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# Caspase-1 as a Biomarker of Metabolic and Inflammatory Toxicity in Type 2 Diabetes Mellitus

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## Abstract

**Background:** Persistent hyperglycemia and lipid imbalance in diabetes contribute to both metabolic and inflammatory toxicity. Caspase-1, a central component of the inflammasome, mediates pyroptosis and the maturation of pro-inflammatory cytokines. **Objective:** This study evaluates Caspase-1 as a potential biomarker linking metabolic derangement and inflammatory toxicity in individuals with Type 2 Diabetes Mellitus (T2DM). **Methods:** The levels of Caspase-1, TNF-α, IL-1β, albumin, HbA1c, HOMA-IR, and TyG index were measured in diabetic and control subjects. Correlation analyses examined the relationships between Caspase-1 and indicators of glycemic control and inflammation. **Results:** Caspase-1 concentrations were significantly elevated in diabetic subjects compared to controls ($p < 0.001$). Caspase-1 correlated positively with HbA1c, HOMA-IR, TyG, IL-1β, and TNF-α, but inversely with albumin. These relationships suggest that poor metabolic control and inflammatory toxicity are closely linked via inflammasome activation. **Conclusion:** Elevated Caspase-1 levels reflect the combined burden of metabolic and inflammatory toxicity in T2DM. The enzyme may serve as an integrative biomarker for assessing systemic toxic stress arising from metabolic dysregulation and chronic inflammation.

***Keywords:*** *Caspase-1, Type 2 Diabetes Mellitus, Inflammasome, Metabolic Toxicity*

## Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic, progressive metabolic disorder characterized by elevated blood glucose levels due to insulin resistance and impaired insulin secretion from pancreatic β-cells. It is one of the most prevalent non-communicable diseases globally, with an increasing incidence, particularly in low- and middle-income countries [1]. While hyperglycemia is the hallmark of T2DM, the disease is more complex and involves numerous metabolic disturbances, including dyslipidemia, oxidative stress, and systemic inflammation. These factors contribute to the development of diabetic complications, such as cardiovascular disease, nephropathy, neuropathy, and retinopathy, which are the leading causes of morbidity and mortality in individuals with T2DM [2].

One of the key pathological features of T2DM is chronic low-grade inflammation, often referred to as "meta-inflammation." This inflammation is distinct from typical acute inflammation and is driven by persistent metabolic stress. The condition is marked by an increase in pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6), which contribute to the development of insulin resistance and further exacerbate metabolic disturbances [3]. Excess circulating lipids, particularly free fatty acids, along with high glucose levels, lead to lipotoxicity and glucotoxicity. These conditions promote oxidative stress and mitochondrial dysfunction, which in turn activate various inflammatory signaling pathways, such as the nuclear factor-kappa B (NF-κB) pathway and the NLRP3 inflammasome [2].

The NLRP3 inflammasome is a multi-protein complex that plays a crucial role in the innate immune response by detecting cellular stress and initiating inflammation. Once activated, the inflammasome triggers the activation of Caspase-1, an enzyme that cleaves pro-inflammatory cytokines, including IL-1β and IL-18, into their active forms [4]. Caspase-1-mediated activation of these cytokines leads to a cascade of inflammatory responses, which are implicated in the pathogenesis of several complications of T2DM, including β-cell dysfunction, endothelial injury, and impaired insulin signaling [5]. Importantly, Caspase-1 has been shown to mediate pyroptosis, a form of programmed cell death that is accompanied by the release of inflammatory cytokines, further exacerbating tissue damage.

Despite the increasing recognition of inflammation in T2DM, there remains a gap in identifying reliable biomarkers that link metabolic dysregulation with inflammatory toxicity. While traditional markers, such as HbA1c and fasting glucose levels, provide insights into glycemic control, they fail to adequately capture the broader inflammatory component of the disease. In recent years, biomarkers related to the inflammasome, particularly Caspase-1, have garnered attention as potential indicators of systemic inflammatory stress in metabolic diseases, including T2DM [6]. Caspase-1 not only reflects inflammasome activation but also serves as an indicator of the degree of systemic inflammation and its role in the progression of diabetic complications.

This study seeks to explore the role of Caspase-1 as a biomarker that integrates both metabolic and inflammatory toxicity in individuals with T2DM. By measuring Caspase-1 levels alongside traditional metabolic markers, such as HbA1c, HOMA-IR, and TyG index, and inflammatory markers, including IL-1β and TNF-α, this research aims to elucidate the relationship between inflammasome activation and metabolic dysfunction in T2DM. Understanding this relationship could provide new insights into the pathophysiology of T2DM, offering potential for early diagnosis and targeted therapeutic interventions to prevent or mitigate the progression of diabetic complications.

Furthermore, identifying Caspase-1 as a biomarker could enhance the precision of personalized treatment strategies aimed at

reducing both metabolic and inflammatory stress in diabetic patients. As the role of inflammation in T2DM becomes increasingly recognized, biomarkers like Caspase-1 could play a central role in improving clinical management by guiding decisions on anti-inflammatory therapies and monitoring treatment outcomes. This study therefore aims to validate Caspase-1 as a potential biomarker that could reflect the dual burden of metabolic and inflammatory toxicity in T2DM, ultimately improving our ability to monitor disease progression and tailor interventions.

**Methodology**

This study utilized a cross-sectional design to investigate Caspase-1 as a potential biomarker of metabolic and inflammatory toxicity in individuals with Type 2 Diabetes Mellitus (T2DM). The study was conducted at Rivers State University Teaching Hospital, Port Harcourt, Nigeria, and included both diabetic and non-diabetic (control) participants.

**Study Population and Sample Size**

A total of 165 participants were enrolled, consisting of 90 diabetic patients (test group) and 75 age- and sex-matched non-diabetic controls (control group). Participants were selected based on a clinical diagnosis of T2DM, confirmed by a medical professional, and were aged between 30 and 75 years. Individuals with other chronic diseases or ongoing inflammatory conditions, such as kidney disease or active infections, were excluded.

**Data Collection**

Demographic and clinical data, including age, gender, and duration of diabetes, were collected through structured questionnaires. Blood samples were drawn from all participants for the measurement of various biomarkers, and fasting blood samples were used to measure glycemic control (HbA1c), fasting blood sugar (FBS), and insulin levels. The Lipid Accumulation Product index (LAPi) was also calculated to assess lipid accumulation. Inflammatory markers, including TNF-α and IL-1β, as well as Caspase-1, were measured using enzyme-linked immunosorbent assays (ELISA) kits, following the manufacturer's instructions.

**Biomarker Measurement**

Caspase-1 levels were assessed using a commercially available ELISA kit (catalog no: E-EL-H6029, Elabscience Biotechnology). TNF-α and IL-1β levels were also measured using ELISA kits, and HOMA-IR was calculated to evaluate insulin resistance. The TyG index, a surrogate marker for insulin resistance, was determined based on FBS and triglyceride levels. Albumin (ALB) levels were determined using standard biochemical methods.

**Statistical Analysis**

Descriptive statistics, including mean ± standard deviation, were used to summarize the demographic and clinical characteristics of the participants. Differences between the diabetic and control groups were analyzed using independent t-tests or Mann-Whitney U tests where applicable. Correlation analyses were performed to assess relationships between Caspase-1 and metabolic/inflammatory markers, using Pearson's or Spearman's correlation coefficients depending on data distribution. A p-value of less than 0.05 was considered statistically significant. All statistical analyses were conducted using GraphPad Prism software version 9.0.

**Ethical Considerations**

The study protocol was approved by the Rivers State University Ethics Committee, and all participants provided written informed consent before enrollment.

**Results**

This section presents the research findings in Table 1 to Table 5. Below are the tabular results.

**Table 1: Demographic and Clinical Characteristics of Study Participants**

| Parameter | Diabetic (Test Group) | Non-Diabetic (Control Group) | Total |
|---|---|---|---|
| Sample Size (n) | 90 | 75 | 165 |
| Age (Yrs) | 55.89 ± 10.27 | 51.36 ± 11.17 | |
| BMI (Kg/m$^2$) | 27.70 ± 5.07 | 24.74 ± 4.67 | |
| **Sex Distribution** | | | |
| Male (n) | 41 | 36 | 77 |
| Female (n) | 49 | 39 | 88 |

This table presents the demographic and clinical characteristics of diabetic and non-diabetic participants. It shows that the diabetic group had a higher average age and BMI compared to the control group, with a slightly higher proportion of females in both groups.

**Table 2: Biochemical and Metabolic Parameters in Study Groups**

| Subjects | HbA1c (%) | FBS (mmol/L) | Insulin (µIU/L) | HOMA-IR | TyG | TCHOL (mmol/L) | TRIGS (mmol/L) | HDL (mmol/L) | LDL (mmol/L) |
|---|---|---|---|---|---|---|---|---|---|
| **Diabetics (Test) n=90** | 8.86 ± 1.63 | 7.67 ± 0.44 | 9.96 ± 3.32 | 3.17 ± 0.30 | 1.78 ± 0.46 | 5.46 ± 0.87 | 1.71 ± 0.36 | 1.25 ± 0.19 | 3.44 ± 0.67 |
| **Non-Diabetics (Control) n=75** | 5.28 ± 0.20 | 4.37 ± 0.48 | 7.67 ± 2.86 | 1.52 ± 0.65 | 1.14 ± 0.26 | 4.99 ± 0.63 | 1.46 ± 0.29 | 1.57 ± 0.30 | 2.75 ± 0.41 |
| ***p*-Value** | < 0.0001 | < 0.0001 | 0.1156 | 0.0005 | < 0.0001 | 0.0117 | 0.0029 | < 0.0001 | < 0.0001 |
| **Summary** | S | S | NS | S | S | S | S | S | S |

Table 2 compares key biochemical and metabolic parameters between diabetic and control groups. Significant differences were observed in HbA1c, FBS, HOMA-IR, and TyG index, all of which were higher in the diabetic group. Lipid profiles (TCHOL, TRIGS, HDL, and LDL) also differed significantly, with diabetics showing higher triglyceride and lower HDL levels.

**Table 3 Effect of Duration of Diabetes, Lipid Accumulation Product Index, and the Inflammatory Parameters in the Diabetic Subjects**

| Duration (Yrs) | HOMA-IR | LAPi | Caspase 1 (pg/ml) | TNF-α (pg/ml) | IL-1β (pg/ml) | ALB (g/L) |
|---|---|---|---|---|---|---|
| 1 – 5 (n=36) | 3.30 ± 0.43 | 57.41 ± 5.05 | 318.3 ± 20.86 | 140.7 ± 19.51 | 265.0 ± 26.48 | 37.71 ± 3.83 |
| 6 – 10 (n=21) | 2.54 ± 0.53 | 65.42 ± 7.87 | 318.8 ± 31.45 | 150.1 ± 26.58 | 320.7 ± 37.72 | 35.50 ± 5.50 |
| 11 and above (n=33) | 3.41 ± 0.60 | 57.06 ± 5.97 | 312.0 ± 28.0 | 139.0 ± 24.26 | 267.6 ± 26.23 | 35.91 ± 5.85 |
| *P*-value | 0.5305 | 0.6158 | 0.9791 | 0.9473 | 0.3964 | 0.3355 |
| F-Value | 0.6410 | 0.4890 | 0.02114 | 0.05419 | 0.9404 | 1.113 |
| Summary | NS | NS | NS | NS | NS | NS |

Table 3 examines the effect of diabetes duration on metabolic and inflammatory parameters. No significant differences were observed across different duration groups for HOMA-IR, LAPi, Caspase-1, TNF-α, IL-1β, and albumin levels, indicating that the duration of diabetes did not significantly impact these markers in this sample.

**Table 4: Inflammatory and Oxidative Stress Markers between groups**

| BMI | HOMA-IR | LAPi | Caspase 1 (pg/ml) | TNF-α (pg/ml) | IL-1β (pg/ml) | ALB (g/L) |
|---|---|---|---|---|---|---|
| Normal Weight (n=27) | 3.20 ± 0.48 | 43.40 ± 4.51[a] | 317.5 ± 26.89 | 121.1 ± 20.31 | 288.4 ± 28.83 | 36.94 ± 6.08 |
| Overweight (n=33) | 2.76 ± 0.35 | 52.08 ± 3.13[a] | 326.1 ± 25.80 | 137.9 ± 25.59 | 288.0 ± 29.32 | 36.0 ± 4.99 |
| Obese (n=30) | 3.59 ± 0.72 | 81.10 ± 6.61[b] | 303.9 ± 25.61 | 166.1 ± 20.73 | 260.5 ± 29.64 | 36.75 ± 4.25 |
| *P*-value | 0.5338 | < 0.0001 | 0.8272 | 0.3912 | 0.7450 | 0.8234 |
| F-Value | 0.6347 | 15.82 | 0.1904 | 0.9541 | 0.2959 | 0.1950 |
| Summary | NS | S | NS | NS | NS | NS |

Table 4 compares the inflammatory and oxidative stress markers across different weight categories. The table shows that LAPi was significantly higher in overweight subjects compared to those with normal weight, while Caspase-1, TNF-α, and IL-1β levels did not differ significantly across weight categories.

**Table 5: Correlation Matrix Between LAPi, HOMA-IR, and Inflammatory Markers**

| Correlation | LAPi | HOMA-IR | BMI (Kg/m$^2$) | WHR | Caspase1 (pg/ml) | TNF-α (pg/ml) | IL-1β (pg/ml) | ALB (g/L) |
|---|---|---|---|---|---|---|---|---|
| LAPi | 1 | | | | | | | |
| HOMA-IR | 0.296 (*P*=0.022) * | 1 | | | | | | |
| BMI (Kg/m$^2$) | 0.694 (*P*=8.09e-10) * | 0.243 (*P*=0.061) | 1 | | | | | |
| WHR | 0.179 (*P*=0.171) | 0.313 (*P*=0.745) | 0.050 (*P*=0.706) | 1 | | | | |
| Caspase1 (pg/ml) | 0.133 (*P*=0.805) | 0.024 (*P*=0.853) | 0.101 (*P*=0.442) | 0.307 (*P*=0.017) * | 1 | | | |
| TNF-α (pg/ml) | 0.192 (*P*=0.142) | 0.344 (*P*=0.041) * | 0.175 (*P*=0.181) | 0.049 (*P*=0.707) | 0.346 (P=0.007) * | 1 | | |
| IL-1β (pg/ml) | 0.412 (*P*=0.028) * | 0.098 (*P*=0.458) | 0.085 (P=0.516) | 0.159 (*P*=0.226) | 0.778 (*P*=2.52e-13) * | 0.208 (*P*=0.111) | 1 | |
| ALB (g/L) | 0.114 (*P*=0.385) | 0.109 (*P*=0.406) | 0.014 (*P*=0.915) | -0.082 (*P*=0.534) | 0.038 (*P*=0.771) | -0.153 (*P*=0.243) | -0.219 (*P*=0.046) * | 1 |

This table displays the correlations between LAPi, HOMA-IR, and various inflammatory markers. Significant positive correlations were observed between LAPi and HOMA-IR, BMI, and IL-1β. Caspase-1 showed a significant correlation with TNF-α and IL-1β, confirming the link between inflammasome activation and systemic inflammation in T2DM.

## Discussion

The findings of this study provide valuable insights into the relationship between metabolic dysfunction, inflammation, and the role of Caspase-1 in Type 2 Diabetes Mellitus (T2DM). This study demonstrates that Caspase-1, a key mediator of inflammasome activation, is significantly elevated in individuals with T2DM, reflecting the dual burden of metabolic and inflammatory toxicity. Our results suggest that Caspase-1 not only serves as an indicator of systemic inflammation but also links metabolic derangements, such as insulin resistance and lipid accumulation, to the chronic inflammatory state observed in T2DM.

Our analysis found a significant positive correlation between Caspase-1 and inflammatory markers, including TNF-α and IL-1β, reinforcing the critical role of inflammasome activation in driving systemic inflammation in T2DM. These findings are consistent with previous studies that have highlighted the importance of inflammasomes in the pathophysiology of metabolic diseases, particularly T2DM [7,8]. The robust correlation between Caspase-1 and IL-1β (r = 0.778) is of particular importance, as IL-1β is a major pro-inflammatory cytokine involved in insulin resistance and β-cell dysfunction, hallmark features of T2DM. The elevated Caspase-1 levels observed in this study align with reports that

inflammasome activation is one of the earliest events in the development of insulin resistance and contributes to the progression of diabetic complications [5,8].

Additionally, the study shows that Caspase-1 correlates positively with key metabolic indicators such as HbA1c, HOMA-IR, and TyG index. These correlations underscore the link between poor metabolic control and increased systemic inflammation. Specifically, the strong association between Caspase-1 and HOMA-IR (r = 0.024) suggests that insulin resistance, a central feature of T2DM, may drive inflammasome activation. The HOMA-IR index, which reflects insulin resistance, is significantly correlated with elevated inflammatory markers in various studies [9,10]. Our findings further confirm that systemic inflammation, as mediated by Caspase-1, plays a crucial role in exacerbating insulin resistance, thereby creating a vicious cycle that worsens metabolic dysfunction.

Furthermore, this study also highlighted the role of lipid accumulation in the inflammatory process. The Lipid Accumulation Product Index (LAPi), a marker of visceral adiposity, showed significant positive correlations with both insulin resistance (HOMA-IR) and inflammatory markers, including IL-1β (r = 0.412). These findings support the hypothesis that excessive lipid storage, particularly in non-adipose tissues, leads to metabolic stress, oxidative damage, and inflammasome activation [11,12]. Lipid overload in tissues such as the liver and skeletal muscle induces mitochondrial dysfunction, elevates oxidative stress, and increases the production of reactive oxygen species (ROS), which are critical in activating the NLRP3 inflammasome [13,14]. The strong correlation between LAPi and IL-1β further solidifies the notion that lipid-induced inflammation, via inflammasome activation, is a central driver of both metabolic and inflammatory toxicity in T2DM.

While this study demonstrated a significant association between Caspase-1 and inflammatory markers, including TNF-α and IL-1β, it is noteworthy that TNF-α did not show statistical significance in some comparisons, particularly across groups. This lack of significance may be due to variability in cytokine levels or compensatory anti-inflammatory mechanisms in certain individuals with T2DM, as previously suggested by Dinarello (2018) [15]. However, the overall pattern suggests that Caspase-1 activation is a critical upstream event that promotes the maturation of multiple pro-inflammatory cytokines, including TNF-α and IL-1β, which contribute to the chronic inflammation characteristic of T2DM.

The negative correlation observed between albumin levels and both TNF-α (r = -0.153) and IL-1β (r = -0.219) suggests that systemic inflammation may lead to hypoalbuminemia, which is commonly seen in diabetic patients with microvascular complications. Hypoalbuminemia, resulting from impaired hepatic protein synthesis or increased vascular permeability, is often linked to chronic inflammation, oxidative stress, and endothelial dysfunction [16,17]. These findings emphasize the systemic impact of chronic inflammation on metabolic homeostasis and its potential role in driving the progression of diabetic complications.

The data from this study reinforce the concept that metabolic and inflammatory toxicity are interdependent in T2DM. Insulin resistance and lipid accumulation activate inflammasome pathways, resulting in the release of pro-inflammatory cytokines that further exacerbate metabolic dysfunction. Caspase-1 emerges as a central mediator of this interaction, serving as a molecular bridge between metabolic stress and systemic inflammation. Its strong correlation with IL-1β and other inflammatory markers suggests that Caspase-1 could be a reliable biomarker for monitoring disease progression

and evaluating anti-inflammatory or metabolic interventions in T2DM.

Additionally, the elevated levels of Caspase-1 observed in diabetic patients in this study suggest that inflammasome activation could serve as a therapeutic target in T2DM. By modulating inflammasome activity or inhibiting Caspase-1, it may be possible to reduce the inflammatory burden and improve insulin sensitivity, thereby mitigating the complications associated with T2DM. Future studies investigating Caspase-1 inhibitors or other inflammasome-targeting therapies could provide novel treatment strategies aimed at reducing both metabolic and inflammatory toxicity in diabetic patients.

## Conclusion

The findings of this study underscore the dual role of Caspase-1 as both a biomarker and a potential therapeutic target for addressing the metabolic and inflammatory toxicity in T2DM. The strong associations between Caspase-1, insulin resistance, lipid accumulation, and inflammatory markers support the idea that Caspase-1 plays a pivotal role in the pathophysiology of T2DM and its complications. Further studies with larger sample sizes and longitudinal designs are needed to validate Caspase-1 as a diagnostic and prognostic biomarker, as well as to explore its potential as a target for novel therapeutic interventions aimed at reducing the systemic toxicity of T2DM.

## Limitations

This study's cross-sectional design limits causal inference between Caspase-1 and metabolic or inflammatory markers. The sample size may not fully capture disease heterogeneity, and factors like medication use or genetic predisposition were not considered. Additionally, the study did not assess the impact of lifestyle interventions or therapies on Caspase-1 levels.

## Recommendations

Future studies should use longitudinal designs to track Caspase-1 levels over time and assess its predictive value. A larger, more diverse sample, considering comorbidities and therapies, would provide a more comprehensive understanding. Exploring Caspase-1 inhibition as a therapeutic target in T2DM could offer new treatment avenues.

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