value ($P < 0.001$) suggest a statistically significant difference between the groups. The ANOVA results point to significant differences in insulin resistance between nulliparous women and multiparous women. Multiparous women show higher levels of insulin resistance compared to nulliparous women. This finding suggests that the number of childbirths may have an impact on insulin resistance levels, warranting further research and investigation into the contributing factors of this relationship.

Table 3: One-Way ANOVA for Insulin Resistance (HOMA) Between Nulliparous and Multiparous Women.

Source	Sum of Squares	Df	Mean Square	F	Sig.
Between Groups	131.538	5	26.308	9.066	< 0.001
Within Groups	278.577	96	2.902	-	-
Total	410.116	101	-	-	-

Between Groups: The sum of squares (131.538) and the mean square (26.308) with 5 degrees of freedom (df) resulted in an F-value of 9.066. The significance level ($P < 0.001$) indicates a statistically significant difference in insulin resistance between the two groups.
Within Groups: The sum of squares (278.577) with 96 degrees of freedom (df) produced a mean square value of 2.902.

The combined sum of squares for the entire dataset was 410.116 with 101 degrees of freedom (df). The high F-ratio (9.066) and the highly significant p-value ($P < 0.001$) demonstrate that there is a statistically significant difference in insulin resistance between nulliparous and multiparous women. This finding suggests that multiparous women exhibit higher levels of insulin resistance compared to their nulliparous counterparts. The results underscore the potential impact of parity on insulin resistance, highlighting the importance of considering reproductive history in the assessment and management of insulin resistance in women. The Tukey HSD (Honestly Significant Difference) test was employed to compare the HOMA index across different parity groups due to its robustness in performing pairwise comparisons while controlling for Type I error in multiple group analyses.

The results revealed several statistically significant differences ($p < 0.05$). Notably, HOMA index values were significantly lower in Group 0 (control) compared to Groups 3, 4, and 5, and in Group 1 compared to Groups 3, 4, 5, and 6. Conversely, some comparisons, such as 0 vs 6 and 3 vs 4, were not significant, indicating similar HOMA index values between these groups, where the numbers indicate groups such as 3, 4, 5, and 6, referring to the parity or the number of deliveries in the cases under study.

Clinically, the significant findings suggest that higher parity (e.g., Groups 3, 4, 5, and 6) might be associated with increased insulin resistance as measured by the HOMA index, highlighting a potential metabolic impact of parity. Additionally, the 95% confidence intervals reinforce these conclusions, showing non-overlapping ranges in significant comparisons while confirming similarity in non-significant pairs.

Table 4: Multiple Comparisons of HOMA Index Across Parity Groups (Tukey HSD).

Group Comparison (I-J)	Mean Difference (I-J)	Standard Error	p-Value (Significance)	95% Confidence Interval Lower Bound	95% Confidence Interval Upper Bound
0 vs 1	0.59801	0.52035	0.859	-0.9152	2.1112
0 vs 3	-2.04905*	0.52035	0.002	-3.5623	-0.5358
0 vs 4	-1.93475*	0.56858	0.012	-3.5882	-0.2813
0 vs 5	-2.18274*	0.51115	0.001	-3.6692	-0.6963
0 vs 6	-1.69677	0.68029	0.136	-3.6751	0.2816
1 vs 0	-0.59801	0.52035	0.859	-2.1112	0.9152
1 vs 3	-2.64706*	0.58429	0.000	-4.3462	-0.9479
1 vs 4	-2.53276*	0.62763	0.001	-4.3580	-0.7076
1 vs 5	-2.78075*	0.57612	0.000	-4.4562	-1.1054
1 vs 6	-2.29478*	0.73036	0.026	-4.4187	-0.1708
3 vs 4	0.11430	0.62763	1.000	-1.7109	1.9395
3 vs 5	-0.13369	0.57612	1.000	-1.8091	1.5417
3 vs 6	0.35228	0.73036	0.997	-1.7717	2.4762
4 vs 5	-0.24799	0.62003	0.999	-2.0511	1.5551
4 vs 6	0.23798	0.76547	1.000	-1.9881	2.4640
5 vs 6	0.48597	0.72384	0.985	-1.6190	2.5910

Statistically significant differences at $p < 0.05$ are marked with an asterisk.

Using Pearson's correlation coefficient to determine the relationship between two variables, it was found that there is a statistically significant positive correlation between parity (number of deliveries) and HOMA-IR ($r = 0.489$, $p < 0.01$). This indicates that as the number of deliveries increases, there is a tendency for HOMA-IR to rise, suggesting a potential association between higher parity and insulin resistance. The sample size ($N = 102$) provides strong statistical power to validate these findings.

Table 5: Correlation Analysis Between Parity and HOMA-IR.

	Parity (Number of Deliveries)	HOMA-IR
Parity count	1.000	0.489**
HOMA-IR	0.489**	1.000
Sig. (2-tailed)	-	0.000
N	102	102

These results underline the necessity for further investigations to explore the underlying mechanisms of this relationship, such as hormonal changes or metabolic adaptations associated with pregnancy and childbirth. The findings may have clinical implications in identifying parity as a potential

risk factor for insulin resistance in certain populations.

Discussion

The relationship between insulin resistance and the number of births (parity) is a complex and multifaceted topic that has been explored in various studies. Insulin resistance, a condition where cells in the body become less responsive to insulin, is a precursor to metabolic disorders such as type 2 diabetes and cardiovascular diseases. Parity, or the number of times a woman has given birth, has been hypothesized to influence insulin sensitivity due to physiological and hormonal changes during pregnancy [15,16,17].

Pregnancy is a unique physiological state characterized by profound hormonal and metabolic changes. Among these, the rise in hormones such as estrogen, progesterone, and human placental lactogen (hPL) plays a pivotal role in modulating insulin sensitivity. These hormonal shifts are essential adaptations to ensure that the growing fetus receives an adequate supply of glucose, the primary energy source for fetal development [18,19], Estrogen promotes vascular changes and enhances uteroplacental blood flow, indirectly influencing glucose metabolism. Progesterone, on the other hand, supports the maintenance of pregnancy but can reduce insulin sensitivity in peripheral tissues, contributing to a state of insulin resistance [20], Human Placental Lactogen (hPL) is a key hormone produced by the placenta, with levels increasing as pregnancy progresses. It plays a pivotal role in modifying maternal metabolism to prioritize glucose availability for the fetus, ensuring optimal energy supply for fetal development.

This is achieved by inducing insulin resistance in maternal tissues, a physiological adaptation that diverts glucose to the fetus. Alongside other hormonal changes, such as the rise in estrogen and progesterone, hPL contributes to the complex metabolic adjustments required during pregnancy. While these changes are essential for fetal growth, repeated pregnancies may exacerbate or prolong insulin resistance in some women, potentially increasing the risk of metabolic disorders [21], Elevated levels of cortisol during pregnancy further contribute to insulin resistance by promoting gluconeogenesis and lipolysis. These changes are part of the body's natural adaptation to meet the increased energy demands of pregnancy [22,23] With each successive pregnancy, the cumulative effects of these hormonal changes may lead to prolonged or exacerbated insulin resistance in some women. This could increase the risk of developing metabolic conditions such as gestational diabetes mellitus (GDM) or type 2 diabetes later in life [24] In this study has revealed a significant relationship between parity and insulin resistance, demonstrating that women with higher parity exhibited elevated levels of insulin resistance, whereas those with lower or no parity were found to have a reduced susceptibility to insulin resistance.

A comprehensive study conducted in the United States examined the association between parity and the risk of type 2 diabetes (T2DM). The findings demonstrated that each additional childbirth increased the risk of T2DM by 16% (HR: 1.16; 95% CI: 1.13–1.16). These results align with our study, further confirming that parity contributes significantly to the development of insulin resistance. Additionally, the study highlighted that women with obesity or abdominal obesity were at a heightened risk, while those with normal BMI or waist circumference exhibited no significant increase. These consistent outcomes reinforce the role of parity as a key factor in T2DM risk [25]. Another study conducted in Colombia involving 1,795 women evaluated the impact of parity on insulin sensitivity. Anthropometric characteristics were recorded, and the analysis revealed a positive association between parity and diabetes. This correlation remained significant after adjusting for age, body mass index (BMI), and a family history of diabetes in women with multiple childbirths compared to those with no childbirths (referent group).

The findings of this study align with our results, further reinforcing the evidence that parity plays a role in the development of insulin resistance and diabetes [26]. A study conducted in Ontario, Canada, between 2002 and 2011, included 738,440 women aged 18 to 50 years who delivered between April 1, 2002, and March 31, 2011. The incidence of postpartum diabetes was calculated for each parity category and ethnic group. The diabetes incidence rate per 1,000 person-years was 3.69 in women with one delivery, 4.12 in women with three deliveries, and 7.62 in women with ≥5 deliveries. Women with ≥3 deliveries exhibited a higher risk of diabetes compared to those with only one delivery. A similar increase in risk was observed among Chinese and South Asian women, with the highest influence noted in Chinese women. These findings are consistent with our study's results, further supporting the association between parity and an increased risk of developing diabetes [27].

Another study conducted in Denmark, which also aligns with our findings, included all Danish women who had a singleton delivery between 1982 and 1983 (n = 100,669) and subsequently gave birth to 74,966 children. These women were followed through national registries until the end of 2006 to monitor subsequent deliveries, diabetes diagnoses, or instances of death/emigration. This study further supports the evidence linking parity to an increased risk of developing diabetes [28]. A study

investigating the factors affecting insulin sensitivity suggested that obesity is the primary determinant. However, these findings do not align with the results of our study, which indicate that parity plays a significant role in influencing insulin sensitivity. While obesity undoubtedly impacts insulin sensitivity, it may not be the sole factor, as our research highlights the importance of other contributors such as parity [29,30]. Another study indicated that genetic factors are among the primary determinants influencing insulin sensitivity. However, it cannot be concluded that they are the sole contributing factor [31,32].

Conclusion:

The study's results indicate that increased parity is associated with heightened insulin resistance, as evidenced by higher HOMA-IR values in women with five or more deliveries. These findings underscore the need for further research to understand the biological mechanisms underlying this relationship and its potential implications for women's health. Such insights should be considered in strategies for managing insulin resistance among women with varying reproductive histories.

Recommendations:

- Further Research: Additional studies are needed to uncover the underlying mechanisms that connect parity with increased insulin resistance, focusing particularly on the hormonal and metabolic changes induced by pregnancy.
- Preventive Measures: Introduce specific interventions aimed at reducing insulin resistance risks in multiparous women, especially those with five or more deliveries.
- Regular Monitoring: Advocate for consistent clinical monitoring of insulin sensitivity and glucose metabolism among women with higher parity to ensure early detection and effective management of potential issues.
- Educational Campaigns: Raise public awareness about the impact of parity on metabolic health, emphasizing lifestyle adjustments such as healthy eating habits and physical activity to mitigate associated risks.
- Customized Care Plans: Promote personalized healthcare approaches for multiparous women, considering their elevated vulnerability to insulin resistance and related metabolic conditions.

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