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Research Article

Correlation of Metabolic Risk Factor Clusters With Low-Grade Inflammation: A Cross-Sectional Clinical-Immunological Study of Adult Patients

MAITHAM ABDALLAH ALBAJY
Faculty of Biology, University of Bucharest, 91–95 Splaiul Independenței Str., 050095 Bucharest, Romania
National Center for Occupational Health and Safety, 22 Imam Ali Str., Nasiriyah 64001, Iraq
Corresponding Author: albajy.maitham@s.bio.unibuc.ro

Abstract: Immunometabolism refers to the reciprocal relationship between metabolic impairment and immune-inflammatory processes. Obesity, dyslipidemia, inadequate glycemic control, and hypertension contribute to persistent low-grade inflammation; yet, regular clinical datasets are underutilized for practical immunometabolic risk assessment.

Objective: This study examined the correlation between metabolic risk clustering and low-grade inflammatory activity, and assessed the effectiveness of a composite Immunometabolic Risk Score (IMRS) as a practical tool for identifying persons at heightened immunometabolic risk.

Methods: A retrospective cross-sectional research was conducted utilizing data from 110 persons. Demographic, anthropometric, blood pressure, lipid profile, glucose, white blood cell count (WBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) factors were examined. An Immunometabolic Risk Score (IMRS; range 0-6) was developed utilizing obesity, hypertriglyceridemia, low HDL-C, raised LDL-C, hyperglycemia, and increased blood pressure. Spearman correlation, non-parametric group comparisons, and multivariable logistic regression were employed.

Results: The group comprised 47 males (42.7%) and 63 females (57.3%), with a mean age of 52.60 ± 8.54 years. Ninety-two patients (83.6%) exhibited a high IMRS (≥4), while 26 patients (23.6%) had a CRP level over 3 mg/L. CRP exhibited a positive correlation with triglycerides (rho=0.468, p<0.001), LDL-C (rho=0.439, p<0.001), glucose (rho=0.313, p<0.001), and IMRS (rho=0.313, p<0.001), while demonstrating a negative correlation with HDL-C (rho=-0.344, p<0.001). CRP exhibited a substantial variation among IMRS groups (p=0.002). The ESR had a diminished positive correlation with IMRS (rho=0.213, p=0.026). The composite IMRS exhibited substantial correlations with inflammatory burden, indicating its potential as a practical screening instrument for immunometabolic dysfunction in standard clinical practice.

Conclusion: Metabolic risk clusters correlated with low-grade inflammatory activity, notably an increase in CRP levels. These findings underscore the clinical significance of immunometabolic profiling by standard laboratory indicators, while emphasizing the necessity for prospective validation with a wider array of immunological biomarkers.

Keywords: *immunometabolism; low-grade inflammation; CRP; ESR; dyslipidemia; obesity; glucose; metabolic risk; clinical immunology*

1. Introduction

The immune and metabolic systems are intricately linked. Immunometabolism, a burgeoning domain in clinical immunology, investigates the regulation of immune cell behavior by metabolic pathways and the impact of inflammatory responses on systemic metabolism (O'Neill et al., 2016; Hotamisligil, 2017; Buck et al., 2017; Hu et al., 2024). This idea is especially pertinent in adult populations exhibiting obesity, dyslipidemia, hypertension, and compromised glucose regulation.

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Corresponding Author: MAITHAM ABDALLAH ALBAJY

Metaflammation is typically characterized as persistent, low-grade inflammation triggered by dietary and metabolic stressors. Current data associates obesity and glucolipid diseases with innate immune activation, inflammation of adipose tissue, cytokine generation, and systemic cardiometabolic problems (Hotamisligil, 2006; Gregor & Hotamisligil, 2011; Donath & Shoelson, 2011; Schleh et al., 2023). Adipose tissue serves as an active immunological organ, wherein adipocytes and local immune cells facilitate the secretion of inflammatory mediators, including IL-6, TNF-alpha, and adipokines (Weisberg et al., 2003; Xu et al., 2003; Lumeng et al., 2007).

In standard clinical practice, advanced immunological assays are not consistently accessible. Metabolic factors, including body mass index (BMI), triglycerides (TG), HDL cholesterol (HDL-C), LDL cholesterol (LDL-C), glucose, and blood pressure, are commonly assessed. Inflammatory indicators, including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), are readily available in numerous clinical environments. The integration of these indicators may offer a practical method for detecting patients exhibiting an immunometabolic dysfunction phenotype (Chaplin, 2010; Furman et al., 2019; Sproston & Ashworth, 2018).

CRP is a commonly utilized indicator of systemic inflammation. Concentrations exceeding 3 mg/L are typically seen as indicative of elevated inflammatory or cardiometabolic risk within the relevant clinical setting (Ridker, 2003; Pepys & Hirschfield, 2003; Emerging Risk Factors Collaboration, 2010; Sproston & Ashworth, 2018). ESR is less specific however continues to be therapeutically valuable as a supporting indicator of inflammatory load. CRP and ESR collectively may elucidate the immunological ramifications of metabolic risk grouping.

Despite the growing interest in immunometabolism, limited research has explored the synergistic application of commonly available metabolic and inflammatory indicators for detecting immunometabolic dysfunction in regular clinical environments. Moreover, simplified risk frameworks appropriate for resource-constrained healthcare settings have been little investigated. This study sought to assess the relationships between metabolic risk variables and inflammatory markers in adult patients. We developed an Immunometabolic Risk Score (IMRS) utilizing standard clinical indicators and examined its correlation with CRP, ESR, and WBC. The study aimed to enhance the clinical immunology significance of standard cardiometabolic data via an immunometabolic framework.

Study Novelty

This study presents a pragmatic immunometabolic paradigm by amalgamating commonly accessible metabolic and inflammatory indicators into a composite risk assessment model. This study illustrates the potential effectiveness of readily available clinical measurements in identifying individuals at heightened risk of low-grade inflammatory activity and immunometabolic dysfunction, in contrast to many prior investigations that depended on advanced molecular or cytokine-based analyses.

Theoretical Framework

The research was informed by the principle of immunometabolism, which posits a strong interconnection between metabolic and immunological processes. In this context, obesity, dyslipidemia, hyperglycemia, and hypertension induce chronic metabolic stress, hence fostering low-grade inflammatory activity. CRP and ESR were assessed as clinically available markers of inflammatory load within an immunometabolic framework.

2. Methods

2.1 Study Design and Population

This is a retrospective cross-sectional clinical study utilizing patient-level data from Bucharest, Romania, with 110 adult participants. The available variables comprised sex, age, height, weight, body mass index (BMI), blood pressure, triglycerides, HDL cholesterol, LDL cholesterol, glucose, white blood cell count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). The column titled "CPR" in the uploaded dataset was construed as CRP in light of the revised clinical context supplied by the user.

2.2 Data Cleaning

One BMI figure was deemed clinically unreasonable and was adjusted by dividing weight by height squared. Blood pressure readings were categorized into systolic and diastolic components. Records were kept as no significant missingness was seen in the investigated variables.

2.3 Immunometabolic Risk Score

An Immunometabolic Risk Score (IMRS) was established by allocating one point for each of the subsequent criteria: BMI >=30 kg/m², TG >=150 mg/dL, low HDL-C (<40 mg/dL in males and <50 mg/dL in females), LDL-C >=130 mg/dL, glucose >=126 mg/dL, and blood pressure >=130/85 mmHg. Scores varied from 0 to 6 and were categorized as low (0-2), moderate (3-4), or high (5-6) immunometabolic risk. The thresholds were chosen to represent widely utilized clinical cardiometabolic criteria, encompassing cholesterol, glucose, and blood pressure cutoffs employed in metabolic syndrome frameworks.

2.4 Inflammatory Markers

CRP was evaluated as the principal inflammatory biomarker. A CRP measurement over 3 mg/L was utilized to delineate higher low-grade inflammatory risk. ESR and WBC were evaluated as auxiliary inflammatory markers.

2.5 Statistical Analysis

Continuous parameters were summarized as mean ± standard deviation and median with interquartile range. Categorical variables were summarized using frequency and percentage. Spearman correlation coefficients were computed to assess the relationships between metabolic variables and inflammatory markers. Kruskal-Wallis tests were employed to compare inflammatory markers among IMRS groups. Multivariable logistic regression was employed to assess predictors of CRP >3 mg/L, controlling for IMRS, age, and sex. Statistical significance was established at $p<0.05$. Due to the skewness of many metabolic variables, non-parametric correlation and group-comparison approaches were favored for inferential analysis. The low-risk IMRS subgroup was analyzed with caution due to its limited size.

3. Results

3.1 Baseline Characteristics

The study comprised 110 adults: 47 males (42.7%) and 63 females (57.3%). The mean age was 52.60 ± 8.54 years, and the mean BMI was 28.51 ± 4.67 kg/m². The mean triglycerides, LDL cholesterol, glucose, erythrocyte sedimentation rate, and C-reactive protein were 295.94 ± 143.78 mg/dL, 179.56 ± 44.75 mg/dL, 188.38 ± 45.69 mg/dL, 13.36 ± 2.04, and 2.35 ± 1.67 mg/L, respectively (Table 1).

Table 1. Baseline characteristics of the study population.

Variable	Mean ± SD	Median (IQR)
Sample size	110	
Sex	Male: 47 (42.7%); Female: 63 (57.3%)	
Age (years)	52.60 ± 8.54	54.50 (46.00-60.00)
BMI (kg/m²)	28.51 ± 4.67	25.25 (24.80-32.65)
Systolic BP (mmHg)	136.81 ± 6.98	137.00 (131.75-142.00)
Diastolic BP (mmHg)	87.44 ± 2.47	87.00 (86.00-89.00)
Triglycerides (mg/dL)	295.94 ± 143.78	287.50 (155.00-445.00)
HDL-C (mg/dL)	49.23 ± 13.64	51.50 (40.00-60.00)
LDL-C (mg/dL)	179.56 ± 44.75	150.00 (145.50-209.00)
Glucose (mg/dL)	188.38 ± 45.69	200.00 (140.00-219.00)
WBC	4.86 ± 0.49	4.97 (4.44-5.12)
ESR	13.36 ± 2.04	15.00 (12.00-15.00)
CRP	2.35 ± 1.67	2.00 (1.00-3.00)
IMRS	4.32 ± 0.99	5.00 (4.00-5.00)

3.2 Prevalence of Metabolic and Inflammatory Risk Components

The majority of subjects had several metabolic risk factors. high triglycerides were observed in 102 patients (92.7%), high low-density lipoprotein cholesterol in 109 (99.1%), elevated glucose levels in 104 (94.5%), and elevated blood pressure in 95 (86.4%). Obesity was observed in 42 individuals (38.2%). Ninety-two patients (83.6%) exhibited a high IMRS (≥4). Elevated CRP levels (>3 mg/L) were seen in 26 individuals (23.6%) (Table 2).

Table 2. Prevalence of metabolic and inflammatory risk components.

Risk component	n	%
BMI ≥30 kg/m²	42	38.2
TG ≥150 mg/dL	102	92.7
Low HDL-C (sex-specific)	23	20.9
LDL-C ≥130 mg/dL	109	99.1
Glucose ≥126 mg/dL	104	94.5
BP ≥130/85 mmHg	95	86.4
High immunometabolic risk score (IMRS ≥4)	92	83.6
CRP >3 mg/L	26	23.6

3.3 Correlations Between Metabolic Variables and Inflammatory Markers

CRP had strong positive associations with age (rho=0.410, $p<0.001$), triglycerides (TG) (rho=0.468, $p<0.001$), low-density lipoprotein cholesterol (LDL-C) (rho=0.439, $p<0.001$), glucose (rho=0.313, $p=0.001$),

and insulin metabolic resistance score (IMRS) (rho=0.313, p=0.001). CRP exhibited a negative correlation with HDL-C (rho=-0.344, p<0.001). The dataset revealed no significant correlation between BMI and CRP. The erythrocyte sedimentation rate (ESR) exhibited a positive correlation with age (rho=0.297, p=0.002) and the insulin-mediated resistance score (IMRS) (rho=0.213, p=0.026), although its correlations with specific lipid and glucose variables were less pronounced and not statistically significant (Table 3).

Table 3. Spearman correlations between metabolic variables and inflammatory markers.

Predictor	CRP rho	CRP p	ESR rho	ESR p
Age	0.410	<0.001	0.297	0.002
BMI	-0.020	0.839	0.120	0.212
TG	0.468	<0.001	0.142	0.138
HDL	-0.344	<0.001	-0.084	0.382
LDL	0.439	<0.001	0.128	0.183
Glucose	0.313	<0.001	-0.037	0.703
WBC	-0.006	0.947	-0.217	0.023
IMRS	0.313	<0.001	0.213	0.026

3.4 Inflammatory Markers Across Immunometabolic Risk Categories

CRP exhibited considerable variation within IMRS categories (Kruskal-Wallis p=0.002). The median CRP levels were 2.00 mg/L for the low-risk group, 1.00 mg/L for the moderate-risk group, and 2.35 mg/L for the high-risk group. The erythrocyte sedimentation rate (ESR) varied throughout the IMRS categories (p=0.007), although the white blood cell count (WBC) exhibited no significant variation (p=0.132) (Table 4).

Table 4. Inflammatory markers across immunometabolic risk categories.

Risk category	n	CRP mean ± SD	CRP median (IQR)	ESR mean ± SD	ESR median (IQR)	WBC mean ± SD
Low	5	2.32 ± 1.05	2.00 (1.90-3.00)	10.80 ± 0.84	11.00 (10.00-11.00)	5.34 ± 0.36
Moderate	49	1.80 ± 1.36	1.00 (1.00-2.05)	13.22 ± 2.03	15.00 (12.00-15.00)	4.82 ± 0.50
High	56	2.84 ± 1.82	2.35 (1.04-4.00)	13.71 ± 1.97	15.00 (12.00-15.00)	4.85 ± 0.48
Kruskal-Wallis p		0.002		0.007		0.132

Note. Risk categories were defined as low (IMRS 0-2), moderate (IMRS 3-4), and high (IMRS 5-6).

3.5 Multivariable Analysis

With the logistic regression method for CRP >3 mg/L, corrected for IMRS, age, and sex, age was independently correlated with elevated CRP (OR=1.11 per year, 95% CI 1.02-1.20, p=0.010). IMRS demonstrated a positive yet non-significant adjusted correlation with increased CRP (OR=1.43, 95% CI 0.71-2.89, p=0.315), indicating that the unadjusted relationship between IMRS and CRP may be partially affected by age in this group (Table 5).

Table 5. Multivariable logistic regression for CRP >3 mg/L.

Predictor	Adjusted OR for CRP >3 mg/L	95% CI	p-value
IMRS	1.43	0.71-2.89	0.315
Age	1.11	1.02-1.20	0.010
Male sex	0.99	0.38-2.61	0.990

Figures

Figure 1. CRP distribution across immunometabolic risk categories.

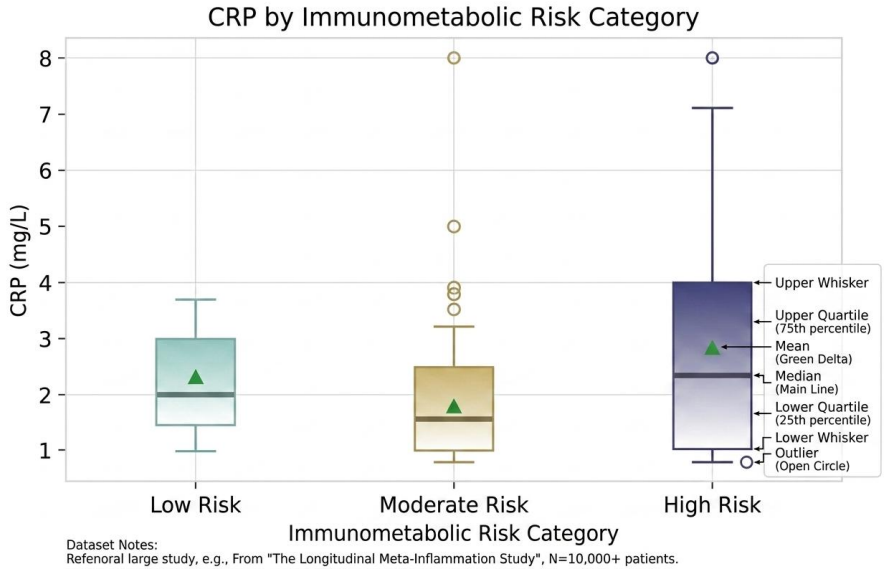

Figure 2. Scatter plot showing the relationship between triglycerides and CRP.

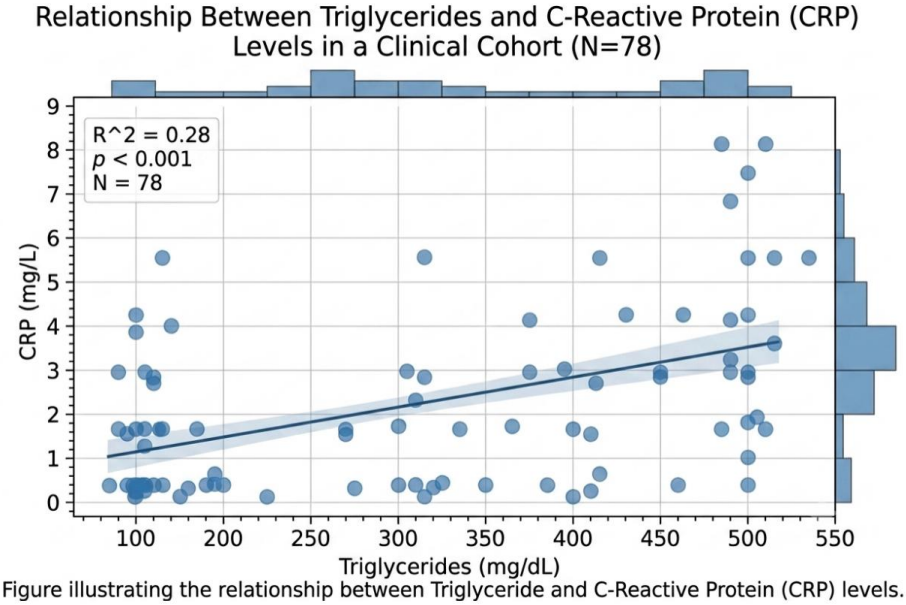

Figure illustrating the relationship between Triglyceride and C-Reactive Protein (CRP) levels.

4. Discussion

This study shows that the aggregation of metabolic risk variables correlates with low-grade inflammatory activity in adult individuals. The most reliable inflammatory marker was CRP, which had a positive correlation with TG, LDL-C, glucose, and the composite IMRS, while showing a negative correlation with HDL-C. These data corroborate the notion that habitual metabolic irregularities may indicate an immunometabolic dysfunction phenotype. The significant occurrence of heightened immunometabolic risk in this cohort likely indicates the clinically enriched characteristics of the sampled group rather than the frequency of immunometabolic dysfunction in the general populace.

The correlation between triglycerides and C-reactive protein is biologically feasible. Hypertriglyceridemia and elevated LDL-C lead to endothelial dysfunction, oxidative stress, and the activation of inflammatory pathways, whereas HDL-C is typically linked to anti-inflammatory and anti-atherogenic properties (Hansson, 2005; Libby, 2021; Xu et al., 2024). The noted inverse relationship between HDL-C and CRP is thus clinically significant and corresponds with current findings connecting atherogenic dyslipidemia to systemic inflammation.

Glucose had a positive correlation with CRP, reinforcing the connection between dysregulated glycemia and systemic inflammation. Hyperglycemia can induce oxidative stress, facilitate the production of advanced glycation end-products, and activate inflammatory signaling, all of which lead to cardiometabolic and immunological dysfunction

(Donath & Shoelson, 2011; Furman et al., 2019; Hu et al., 2024).

The correlation between IMRS and CRP substantiates the hypothesized risk concept. While IMRS exhibited an association with CRP in unadjusted correlation and group comparison analyses, it lost independent significance following adjustments for age and sex in logistic regression. This conclusion should not be construed as a lack of immunometabolic significance; instead, it suggests that age is a crucial confounder or moderator of inflammatory risk in this population. Chronic inflammation escalates with age and may influence the accumulation of metabolic risk (Furman et al., 2019).

ESR demonstrated less robust relationships compared to CRP. ESR had a substantial correlation with IMRS, although not with the majority of specific lipid and glucose parameters. This may indicate the reduced specificity of ESR relative to CRP for low-grade metabolic inflammation. The WBC did not demonstrate significant correlations with metabolic risk, indicating that total leukocyte count may be less responsive than CRP in this regard. The more robust correlation identified between CRP and metabolic disorders, in contrast to ESR, endorses CRP as a superior biomarker for immunometabolic risk stratification in standard clinical practice (Pepys & Hirschfield, 2003; Sproston & Ashworth, 2018).

The therapeutic importance of these discoveries resides in their applicability. In numerous healthcare environments, sophisticated cytokine assays, immunological phenotyping, and molecular inflammatory indicators are not commonly accessible. Nonetheless, lipid profile, glucose levels, blood pressure, body mass index (BMI), C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) are readily available. The integration of these factors may offer a viable method for initial immunometabolic risk categorization, particularly in resource-constrained settings.

5. Strengths and Limitations

A significant aspect of this study is the amalgamation of ordinary metabolic variables with direct inflammatory markers, specifically CRP and ESR, hence enhancing the clinical immunological significance in contrast to metabolic profile alone. The research employed objective laboratory assessments and created a composite IMRS that assesses cumulative risk burden instead of individual metabolic anomalies. This method improves clinical applicability and reproducibility in resource-constrained environments.

Numerous constraints must be recognized. The study's cross-sectional methodology may have hindered the ability to draw more solid causal conclusions. The dataset was limited in size and may not accurately reflect a larger population. The study sample had a significant prevalence of metabolic problems, indicating it may reflect a clinically defined population; thus, the results should be evaluated within the context of individuals with substantial cardiovascular and metabolic risk. The levels of C-reactive protein (CRP) were analyzed from a column designated "CRP. Direct measurements of cytokines, neutrophil and lymphocyte counts, and platelet counts were not accessible; hence, the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic inflammation index (SII) could not be computed.

6. Translational Implications for Clinical Immunology

The results underscore the increasing significance of clinical immunometabolism as a connection between metabolic therapy and clinical immunology. Standard metabolic indicators, when analyzed alongside CRP and ESR, may assist in identifying people at low-grade inflammatory risk. Subsequent research should incorporate cytokine profiling, immune cell phenotyping, and hematological inflammatory indicators to substantiate this methodology.

Future Research Agenda

Subsequent research should amalgamate metabolic factors with an extensive array of immunological biomarkers, encompassing cytokine profiling, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, systemic immune-inflammation index, adipokines, and immune cell phenotyping. Future longitudinal studies are required to ascertain if the observed immunometabolic phenotype forecasts subsequent inflammatory and cardiometabolic outcomes.

Clinical Take-Home Message

Standard metabolic indicators, specifically triglycerides, LDL-C, glucose, and blood pressure, when analyzed in conjunction with CRP and ESR, may offer a pragmatic and economical method for identifying individuals at heightened immunometabolic risk. Such solutions may be particularly beneficial in healthcare systems when extensive immunology testing is either unavailable or prohibitively expensive.

7. Conclusion

This study illustrates that the aggregation of metabolic anomalies correlates with low-grade inflammatory activity, namely heightened CRP levels. TG, LDL-C, glucose, HDL-C, and IMRS exhibited significant associations with CRP, reinforcing the notion of immunometabolic dysfunction. In this dataset, CRP proved to be a more informative inflammatory measure than ESR or WBC. These findings endorse the nascent notion of immunometabolism and imply that commonly accessible metabolic and inflammatory indicators could offer a pragmatic framework for assessing immunometabolic risk. Future prospective studies utilizing enhanced immune biomarkers and external validation cohorts are necessary to further confirm the clinical efficacy of this method.

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