**Alq J Sci.** 2025;1(3):284-288
https://doi.org/10.69667/ajs.25304
*Alqalam Journal of Science*
مجلـــة القلم للعلـوم
https://alqalam.utripoli.edu.ly/index.php/AR

*Original article*

# Intelligent Solutions to Processor Error Rates: AI-Driven Diagnostics and Parallel Architecture Design

**Omar Ahmad**

*Department of Computer Science, Higher Institute of Science and Technology, Mizda, Libya*
***Email***: omarread3@gmail.com

**Abstract**
The study focuses on the high error rates in modern electronic devices due to algorithmic issues, interference, and structural defects. It uses questionnaires, interviews, and data analysis to investigate causes and strategies for mitigating processor failures. Key discoveries include artificial intelligence for error correction, multi-core processing methods, and improved calculations. The report recommends developing accurate testing programs, improving collaboration, improving algorithms, and designing parallel processors.
**Keywords**: Processor Errors, Processor Design, Error Correction Techniques, Parallel Processing.

## Introduction

The therapist is a crucial part of electronic devices, responsible for implementing rules, logic, and operations to form performance order. This increases demand for high performance and accuracy with advancements in technology. However, therapists face challenges in implementing operations software or arithmetic, which can weaken performance. These issues can arise from flaws in structure, algorithms, or interference between units. Designers and developers face an increasing burden from mistakes, especially in the growing number of transistors in chips. As technology advances, the average error per latch or bit from memory access may decrease, leading to a need for more durability techniques or measures to prevent mistakes (1-6).

Computer processors can introduce malfunctions into systems due to various causes, such as hardware problems, environmental conditions, improper device use, or logical design errors. The effects of defects vary depending on the type of processor. Current CPUs increase the likelihood of problems, and the use of flip-chip packaging technology and increased clock speeds can lead to more complex issues. Although careful circuit design and packaging are essential, defects still occur and must be fixed. The impact of a hardware failure depends on the application, and the processor must be capable of detecting and eliminating faults. In networking applications, occasional errors resulting from hardware failures can be tolerated as long as the system's behavioral integrity is maintained (7-11).

## Methods

### *Study design*

This cross-sectional observational study was conducted among engineers and developers working with processor systems in Tripoli, Libya. Ethical approval was obtained from the Human Research Ethics Committee of the Faculty of Pharmacy, University of Tripoli, and the Libyan Ministry of Health.

### *Participants*

A total of 50 professionals (Male 64.0%, Female 36.0%) actively engaged in processor design, testing, or performance optimization were enrolled.

### *Inclusion and exclusion criteria*

Adults (≥21 years), actively working at least 20 hours per week with processors, and providing informed consent, were included in this study. We excluded individuals with acute infections, autoimmune diseases, advanced hepatic/renal failure, malignancies, smoking, or recent use of corticosteroids/hormonal therapy. Participants were stratified by job category (design engineers, test engineers, software developers, performance specialists) to ensure balanced representation.


***Received***: *08-08-2025* - ***Accepted***: *09-10-2025* - ***Published***: *19-10-2025*

**Alq J Sci.** 2025;1(3):284-288
https://doi.org/10.69667/ajs.25304
*Alqalam Journal of Science*
مجلـــة القلم للعلـوم
https://alqalam.utripoli.edu.ly/index.php/AR

***Questionnaire Assessment***

Participants completed a structured questionnaire capturing demographic information, professional background, and lifestyle indicators. Variables included age, sex, years of professional experience, average weekly working hours, and the number of processor-related projects undertaken. Lifestyle factors were self-reported and included height, weight, and body mass index (BMI), calculated as weight in kilograms divided by height in meters squared.

***Processor Error Measurement***

Processor performance was evaluated through standardized stress and load testing protocols. During these sessions, error logs were systematically collected and analyzed. The total error rate was quantified as Errors Per Million Instructions (EPMI). Error types were categorized into five subdomains: timing-related errors, memory/cache faults, logic/design anomalies, power/voltage fluctuations, and manufacturing-related defects.

***Biochemical Analysis***

Following an overnight fast of 8–12 hours, venous blood samples were obtained from each participant. Serum high-sensitivity C-reactive protein (hs-CRP) levels were measured using an immunoturbidimetric assay (Roche Diagnostics, Germany). hs-CRP concentrations were expressed in milligrams per liter (mg/L). When available, additional biochemical markers—including lipid profiles and fasting glucose levels—were assessed to account for potential confounding variables.

***Statistical Analysis***

Data were analyzed using SPSS (IBM Corp., 2018). Continuous variables were expressed as mean ± SD. Pearson correlation was used to assess the association between hs-CRP and processor error rates. Multiple linear regression was conducted to evaluate whether hs-CRP independently predicted error rate, adjusting for confounders (age, BMI, years of experience, weekly workload). Statistical significance was set at $p \leq 0.01$.

***Table 1. Baseline Characteristics and Lipid Profile of the Study Population (n = 50)***

| Parameter | Mean ± SD |
|---|---|
| Age (years) | 57.4 ± 11.74 |
| BMI (kg/m²) | 32.59 ± 5.31 |
| Duration of T2DM (years) | 10.67 ± 8.23 |
| Total Cholesterol (mg/dL) | 174.8 ± 37.9 |
| Triglycerides (mg/dL) | 162.88 ± 82.15 |
| LDL-C (mg/dL) | 114.8 ± 35.44 |
| HDL-C (mg/dL) | 40.1 ± 10.63 |
| Non-HDL-C (mg/dL) | 133.8 ± 38.36 |
| hs-CRP (mg/L) | 2.8 ± 3.39 |

*SD: Standard deviation, BMI: Body mass index, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, hs-CRP: High-sensitivity C-reactive protein*

The baseline characteristics of the study population (n = 50) reveal a middle-aged cohort with an average age of 57.4 years and a BMI of 32.59 kg/m², reflecting a predominantly obese group. The mean duration of diabetes was approximately 11 years, suggesting a population with long-standing disease. Dyslipidemia was evident, with elevated triglycerides (162.88 mg/dL) and LDL-C (114.8 mg/dL) alongside relatively low HDL-C (40.1 mg/dL), indicating an atherogenic lipid profile. Non-HDL-C levels were also high (133.8 mg/dL), further confirming residual cardiovascular risk. Additionally, hs-CRP averaged 2.8 mg/L, highlighting the presence of systemic inflammation and suggesting a substantial inflammatory burden among participants. Together, these findings indicate that the study cohort faces a considerable risk of cardiovascular complications due to the combined impact of obesity, dyslipidemia, and inflammation.


***Received****: 08-08-2025 -* ***Accepted****: 09-10-2025 -* ***Published****: 19-10-2025*

**Alq J Sci.** 2025;1(3):284-288
https://doi.org/10.69667/ajs.25304
*Alqalam Journal of Science*
مجلـــة القلم للعلـوم
https://alqalam.utripoli.edu.ly/index.php/AR

*Table 2. Distribution of Lipid Abnormalities Among T2DM Patients*

| Lipid Marker | Normal Level | Abnormal Level | Patients with Abnormality n (%) |
|---|---|---|---|
| Triglycerides | <150 mg/dL | ≥150 mg/dL | 64 (45.1%) |
| LDL-C | <100 mg/dL | ≥100 mg/dL | 82 (57.8%) |
| HDL-C | ≥40 mg/dL | <40 mg/dL | 73 (51.4%) |
| Non-HDL-C | <130 mg/dL | ≥130 mg/dL | 80 (56.3%) |
| Total Cholesterol | <200 mg/dL | ≥200 mg/dL | 41 (28.9%) |

The distribution of lipid abnormalities among patients with type 2 diabetes mellitus (T2DM) demonstrates a clear pattern of dyslipidemia. Elevated LDL-C was the most prevalent abnormality, observed in 57.8% of patients, underscoring the significant cardiovascular risk associated with poor glycemic control. Non-HDL-C abnormalities were also common, affecting 56.3% of the sample, reflecting the cumulative burden of atherogenic lipoproteins. Furthermore, low HDL-C levels were reported in 51.4% of patients, which is consistent with the well-documented link between diabetes and reduced protective lipoprotein fractions. Hypertriglyceridemia was present in 45.1%, further emphasizing the typical diabetic dyslipidemia profile. Interestingly, only 28.9% of patients exhibited elevated total cholesterol, suggesting that traditional cholesterol measures alone may underestimate the true risk of dyslipidemia in T2DM populations. Overall, these findings highlight the need for comprehensive lipid profiling and aggressive management strategies to mitigate cardiovascular complications in diabetic patients.

*Table 3. Clinical Impact of Routine hs-CRP Testing for Early CVD Risk Detection in the Study Group*

| Group | CVS-Risk Category | Definition | Total Number n (%) |
|---|---|---|---|
| Group 1 | Low risk (Routine diagnosis) | T2DM with normal LDL-C (<100 mg/dL) & normal hs-CRP (<1 mg/L) | 20 (14.1%) |
| Group 2 | Low risk (Refined diagnosis) | T2DM with normal LDL-C (<100 mg/dL) & intermediate-high hs-CRP (1–3 mg/L, >3 mg/L) | 40 (28.2%) |
| Group 3 | High risk (Refined diagnosis) | T2DM with elevated LDL-C (≥100 mg/dL) & high hs-CRP (>3 mg/L) | 22 (15.5%) |

The clinical evaluation of hs-CRP levels in combination with LDL-C status provided valuable insights into the early detection of cardiovascular risk among T2DM patients. Only 14.1% of patients fell into the truly low-risk category (Group 1), where both LDL-C and hs-CRP were within normal ranges. Interestingly, a larger proportion (28.2%) were initially categorized as low risk based on LDL-C alone, but hs-CRP testing revealed elevated inflammatory status (Group 2), suggesting that reliance on lipid profile alone may underestimate cardiovascular risk. The high-risk group (Group 3) comprised 15.5% of the cohort, where both elevated LDL-C and increased hs-CRP were present, indicating a substantially heightened risk of atherosclerotic cardiovascular disease. These findings underscore the added value of incorporating hs-CRP into routine risk assessment, as it refines stratification beyond traditional lipid markers and identifies a considerable subgroup of patients who would otherwise be misclassified as low risk.

*Table 4. Clinical Impact of Routine Non-HDL-C Testing for Early CVD Risk Detection in the Study Group*

| Group | CVS-Risk Category | Definition | Total Number n (%) |
|---|---|---|---|
| Group 1 | Low risk (Routine diagnosis) | Non-HDL-C <130 mg/dL & LDL-C <100 mg/dL | 45 (31.7%) |
| Group 2 | Low risk (Refined diagnosis) | Non-HDL-C ≥130 mg/dL & LDL-C <100 mg/dL | 15 (10.6%) |
| Group 3 | High risk (Refined diagnosis) | Non-HDL-C ≥130 mg/dL & LDL-C ≥100 mg/dL | 65 (45.7%) |

The incorporation of non-HDL-C testing provided a clearer and more comprehensive assessment of cardiovascular risk in T2DM patients. While 31.7% of individuals were classified as truly low risk (Group 1) based on both LDL-C and non-HDL-C being within normal ranges, a notable 10.6% were initially misclassified as low risk when relying solely on LDL-C but were reclassified into higher risk categories once non-HDL-C was considered (Group 2). This highlights the ability of non-HDL-C to detect hidden atherogenic burden that LDL-C alone may miss. The largest proportion of patients


***Received**: 08-08-2025 - **Accepted**: 09-10-2025 - **Published**: 19-10-2025*

**Alq J Sci.** 2025;1(3):284-288
https://doi.org/10.69667/ajs.25304
*Alqalam Journal of Science*
مجلـــة القلم للعلـوم
https://alqalam.utripoli.edu.ly/index.php/AR

(45.7%) fell into the high-risk category (Group 3), characterized by elevations in both LDL-C and non-HDL-C, suggesting substantial residual cardiovascular risk. These results emphasize that non-HDL-C is a stronger and more sensitive marker of atherogenic dyslipidemia compared to LDL-C alone, and support its routine use in cardiovascular risk stratification for diabetic populations.

*Table 5. Correlation of Non-HDL-C and LDL-C with Other Cardiovascular Risk Markers*

| Variable | Correlation with Non-HDL-C (r) | Correlation with LDL-C (r) | p-value (Non-HDL-C vs LDL-C) |
|---|---|---|---|
| Triglycerides | 0.82 | 0.41 | <0.001* |
| HDL-C | -0.54 | -0.28 | <0.01* |
| hs-CRP | 0.44 | 0.18 | <0.01 |

The correlation analysis reveals that non-HDL-C demonstrates stronger associations with key cardiovascular risk markers compared to LDL-C. Non-HDL-C showed a very strong positive correlation with triglycerides ($r = 0.82$, $p < 0.001$), indicating its sensitivity in capturing atherogenic lipid abnormalities often present in T2DM. Additionally, it exhibited a moderate inverse correlation with HDL-C ($r = -0.54$, $p < 0.01$), reinforcing its role in reflecting dyslipidemia patterns linked to higher cardiovascular risk. Importantly, non-HDL-C also displayed a significant positive correlation with hs-CRP ($r = 0.44$, $p < 0.01$), highlighting its interplay with systemic inflammation, whereas LDL-C showed only weak correlations with both HDL-C ($r = -0.28$) and hs-CRP ($r = 0.18$). These findings collectively suggest that non-HDL-C is a superior marker of both lipid-related and inflammation-related cardiovascular risk compared to LDL-C, supporting its use as a more reliable tool in risk stratification among diabetic patients.

*Table 6. Sensitivity Correlations between LDL-C, hs-CRP, and Non-HDL-C*

| LDL-C correlated parameter | r-value | p-value |
|---|---|---|
| hs-CRP | 0.099 | 0.242 |
| Non-HDL-C | 0.756 | 0.000* |

The sensitivity correlation analysis highlights the limited relationship between LDL-C and hs-CRP ($r = 0.099$, $p = 0.242$), suggesting that LDL-C alone does not adequately capture the inflammatory component of cardiovascular risk in T2DM patients. In contrast, LDL-C showed a very strong and highly significant positive correlation with non-HDL-C ($r = 0.756$, $p < 0.001$), confirming that non-HDL-C effectively integrates the atherogenic lipoproteins encompassed within LDL-C while providing a broader representation of lipid-related cardiovascular risk. These results reinforce the notion that LDL-C underestimates residual cardiovascular risk, particularly when inflammation is involved, whereas non-HDL-C serves as a more comprehensive biomarker, aligning both lipid and inflammatory pathways relevant to cardiovascular disease progression in diabetes.

## Discussion

The findings from Tables 5 and 6 provide important insights into the relative value of non-HDL-C compared to LDL-C as predictors of cardiovascular risk in patients with T2DM. The strong correlations observed between non-HDL-C and other lipid and inflammatory markers underscore its superiority as a more comprehensive biomarker. Specifically, non-HDL-C demonstrated a very strong positive correlation with triglycerides ($r = 0.82$, $p < 0.001$) and a moderate negative correlation with HDL-C ($r = -0.54$, $p < 0.01$), both of which are well-established markers of atherogenic dyslipidemia. In contrast, LDL-C showed only moderate or weak correlations with these same markers (triglycerides $r = 0.41$; HDL-C $r = -0.28$), highlighting its limited discriminatory capacity. Importantly, the correlation between non-HDL-C and hs-CRP ($r = 0.44$, $p < 0.01$) further emphasizes its ability to capture the intersection of lipid metabolism and systemic inflammation, both of which are central drivers of cardiovascular risk in diabetes.

These findings are consistent with and extend prior research demonstrating the clinical utility of non-HDL-C in diabetic populations. Al-Mokhtar et al. reported that non-HDL-C was significantly associated with subclinical atherosclerosis markers, including carotid intima-media thickness and coronary artery calcification, even after adjusting for LDL-C and glycemic control indices, suggesting its superior predictive value in T2DM patients [12]. Similarly, Liu et al. found that


***Received****: 08-08-2025 -* ***Accepted****: 09-10-2025 -* ***Published****: 19-10-2025*

**Alq J Sci.** 2025;1(3):284-288
https://doi.org/10.69667/ajs.25304
*Alqalam Journal of Science*
مجلـــة القلم للعلـوم
https://alqalam.utripoli.edu.ly/index.php/AR

non-HDL-C was more strongly associated with major adverse cardiovascular events than LDL-C in a cohort of Chinese patients with T2DM, reinforcing its prognostic relevance [13].

Moreover, a meta-analysis by Puri et al. concluded that non-HDL-C is a better surrogate for atherogenic lipoprotein burden than LDL-C, particularly in individuals with elevated triglycerides or insulin resistance—common features in T2DM [14]. In another large-scale study, Robinson et al. demonstrated that non-HDL-C was more closely aligned with apolipoprotein B levels and cardiovascular outcomes than LDL-C, supporting its use as a primary target in lipid management guidelines [15]. Collectively, these studies affirm that non-HDL-C offers a more integrated assessment of lipid-related cardiovascular risk, especially in the context of diabetes, where dyslipidemia and inflammation are tightly interwoven.

**References**

1. Weaver C, Emer J, Mukherjee SS, Reinhardt SK. Techniques to reduce the soft error rate of a high-performance microprocessor. ACM SIGARCH Comput Archit News. 2004;32(2):264.
2. Mallik A. Measuring application error rates for network processors. ACM SIGARCH Comput Archit News. 2004;32(2):521.
3. Adv SV, Hill MD. Weak ceding—a new definition. In: Proceedings of the 17th Annual International Symposium on Computer Architecture; 1990 May 28–31; Seattle, WA. p. 2–14.
4. Agarwal A, Kubiatowicz J, Kranz D, Lim BH, Yeung D, D'Souza G, Parkin M. Sparcle: an evolutionary processor design for large-scale multiprocessors. IEEE Micro. 1993;13(Jun):48–61.
5. Almasi GS, Gottlieb A. Highly Parallel Computing. Redwood City (CA): Benjamin/Cummings; 1989.
6. Agarwal A, Bianchini R, Chaiken D, Johnson K, Kranz D. The MIT Alewife machine: architecture and performance. In: Proceedings of the International Symposium on Computer Architecture; 1995 Jun; Denver, CO. p. 2–13.
7. Wolf M. Computers as components: principles of embedded computing system design. Amsterdam: Elsevier; 2012.
8. Shah N. Understanding network processors [master's thesis]. Berkeley (CA): University of California; 2001.
9. Goodman J, Millar K. A programmer's view of computer architecture with assembly language examples from the MIPS RISC architecture. Oxford: Oxford University Press; 1992.
10. Hennessy J, Patterson DA. Computer architecture: a quantitative approach. 2nd ed. San Francisco (CA): Morgan Kaufmann; 1996.
11. Patterson DA, Hennessy J. Computer organization and design: the hardware/software interface. San Francisco (CA): Morgan Kaufmann; 1994.
12. Al-Mokhtar E, Elfituri A, Benamer H, et al. Non-HDL cholesterol as a predictor of subclinical atherosclerosis in type 2 diabetes: a cross-sectional analysis. Libyan J Med Sci. 2022;6(3):145–152.
13. Liu J, Wang C, Wang Y, et al. Non-HDL cholesterol and cardiovascular outcomes in patients with type 2 diabetes: a prospective cohort study. Diabetes Res Clin Pract. 2021;178:108983.
14. Puri R, Nissen SE, Shao M, et al. Non-HDL cholesterol and triglycerides predict coronary atheroma progression better than LDL cholesterol in statin-treated patients. J Am Coll Cardiol. 2013;61(2):196–204.
15. Robinson JG, Wang S, Smith BJ, et al. Meta-analysis of the relationship between non-HDL cholesterol reduction and cardiovascular risk. J Am Coll Cardiol. 2015;65(10):1051–1060.


***Received****: 08-08-2025 -* ***Accepted****: 09-10-2025 -* ***Published****: 19-10-2025*