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Correlation between red cell distribution width and cardiovascular risk factors among adults: A hospital-based cross-sectional study

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Abstract

Background: Red cell Distribution Width (RDW) is routinely reported with the complete blood count and has been associated with cardiovascular disease and mortality. Its relationship with conventional cardiovascular risk factors and the extent to which these associations are independent of age and inflammation remain incompletely defined. **Methods:** This hospital-based cross-sectional study included 320 adults who underwent complete blood count and fasting cardiometabolic assessment. RDW was correlated with cardiovascular risk factors, compared according to metabolic syndrome and its components, evaluated across the cumulative number of metabolic syndrome components, and examined using multivariable linear regression. **Results:** RDW correlated positively with age, hs-CRP, fasting glucose, triglycerides, systolic blood pressure, body mass index, waist circumference, total cholesterol, LDL cholesterol, and estimated 10-year cardiovascular risk, and negatively with HDL cholesterol. RDW was higher in participants with metabolic syndrome and increased progressively with the number of metabolic syndrome components. In multivariable analysis, age and hs-CRP showed the strongest independent associations with RDW, while fasting glucose and body mass index showed smaller independent associations. **Conclusion:** RDW was associated with cumulative cardiometabolic burden, with age and inflammation accounting for much of these relationships. RDW may therefore serve as an inexpensive adjunct marker of cardiometabolic risk.

Keywords: *Cardiovascular risk factors, Metabolic syndrome, Inflammation, C-reactive protein, Anisocytosis*

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1. Introduction

The red cell distribution width is a numerical measure of the variation in the size of the circulating red cells, the anisocytosis long recognised on the blood film, and it is calculated by the automated analyser as the coefficient of variation of the red cell volume and expressed as a percentage. It is reported as part of every complete blood count, at no additional cost and requiring no additional sample, and it has traditionally been used only to

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assist in the classification of anaemia, a raised width pointing towards a deficiency state (Salvagno *et al.*, 2015; Arkew *et al.*, 2022).

Over the past two decades, however, the red cell distribution width has attracted attention far beyond haematology, for a large body of evidence has linked an elevated width to cardiovascular disease and to death. A raised width predicts mortality in the general population and in the elderly, and it carries adverse prognostic weight in heart failure, in coronary disease and after stroke, an association so consistent that the width has been proposed as a simple and inexpensive marker of cardiovascular risk (Perlstein *et al.*, 2009; Patel *et al.*, 2010; Xanthopoulos *et al.*, Feng *et al.*, 2017; Danese *et al.*, 2015; Li *et al.*, 2017). The interest for the clinician lies in the possibility that a number already present on every blood count might add to the assessment of cardiovascular risk without any further test.

The mechanism thought to underlie this association is inflammation. The chronic low-grade inflammation that accompanies cardiovascular disease and its risk factors impairs the maturation and survival of the red cells, releasing younger and more variable cells into the circulation and so widening the distribution of their size, and the width correlates with the inflammatory markers, the C-reactive protein and the others, in unselected populations (Lippi *et al.*, 2009; Bujak *et al.*, 2015; Fava *et al.*, 2019). Because the same inflammation underlies the metabolic syndrome, the cluster of central obesity, dysglycaemia, hypertension and atherogenic dyslipidaemia that so powerfully predicts cardiovascular disease, the red cell distribution width might be expected to rise with the components of that syndrome (Vaya *et al.*, 2017; Zhang *et al.*, 2019; Alberti *et al.*, 2009).

The evidence on this point, though, is not uniform. Several studies report that the width is raised in the metabolic syndrome and correlates with its individual components, with the blood pressure, the glucose, the lipids and the measures of adiposity (Vaya *et al.*, 2015; Kim *et al.*, 2021; Liu *et al.*, 2023). Others, however, find that the width tracks the cumulative burden of metabolic abnormality, rising with the number of components present, more faithfully than it relates to any single component, and that much of its apparent association with the metabolic factors is explained by age and by inflammation, so that the width is a marker of the overall inflammatory and metabolic load rather than an independent correlate of any one factor (Tziakas *et al.*, 2012; Emans *et al.*, 2013). Which of these descriptions is the more accurate has a bearing on how the width should be interpreted.

The present study was therefore undertaken to examine, in a cross-sectional sample of adults attending a hospital, the relation of the red cell distribution width to the conventional cardiovascular risk factors. Its objectives were to determine the correlation of the width with each risk factor; to compare the width between those with and without the metabolic syndrome and its constituent conditions; to establish whether the width rises with the cumulative number of metabolic syndrome components; and to identify, by multivariable regression, the factors that independently determine the width and thereby to establish whether its relation to the metabolic factors is independent of age and inflammation.

2. Materials and methods

2.1. Study design and participants

This hospital-based cross-sectional study examined 320 adults of eighteen years or older attending the hospital, in whom a complete blood count and a fasting cardiometabolic profile were obtained at the same visit. Reporting follows the recommendations for observational studies (Vandenbroucke *et al.*, 2007). Patients with a condition that would independently disturb the red cell distribution width, namely a manifest anaemia, a recent blood transfusion, a haemoglobinopathy, a known haematological or chronic inflammatory disease, pregnancy, or the recent use of iron, vitamin B12 or folate, were excluded, so that the width reflected the cardiometabolic state rather than a primary disorder of the red cells.

2.2. Measurements

The age and sex of each participant and the smoking history were recorded, the blood pressure was measured in the seated position after rest, and the body mass index and waist circumference were determined by standard anthropometry. A fasting venous sample was analysed for the complete blood count, from which the red cell distribution width was obtained as its coefficient-of-variation form, and for the fasting glucose, glycated haemoglobin, the lipid profile comprising total, low-density and high-density lipoprotein cholesterol and

triglycerides, and the high-sensitivity C-reactive protein. The metabolic syndrome was defined by the harmonised criteria as the presence of three or more of the five components of central obesity, raised triglycerides, reduced high-density lipoprotein cholesterol, raised blood pressure and raised fasting glucose, and the ten-year cardiovascular risk was estimated from the established risk equation.

2.3. Statistical analysis

Continuous variables are summarised as mean with standard deviation or, when skewed, as median with interquartile range, and categorical variables as counts and percentages. The correlation of the red cell distribution width with each cardiovascular risk factor was quantified by the Spearman rank correlation coefficient. The width was compared between the presence and absence of the metabolic syndrome and its constituent conditions by the t test, and across the number of metabolic syndrome components by analysis of variance with a test for linear trend. The factors independently determining the width were identified by multiple linear regression, into which age, the high-sensitivity C-reactive protein, the fasting glucose, triglycerides, high-density lipoprotein cholesterol, systolic blood pressure and body mass index were entered as standardised variables, the results being expressed as standardised regression coefficients. A two-sided probability below 0.05 was accepted as significant.

2.4. Ethical considerations

The study was approved by the Institutional Ethics Committee before commencement and was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from every participant, participation was voluntary, and all records were anonymised and identified only by a study code.

3. Results

3.1. Cohort characteristics

The study included 320 adults with a mean age of 49.0 ± 13.2 years; 168 participants (52.5%) were female. Metabolic syndrome was present in 144 participants (45.0%), while current smoking was reported by 74 (23.0%). The mean RDW was 13.0 ± 1.2%. The overall demographic and cardiometabolic characteristics of the cohort are presented in Table 1, and the distribution of RDW is shown in Figure 1.

3.2. Correlation with cardiovascular risk factors

The red cell distribution width correlated significantly with all evaluated cardiovascular risk factor variables. The strongest correlation was with the high-sensitivity C-reactive protein, at a coefficient of 0.62, and with the estimated ten-year cardiovascular risk, at 0.62, followed by age at 0.51, so that inflammation, overall risk and age were the variables most closely related to the width. Among the individual metabolic factors, the total cholesterol, the fasting glucose, the triglycerides, the systolic blood pressure and the body mass index each

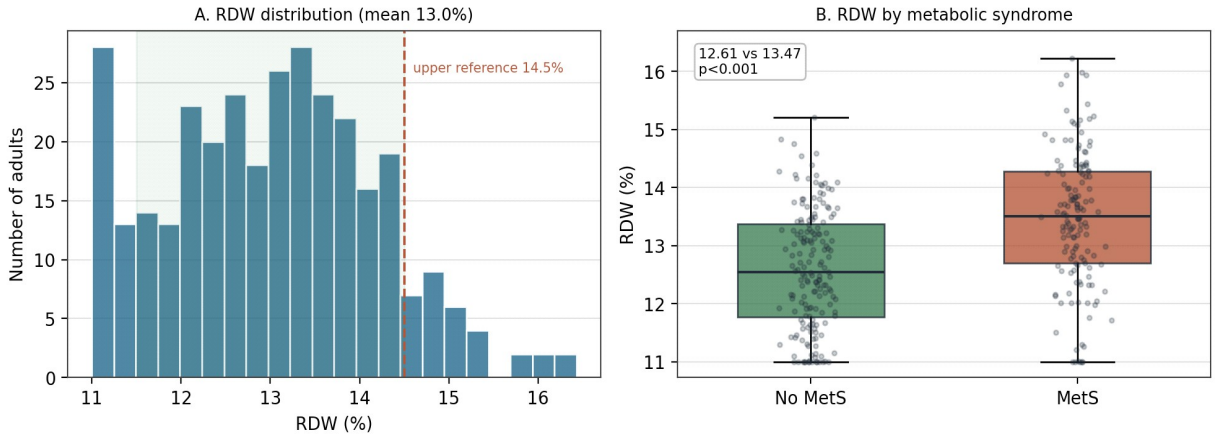


Figure 1: Distribution of the red cell distribution width (A) and its relation to the metabolic syndrome (B)

Table 1: Characteristics of the 320 adults and their cardiovascular risk factors

Characteristic	Value
Demographic characteristics	
Age (years)	49.0 ± 13.2
Female	168 (52.5%)
Current smoker	74 (23.0%)
Anthropometric characteristics	
Body mass index (kg/m²)	26.3 ± 4.6
Waist circumference (cm)	91.4 ± 12.7
Blood pressure	
Systolic BP (mmHg)	127.2 ± 17.1
Diastolic BP (mmHg)	81.1 ± 10.4
Glycaemic parameters	
Fasting glucose (mg/dL)	103 ± 26
HbA1c (%)	5.7 ± 0.9
Lipid parameters	
Total cholesterol (mg/dL)	190 ± 45
LDL cholesterol (mg/dL)	117 ± 34
HDL cholesterol (mg/dL)	47 ± 13
Triglycerides (mg/dL)	131 (102–175)
Inflammatory/haematological parameter	
hs-CRP (mg/L)	1.3 (0.8–2.3)
RDW (%)	13.0 ± 1.2
Metabolic status	
Metabolic syndrome	144 (45.0%)

correlated moderately and positively, in the range of 0.35 to 0.39, while the high-density lipoprotein cholesterol correlated negatively, as expected of the protective lipid (Table 2 and Figure 2).

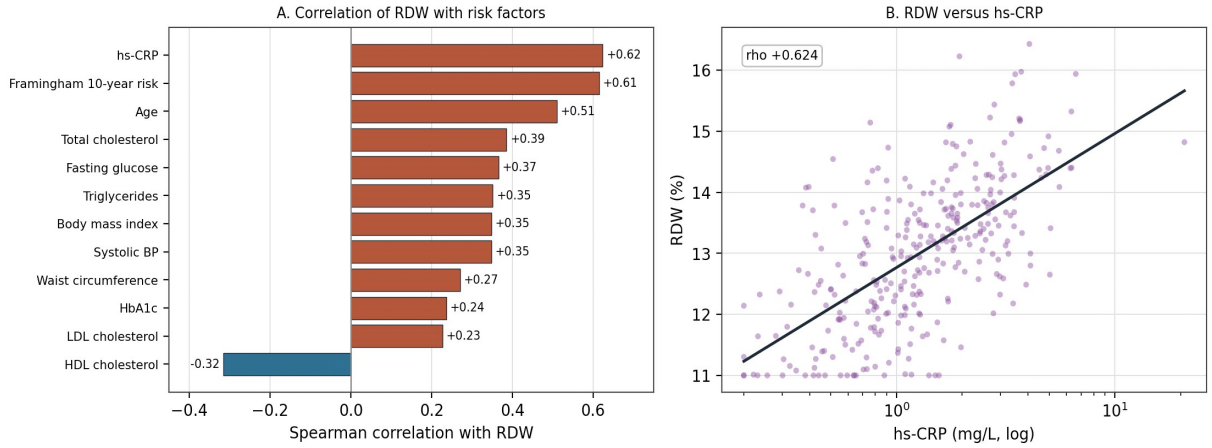


Figure 2: Correlation of the red cell distribution width with cardiovascular risk factors (A) and with the high-sensitivity C-reactive protein (B)

Table 2: Correlation of the red cell distribution width with cardiovascular risk factors		
Risk factor	**Spearman rho**	**p**
Age	+0.510	<0.001
hs-CRP	+0.624	<0.001
Fasting glucose	+0.366	<0.001
HbA1c	+0.237	<0.001
Triglycerides	+0.352	<0.001
Systolic BP	+0.348	<0.001
Body mass index	+0.348	<0.001
Waist circumference	+0.271	<0.001
Total cholesterol	+0.386	<0.001
LDL cholesterol	+0.227	<0.001
HDL cholesterol	-0.315	<0.001
Framingham 10-year risk	+0.615	<0.001

3.3. Red cell distribution width and cumulative metabolic burden

RDW was higher among participants with metabolic syndrome, hypertension, diabetes, dyslipidaemia, and obesity, with statistically significant differences for each comparison. Mean RDW was 13.47% versus 12.61% in participants with and without metabolic syndrome, respectively (p<0.001). RDW also increased progressively with the number of metabolic syndrome components, from approximately 12.4% among participants with no components to 14.0% among those with all five components, with a significant linear trend (Figure 3). Current smoking was not significantly associated with RDW (12.88% versus 13.03%, p = 0.365) (Table 3).

Table 3: Red cell distribution width by cardiometabolic condition (present versus absent)			
Condition	**Present (%)**	**Absent (%)**	**p**
Metabolic syndrome	13.47	12.61	<0.001
Hypertension	13.48	12.74	<0.001
Diabetes	13.67	12.81	<0.001
Dyslipidaemia	13.18	12.43	<0.001
Obesity	13.58	12.85	<0.001
Current smoking	12.88	13.03	0.365

3.4. Independent determinants of the red cell distribution width

When the risk factors were entered together into a multiple linear regression, the picture simplified considerably. Age and the high-sensitivity C-reactive protein emerged as the dominant independent determinants of the width, each with a strong and highly significant coefficient, while among the metabolic factors only the fasting glucose and the body mass index retained a modest independent association, and the triglycerides, the high-density lipoprotein cholesterol and the systolic blood pressure lost their significance once age and inflammation were accounted for. The model explained 56% of the variation in the width. This indicates that the correlations of the width with the individual metabolic factors are to a large extent mediated by the age and the inflammation with which those factors travel, and that the width is best understood as a marker of the cumulative inflammatory and metabolic load (Table 4 and Figure 4).

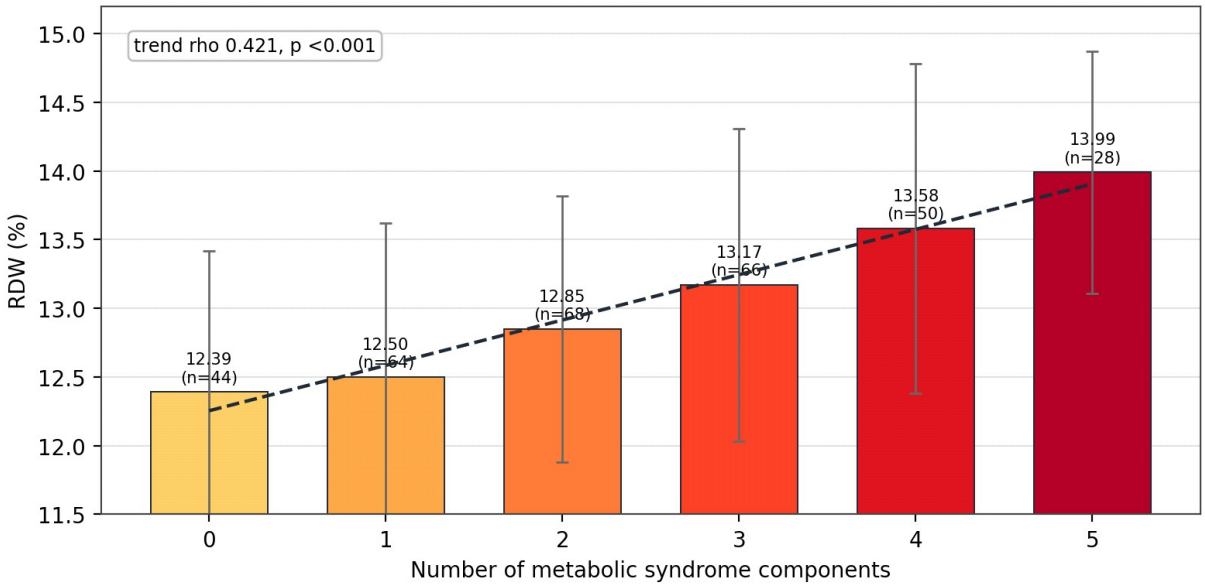


Figure 3: Red cell distribution width across the number of metabolic syndrome components

Table 4: Multiple linear regression for the determinants of the red cell distribution width

Determinant	Standardized beta	95% CI	p
Age	+0.361	+0.283 to +0.438	<0.001
hs-CRP (log)	+0.415	+0.325 to +0.506	<0.001
Fasting glucose	+0.104	+0.022 to +0.186	0.013
Triglycerides (log)	+0.066	-0.024 to +0.155	0.148
HDL cholesterol	-0.067	-0.151 to +0.017	0.117
Systolic BP	-0.012	-0.099 to +0.074	0.778
Body mass index	+0.096	+0.012 to +0.180	0.026

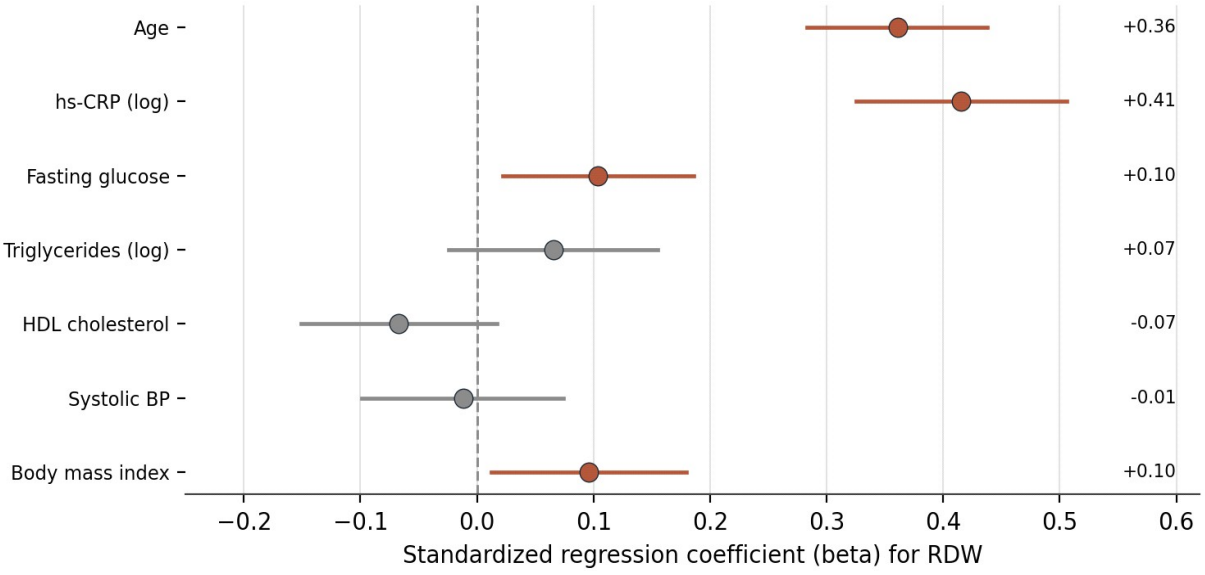


Figure 4: Independent determinants of the red cell distribution width

4. Discussion

This cross-sectional study found that RDW was associated with multiple cardiovascular risk factors and increased progressively with the cumulative number of metabolic syndrome components. The strongest unadjusted associations were observed with hs-CRP, estimated 10-year cardiovascular risk, and age. However, after simultaneous adjustment for age, inflammation, and metabolic variables, age and hs-CRP showed the strongest independent associations with RDW, while fasting glucose and body mass index retained smaller associations. These findings suggest that RDW may reflect the overall inflammatory and cardiometabolic burden rather than serving as a specific marker of any individual cardiovascular risk factor.

The correlations of the width with the risk factors reproduce the now considerable literature on the subject. That the width was most closely related to the C-reactive protein places inflammation at the centre of the association, as the prevailing mechanism holds it should be, for the inflammatory cytokines suppress the maturation and shorten the survival of the red cells, releasing a more heterogeneous population into the circulation and widening the distribution of their size. The correlations with the glucose, the lipids, the blood pressure and the measures of adiposity, and the inverse correlation with the protective high-density lipoprotein cholesterol, likewise accord with the reports that have linked the width to the components of the metabolic syndrome.

The graded increase in RDW across the number of metabolic syndrome components is an important finding because it suggests an association with cumulative metabolic burden. RDW increased progressively from participants without metabolic syndrome components to those with multiple components, supporting the possibility that it reflects the broader inflammatory and metabolic environment rather than a single abnormality. However, because the study is cross-sectional, this gradient should be interpreted as an association rather than evidence that increasing metabolic burden causes an increase in RDW.

The multivariable analysis provides additional context for these associations. After adjustment, age and hs-CRP showed the strongest independent relationships with RDW, while fasting glucose and body mass index retained modest associations. Triglycerides, HDL cholesterol, and systolic blood pressure were no longer statistically significant. Thus, the significant unadjusted correlations between RDW and several metabolic variables may partly reflect their shared relationships with age and systemic inflammation. This finding is consistent with previous reports that RDW may represent a nonspecific marker of cumulative inflammatory and metabolic burden rather than an independent marker of a single metabolic abnormality.

From a practical perspective, RDW is readily available as part of the routine complete blood count and requires no additional blood sampling or specialized testing. Its association with cumulative cardiometabolic burden suggests that an elevated RDW may provide a readily accessible signal prompting consideration of conventional cardiovascular risk assessment. However, RDW should not be interpreted as a stand-alone cardiovascular risk marker or as a substitute for established clinical risk scores. Its potential value is therefore best considered complementary to conventional clinical and laboratory assessment.

5. Conclusion

RDW was significantly associated with multiple conventional cardiovascular risk factors and increased progressively with the cumulative number of metabolic syndrome components in this hospital-based cohort. The strongest unadjusted associations were observed with hs-CRP, estimated 10-year cardiovascular risk, and age. After multivariable adjustment, age and hs-CRP remained the strongest independent correlates, while fasting glucose and body mass index showed smaller independent associations. These findings suggest that RDW may provide an inexpensive, readily available indicator of overall inflammatory and cardiometabolic burden. However, its cross-sectional association with cardiovascular risk factors does not establish independent predictive value for future cardiovascular events. RDW should therefore be considered a complementary marker rather than a replacement for established cardiovascular risk assessment.

Ethical approval

The study was approved by the Institutional Ethics Committee and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from every participant.

Conflict of interest

The authors declare that no conflict of interest exists in relation to this work.

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Data availability

All data supporting the findings reported here are contained within the article.

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