




ORIGINAL RESEARCH

Use of risk chart algorithms for the identification of psoriatic arthritis patients at high risk for cardiovascular disease: findings derived from the project CARMA cohort after a 7.5-year follow-up period

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ABSTRACT

Objective To assess the predictive value of four cardiovascular (CV) risk algorithms for identifying high-risk psoriatic arthritis (PsA) patients.

Methods Evaluation of patients with PsA enrolled in the Spanish prospective project CARDIOVASCULAR in Rheumatology. Baseline data of 669 PsA patients with no history of CV events at the baseline visit, who were followed in rheumatology outpatient clinics at tertiary centres for 7.5 years, were retrospectively analysed to test the performance of the Systematic Coronary Risk Assessment (SCORE), the modified version (mSCORE) European Alliance of Rheumatology Associations (EULAR) 2015/2016, the SCORE2 algorithm (the updated and improved version of SCORE) and the QRESEARCH risk estimator version 3 (QRISK3).

Results Over 4790 years of follow-up, there were 34 CV events, resulting in a linearised rate of 7.10 per 1000 person-years (95% CI 4.92 to 9.92). The four CV risk scales showed strong correlations and all showed significant associations with CV events ($p < 0.001$). SCORE, mSCORE EULAR 2015/2016 and QRISK3 effectively differentiated between low and high CV risk patients, although the cumulative rate of CV events observed over 7.5 years was lower than expected based on the frequency predicted by these risk scales. Additionally, model improvement was observed when combining QRISK3 with any other scale, particularly the combination of QRISK3 and SCORE2, which yielded the lowest Akaike information criterion (411.15) and Bayesian information criterion (420.10), making it the best predictive model.

Conclusions Risk chart algorithms are very useful for discriminating PsA at low and high CV risk. An integrated model featuring QRISK3 and SCORE2 yielded the optimal synergy of QRISK3's discrimination ability and SCORE2's calibration accuracy.

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ Cardiovascular (CV) mortality and events are more prevalent in patients with psoriatic arthritis (PsA) compared with the general population, resembling rates seen in rheumatoid arthritis patients.
- ⇒ Several studies have highlighted that Systematic Coronary Risk Assessment (SCORE) risk charts consistently underestimate CV risk in patients with PsA, emphasising the imperative need to develop new algorithms that align with modern treatments and effective management of the disease.

INTRODUCTION

Psoriatic arthritis (PsA) is a chronic inflammatory condition with a broad range of musculoskeletal manifestations, including peripheral arthritis, spondylitis, dactylitis and enthesitis, often linked with cutaneous psoriasis.¹ Patients with PsA may also present other extra-articular manifestations such as uveitis or inflammatory bowel disease.²

PsA correlates with a higher incidence and prevalence of cardiometabolic comorbidities compared with the general population.^{3,4} The elevated risk of cardiovascular (CV) disease in patients with PsA is due in part to a higher frequency of classic CV risk factors and metabolic syndrome.^{3,4} Chronic inflammation also plays an important role, leading to a higher frequency of endothelial dysfunction, early-stage atherogenesis and subclinical atherosclerotic disease, even in the absence of traditional CV risk factors.^{5,6} Additionally,

WHAT THIS STUDY ADDS

- ⇒ This study compares, for the first time, the strengths and limitations of four distinct scales (SCORE, modified SCORE European Alliance of Rheumatology Associations 2015/2016, SCORE2 and QRISK3) to evaluate CV risk in patients with PsA, followed prospectively in rheumatology units over 7.5 years.
- ⇒ Combining QRISK3 with any other risk chart algorithm improves the ability to identify PsA patients at increased CV risk. In particular, a model integrating QRISK3 and SCORE2 emerged as the most effective approach to discriminate between PsA patients with low and high CV risk.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ Understanding the real risk of CV events in patients with PsA treated in referral rheumatology units is crucial to formulating effective health strategies and reducing CV complications.
- ⇒ In assessing CV risk in patients with PsA, the combined application of CV risk scales enhances their effectiveness compared with using each scale individually.
- ⇒ Early assessment of CV risk enables timely detection of CV complications and the implementation of suitable measures to prevent the occurrence of future CV events in patients with PsA.

certain medications, such as glucocorticoids and non-steroidal anti-inflammatory drugs, and other treatments such as conventional synthetic disease-modifying anti-rheumatic drugs (DMARDs), biological DMARDs and targeted synthetic DMARDs, may have an impact on CV risk, whether positive or negative, in these patients.⁷

The high morbidity and mortality due to CV events among patients with PsA compared with the general population,^{8–10} together with that observed in other inflammatory arthritis such as rheumatoid arthritis (RA), led to the European Alliance of Rheumatology Associations (EULAR) an advocate for periodic CV disease risk assessments at least every 5 years for these individuals.^{7,11}

Taking into account the aforementioned factors, it is imperative to optimise prevention strategies, emphasising the crucial need to identify PsA patients at high risk of CV events. However, there is a notable absence of specific indices designed to establish the CV risk of individuals with rheumatic diseases. In this sense, the Systematic Coronary Risk Assessment (SCORE) has been recommended to evaluate CV risk in the European population.^{12,13} However, it is important to note that this algorithm, originally designed for the general population, estimates the 10-year risk of death from CV disease based on conventional CV risk factors (age, sex, systolic blood pressure, total cholesterol and smoking).¹³

Based on growing concern about the increased CV risk observed in patients with inflammatory arthritis, the EULAR Study Group on CV Diseases in Rheumatic Diseases introduced and endorsed the modified SCORE (mSCORE) EULAR 2015/2016 specifically for patients with RA and other inflammatory arthropathies. This adaptation of the SCORE involved a multiplication factor of 1.5 to compensate for the excess CV mortality

observed in these patients compared with the general population.^{7,11}

In 2021, a notable evolution occurred in the CV risk assessment landscape with the introduction of the SCORE2 predictive model, which marked an update to the existing SCORE CV risk algorithm.¹⁴ SCORE2 underwent meticulous calibration and validation processes, specifically designed to predict the 10-year risk of first-time CV disease events in European populations. SCORE2 differs from the traditional SCORE in several aspects. Specifically, it provides risk estimates that encompass fatal and non-fatal CV disease events. Moreover, SCORE2 incorporates consideration of the competing risks posed by non-CV deaths, a feature absent in the original SCORE framework.

SCORE2's recalibration strategy further differentiates it as it now serves four distinct European regions, unlike the two levels of regional stratification offered by SCORE.¹⁴ Additionally, the SCORE2 recalibration is systematically aligned with contemporary CV disease rates, which differentiated from the original SCORE model that was based on data collected before 1986. This transformative update improves the accuracy and relevance of CV risk assessment, aligning it more closely with the current dynamics of CV disease in the European population.

An alternative tool in this scenario is the QRESEARCH risk estimator version 3 (QRISK3), designed to measure the probability of suffering fatal and non-fatal CV events over a 10-year period.¹⁵ QRISK3, which emerged from collaboration between clinicians and academics associated with the UK National Health Service, has gained recognition as a reliable 10-year predictor of CV disease in British and international cohorts.¹⁵ By distinguishing itself, QRISK3 goes beyond traditional CV risk factors, encompassing diabetes mellitus and chronic kidney disease in its considerations. Additionally, it provides the option to take into account the presence of RA or systemic lupus erythematosus in the patient profile.¹⁵

Concerns have been raised regarding the possible limitation of indices developed for the general population in accurately predicting CV risk in people with rheumatic diseases.¹⁰ This discrepancy was highlighted in studies in patients with RA¹⁶ and ankylosing spondylitis (AS).¹⁷ In this context, a study focused on patients with PsA, which used carotid ultrasound to identify subclinical atherosclerosis, highlighted that the SCORE and similar indices may underestimate the actual CV risk of these patients.¹⁸ Similarly, a recent study highlighted that the SCORE algorithm tends to underestimate CV risk, and the mSCORE EULAR 2015/2016 does not provide any improvement in the prediction of CV disease in patients with PsA.¹⁹

In line with the above, a study in patients with RA disclosed that employing both the mSCORE EULAR 2015/2016 index and the QRISK3 index in combination enabled the identification of individuals exhibiting an increased likelihood of developing atheromatous

plaques in the carotid artery, indicative of elevated CV risk.²⁰ Similarly, in keeping with this perspective, another study within the RA patient population indicated that QRISK3 displayed a heightened sensitivity in recognising individuals with a high or very high CV risk compared with the mSCORE EULAR 2015/2016.²¹

Taking all these considerations into account, we aimed to determine the predictive value of SCORE, mSCORE, EULAR 2015/2016⁷ SCORE2 and QRISK3 risk chart algorithms to identify PsA patients at high risk of CV disease. Furthermore, we sought to explore whether combining two risk chart algorithms could improve the identification of PsA patients with high-risk CV disease. To achieve this objective, we evaluated patients enrolled in the Spanish prospective CARdiovascular in RheuMA-tology (CARMA) project. This project included a cohort of patients with PsA prospectively, followed in rheumatology outpatient clinics over a period of 7.5 years.

PATIENTS AND METHODS

Population

The CARMA project is a prospective cohort study with the primary goal of delineating the CV disease risk profile in individuals with chronic inflammatory rheumatic diseases over a 10-year observation period. The study enrolled patients diagnosed with AS, PsA and RA, along with a comparative cohort of individuals without inflammatory diseases. The recruitment process occurred across 67 Spanish hospitals from July 2010 to January 2012.²² This report focuses specifically on data from patients with PsA at 7.5 years from the start of the study.

The initial (baseline) recruitment included 721 patients with PsA.²² As previously reported,²² all met Moll and Wright's criteria for PsA.²³ The reason for using these criteria was that at the time of the first discussion of the project in 2007, the 2006 Classification Criteria for Psoriatic Arthritis (CASPAR)²⁴ were not widespread or used in all centres included in the project.

Data from a set of 669 Spanish patients diagnosed with PsA in the CARMA project without a history of CV events at the time of recruitment were evaluated.²² In this sense, 52 patients with a history of CV events (ischaemic heart disease, stroke, peripheral arterial disease or heart failure) before the start of the study (before the baseline visit) were excluded. Most patients (n=552; 82.5%) continued to periodically attend outpatient clinics at each centre included in the project. At 7.5 years, information on all patients included in the initial cohort was assessed. It was obtained by consulting their medical records or by calling patients or family members directly. When it was not available, we requested information from the Spanish National Statistics Institute, which is the institution in charge of keeping records of vital events, such as births and deaths in Spain. This allowed us to determine information on 669 patients with PsA who at the time of the initial visit had no history of CV events.

Information on this cohort is included in online supplemental table 1.

The retrospective examination involved testing of four risk algorithms, namely SCORE, mSCORE EULAR 2015/2016, SCORE2 and QRISK3, using baseline data acquired at the start of the patient's participation in the CARMA project.

Variable specifications and operative definitions

CV events and CV mortality

The analysis focused on the incidence of CV events and CV mortality. The spectrum of CV events encompassed ischaemic heart disease, stroke, peripheral arterial disease and heart failure. The operational definitions outlining the parameters of the variables under analysis were detailed in a separate report.²²

Calculation of CV risk algorithms

The determination of SCORE and SCORE2 followed the previously described methodologies.^{12 14} The SCORE calculation involved factors such as age, sex, smoking, systolic blood pressure and total cholesterol. On the other hand, SCORE2 was obtained using age, sex, smoking, systolic blood pressure and non-HDL cholesterol.

The SCORE2 calculation was executed using the 'score2risk' command for Stata, accessible at <http://www.phpc.cam.ac.uk/ceu/erfc/programs/>.^{14 25} The SCORE calculation followed the methodology described by Conroy *et al.*¹² SCORE evaluates the 10-year risk of death from CV disease. However, recognising that combining morbidity with mortality provides a more comprehensive perspective on the total burden of atherosclerotic CV disease (ASCVD), SCORE2 now estimates an individual's 10-year risk of fatal and non-fatal CV disease events for people from 40 to 69 years old. For people aged ≥ 70 years, the SCORE2-OP (older people) algorithm estimates the 5-year and 10-year risk of fatal and non-fatal CV disease events.

The determination of mSCORE EULAR 2015/2016 adhered to the updated recommendations from the EULAR group in 2015/2016.^{6 7}

As for QRISK3, its calculation was aligned with previously described procedures.¹⁵ This encompassed the analysis of several variables, including conventional CV risk factors, diabetes mellitus, chronic kidney disease, emerging CV risk factors and other inflammatory rheumatic diseases. QRISK3 measures an individual's risk of having a heart attack or stroke over the next 10 years. It reflects the average risk of individuals who share the same risk factors as those entered for that specific person.¹⁵ The algorithm establishes a threshold of 10%, classifying CV risk as high-very high at or above this level ($\geq 10\%$), and below that level, classifying it as low-moderate CV risk.

Statistical analysis

Patient demographic and clinical variables were presented as mean and SD, or median and IQR for

skewed quantitative variables, and as absolute numbers and relative frequencies (%) for qualitative variables. The correlation between the CV risk scales was quantified using the Pearson correlation coefficient.

Survival analysis was approached in two ways:

- In the first step, we defined failure as the combination of a CV event or death from any cause. Patients without a CV event and still alive at the end of the follow-up period were considered censored. Follow-up time was the duration until the occurrence of the first CV event or death for failures and until the last visit for censored patients. The discriminative effectiveness of any CV scale in identifying failures in this analysis was determined using Cox regression.
- Subsequently, we designated any CV event as a failure and death resulting from any non-CV cause was treated as a competing risk. Follow-up time mirrored that of analysis (a). The correlation between the CV scale and CV events was explored using the method proposed by Fine and Gray. No adjustments were made to any model for age or sex, given their inclusion in the CV scales.

Results from the survival analysis were portrayed as HR, accompanied by their 95% CI and two-tailed p values. Each of the survival analyses (a)–(b) involved the creation of seven models. Models 1–4 incorporated an individual CV scale each. Since SCORE, mSCORE EULAR 2015/2016 and SCORE2 are variants of each other, we abstained from examining their combination. Subsequently, to assess if augmenting QRISK3 with an additional CV risk scale would enhance the model, models 5–7 were constructed. Therefore, models 5–7 were confined to combinations of QRISK3 with any scale from the ‘SCORE family’. Due to the strong correlation between QRISK3 and any other CV scale, the ‘residual method’ was employed. Although we exemplify it here for the QRISK3–SCORE combination, the method is analogous for QRISK3–mSCORE EULAR 2015/2016 and QRISK3–SCORE2 combinations.

The residual method aimed to dissect SCORE into two components: one linearly dependent on QRISK3 (‘linear prediction’) and another independent of QRISK3 (‘residuals’). To achieve this, we executed a linear regression model with SCORE as the Y variable and QRISK3 as the regressor. The residuals derived from this regression represented the portion of SCORE independent of

QRISK3. Subsequently, the survival analysis incorporated both QRISK3 and the residual of SCORE as regressors.

For a comprehensive comparison of all seven models generated in each survival analysis, we computed the Akaike information criterion (AIC) and the Bayesian information criterion (BIC). Both criteria share a common principle: they penalise the introduction of new variables, thereby favouring parsimonious models unless the inclusion of a new variable significantly enhances the model likelihood. In essence, models with lower AIC or BIC are deemed more favourable.

All statistical analyses were conducted by using Stata V.18/SE software (StataCorp). Statistical significance was considered with p values < 0.05.

RESULTS

Correlation among CV risk scales

The correlation analysis revealed a robust association among all four CV risk scales (table 1). It is noteworthy that SCORE and mSCORE EULAR 2015/2016, differing solely by a multiplying factor of 1.5, essentially function as a simple change of units. Consequently, their correlation with each other attains a perfect value of 1.

Evaluation of CV risk scales: compound events (CV event or death by any cause)

A total of 41 compound events, encompassing CV events or deaths by any cause, were observed during the 4790 years of follow-up. The linearised rate was calculated at 8.56 per 1000 person-years, with a 95% CI of 6.14 to 11.61.

The prediction of CV events or deaths by any cause is detailed in table 2 and illustrated in figure 1A–D. In comparing various scales, two essential characteristics must be considered: discrimination and calibration.

Discrimination, which denotes the ability to distinguish between individuals at high and low CV risk, can be measured through the risk indices provided in table 2. A higher risk index means superior discrimination. Additionally, discrimination can be assessed visually by examining the spacing between lines in figure 1A–D. A wider separation indicates greater discrimination.

Calibration involves verifying the accuracy of the predicted risk. For example, if a scale predicts a 10% CV event risk over 10 years for a group of patients, it would be considered well calibrated if approximately 10% of

Table 1 Correlation between CV risk scales in patients with psoriatic arthritis from the CARMA cohort

	QRISK3	SCORE	mSCORE EULAR 2015/2016	SCORE2
QRISK3	1			
SCORE	0.7531	1		
mSCORE EULAR 2015/2016	0.7531	1	1	
SCORE2	0.8362	0.8170	0.8170	1

CARMA, CARdiovascular in rheuMATology cohort; CV, cardiovascular; EULAR, European Alliance of Rheumatology Associations; mSCORE, modified SCORE; QRISK3, QRESEARCH risk estimator version 3; SCORE, Systematic Coronary Risk Assessment.

Table 2 CV risk scales performance with the compound event death by any cause or CV event in the psoriatic arthritis CARMA cohort after 7.5 years of follow-up

Model	CV risk scale	HR (95% CI)	P value	AIC	BIC	Figure
1	QRISK3	1.07 (1.05 to 1.08)	<0.001	489.84	494.34	1A
2	SCORE	1.06 (1.04 to 1.09)	<0.001	485.00	489.50	1B
3	mSCORE EULAR 2015/2016	1.04 (1.03 to 1.06)	<0.001	485.00	489.50	1C
4	SCORE2	1.17 (1.12 to 1.23)	<0.001	463.71	468.19	1D
5	QRISK3	1.07 (1.05 to 1.09)	<0.001	467.13	476.12	
	SCORE residual	1.01 (0.97 to 1.05)	0.58			
6	QRISK3	1.07 (1.05 to 1.09)	<0.001	467.13	476.12	
	m-SCORE residual	1.01 (0.98 to 1.03)	0.58			
7	QRISK3	1.08 (1.06 to 1.10)	<0.001	459.73	468.68	
	SCORE2 residual	1.10 (1.03 to 1.18)	0.006			

AIC, Akaike information criteria; BIC, Bayesian information criteria; CARMA, CARdiovascular in rheuMATology cohort; CV, cardiovascular; EULAR, European Alliance of Rheumatology Associations; mSCORE, modified SCORE; QRISK3, QRESEARCH risk estimator version 3; SCORE, Systematic Coronary Risk Assessment.

the patients in that group experienced a CV event over the 10 years period of follow-up. Conversely, it would be miscalibrated if the rate of CV events observed at follow-up differed significantly, such as occurring in 5% or 15% of patients.

Calibration evaluation can be performed by examining figure 1A–D and comparing each line to the

background grid. For example, if the red line (representing individuals with a predicted risk of 10%) aligns with the 10% line on the grid at the end of the follow-up period, the scale is considered well calibrated. Conversely, if the red line deviates significantly from the 10% line on the grid, the scale is considered poorly calibrated.

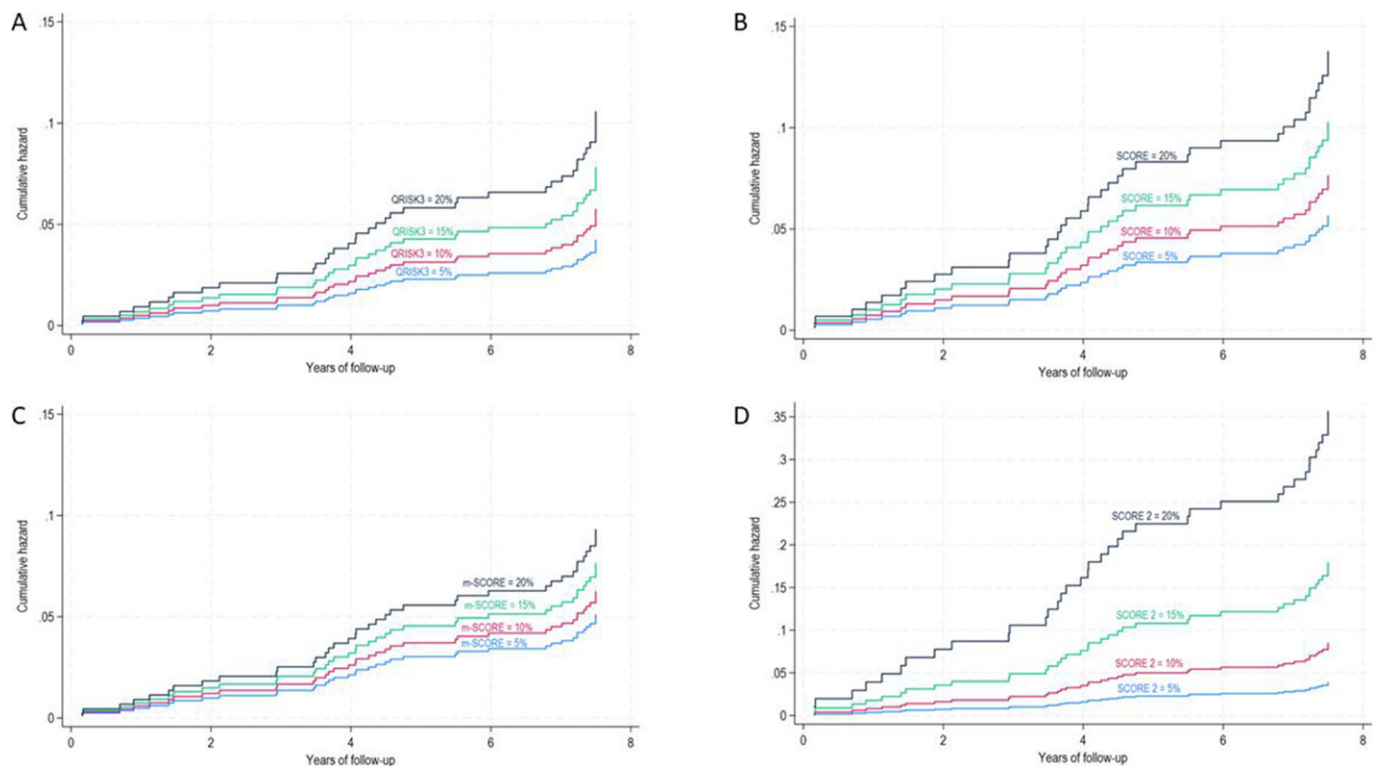


Figure 1 Cardiovascular event or death from any cause. Prediction using: (A) QRISK3, (B) SCORE, (C) mSCORE EULAR 2015/2016, (D) SCORE2. Notice that numerical data in the vertical axis ranges from 0 to 0.15 for subgraphics A–C, and from 0 to 0.35 for subgraphic D. EULAR, European Alliance of Rheumatology Associations; mSCORE, modified SCORE; QRISK3, QRESEARCH risk estimator version 3; SCORE, Systematic Coronary Risk Assessment.

All four scales demonstrated a significant association with events ($p < 0.001$) as presented in [table 2](#). The HRs were 1.07 (95% CI 1.05 to 1.08) for QRISK3, 1.06 (95% CI 1.04 to 1.09) for SCORE, 1.04 (95% CI 1.03 to 1.06) for mSCORE EULAR 2015/2016 and 1.17 (95% CI 1.12 to 1.23) for SCORE2.

QRISK3 effectively differentiated between individuals with lower and higher risk, as illustrated in [figure 1A](#). However, the percentage of events accumulated over 7.5 years was notably lower than expected based on the risk established by QRISK3. For example, individuals with a QRISK3 of 20% in 10 years experienced only about 9% of events after 7.5 years, as depicted by the black line in [figure 1A](#).

SCORE also effectively differentiated between individuals with lower and higher risk, as illustrated in [figure 1B](#). However, after 7.5 years of follow-up, the cumulative risk for individuals with SCORE=5% exceeded the expected percentage of events. Additionally, for those with SCORE=10%, 15% or 20%, the percentage of events by year 7.5 of follow-up was lower than predicted by this index.

mSCORE EULAR 2015/2016 demonstrated comparable discrimination ability to SCORE, although the predicted percentage of events was notably higher than the actual percentage. For example, individuals with mSCORE EULAR 2015/2016=20% experienced approximately 8% of events ([figure 1C](#), black line).

SCORE2 demonstrated robust discrimination ability in terms of CV risk, as evident from the well-separated lines in [figure 1D](#). For individuals with SCORE2=5%, 10% or 15%, the percentage of events by year 7.5 of follow-up aligned closely with predictions (blue, red and green lines in [figure 1D](#), respectively). However, SCORE2 underestimated the risk for individuals with SCORE2=20% black line in [figure 1D](#), where the cumulative hazard reached 33% in 7.5 years).

Combining QRISK3 with any other scale enhanced the model, as indicated in [table 2](#) (AIC and BIC for any model with two scales were lower than for any model with just one scale). The combination of QRISK3 and SCORE2 achieved the lowest AIC and BIC, making it the most effective predictive model.

Performance assessment of CV risk scales considering CV events (with death by non-CV causes as a competitive event)

[Table 3](#) and [figure 2A–D](#) illustrate the prediction of CV events when death by non-CV causes was treated as a competitive event. Over 4790 years of follow-up, there were 34 CV events, resulting in a linearised rate of 7.10 per 1000 person-years (95% CI 4.92 to 9.92). All four scales demonstrated discriminative power, as depicted in [figure 2A–D](#), with p values lower than 0.001 in [table 3](#). Notably, SCORE2 exhibited significantly lower AIC and BIC values. Consequently, among the four scales, SCORE2 stands out as the preferred choice. The discriminative ability of QRISK3 improved when combined with any other scale, evident in lower AIC and BIC values for combined models compared with the QRISK3-alone model. A model that integrated QRISK3 and SCORE2 demonstrated the lowest AIC (411.15) and BIC (420.10) values, making this combination the preferable choice.

DISCUSSION

In this study, we provide information derived from a large cohort of patients with PsA participating in the Spanish prospective CARMA project, focusing on the CV outcomes of people with inflammatory arthritis. This cohort analysis incorporates data from 669 patients with PsA collected over a period of 7.5 years after enrollment. Our research involved the evaluation of four CV risk algorithms. In addition to several versions of the European SCORE, the algorithm traditionally used to determine

Table 3 CV risk scales performance with the CV event in the psoriatic arthritis CARMA cohort after 7.5 years of follow-up

Model	Variables	HR (95% CI)	P value	AIC	BIC	Figure
1	QRISK3	1.05 (1.03 to 1.07)	<0.001	421.82	426.31	2A
2	SCORE	1.06 (1.04 to 1.09)	<0.001	424.65	429.15	2B
3	mSCORE EULAR 2015/2016	1.04 (1.03 to 1.06)	<0.001	424.65	429.15	2C
4	SCORE2	1.16 (1.11 to 1.22)	<0.001	409.43	413.91	2D
5	QRISK3	1.06 (1.04 to 1.08)	<0.001	421.03	430.01	
	Score residual	1.03 (1.00 to 1.07)	0.05			
6	QRISK3	1.06 (1.04 to 1.08)	<0.001	421.03	430.01	
	m-SCORE residual	1.02 (1.00 to 1.04)	0.05			
7	QRISK3	1.07 (1.04 to 1.09)	<0.001	411.15	420.10	
	SCORE2 residual	1.15 (1.07 to 1.23)	<0.001			

Death by non-CV cause was considered as competing event.

AIC, Akaike information criteria; BIC, Bayesian information criteria; CARMA, CARdiovascular in rheuMATology cohort; CV, cardiovascular; EULAR, European Alliance of Rheumatology Associations; mSCORE, modified SCORE; SCORE2, Systematic Coronary Risk Assessment.

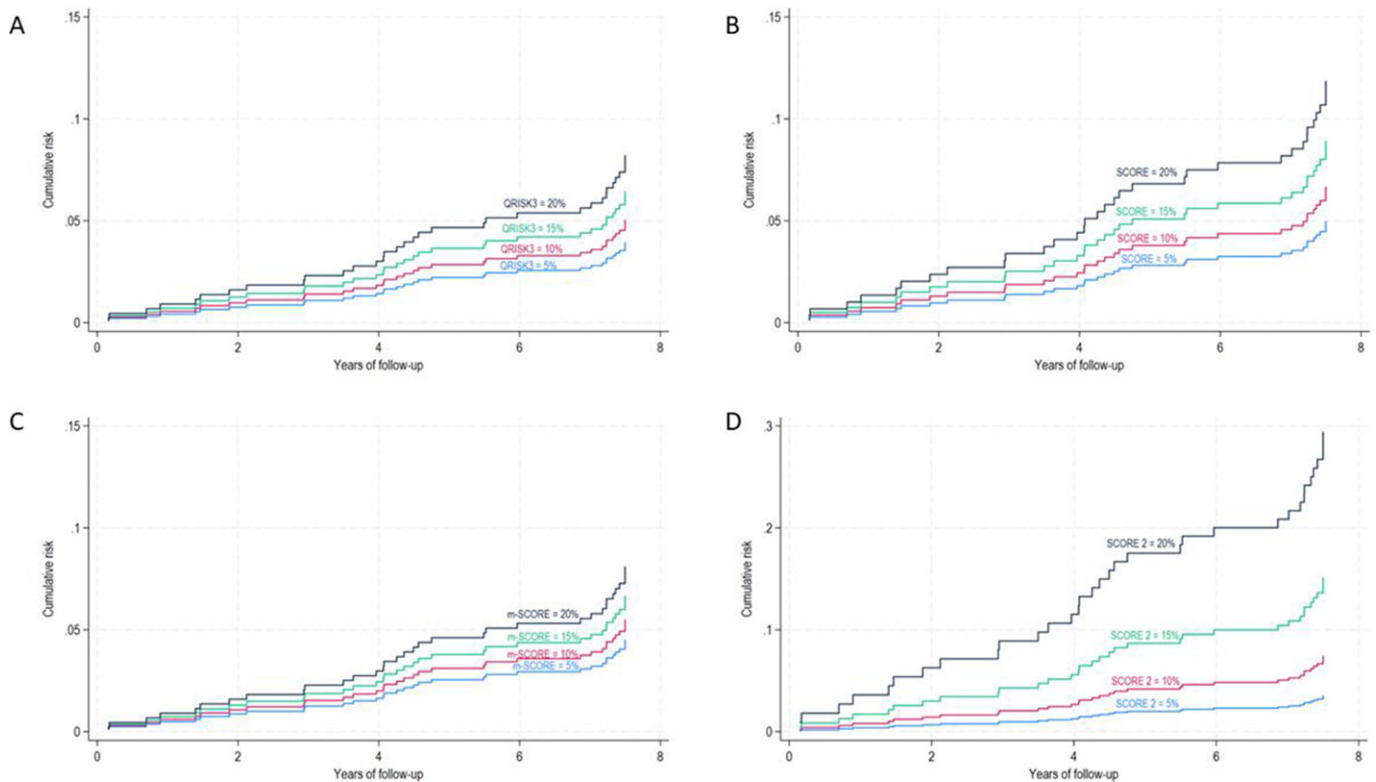


Figure 2 Cardiovascular (CV) event (death from non-CV causes as a competitive event). Prediction using: (A) QRISK3, (B) SCORE, (C) mSCORE EULAR 2015/2016, (D) SCORE2. Notice that numerical data in the vertical axis ranges from 0 to 0.15 for subgraphics A–C, and from 0 to 0.30 for subgraphic D. EULAR, European Alliance of Rheumatology Associations; mSCORE, modified SCORE; QRISK3, QRESEARCH risk estimator version 3; SCORE, Systematic Coronary Risk Assessment.

CV risk in the Spanish population, we also analysed in our cohort the QRISK3, used in the UK to evaluate CV risk.

The study findings suggest that SCORE, mSCORE EULAR 2015/2016, SCORE2 and QRISK3 are useful to assess CV risk PsA patients. In each instance, these risk algorithms aptly differentiated between PsA patients with high and low CV risk. The performance of SCORE2 was better than the other equations. It was evident when SCORE2 was compared with SCORE. This observation suggests that non-HDL cholesterol might play a crucial role in influencing CV events in PsA, given the prevalent metabolic risk in these patients. However, we feel that this insight may be extrapolated to the general population.

Despite strong correlations among the four scales, each failed to encompass all the necessary information about CV risk. Therefore, combining two algorithms could offer a more comprehensive understanding of the CV risk profile in PsA patients. In this regard, a model that integrated QRISK3 and SCORE2 yielded the best approach to identify patients with PsA at high risk for CV events.

There is limited information on the usefulness of CV risk algorithms in patients with PsA. In this context, Shen *et al* conducted an assessment of four CV risk scales: Framingham Risk Score (FRS), QRISK2, the 10-year ASCVD risk algorithm proposed by the American College of Cardiology and the American Heart Association, and

the mSCORE (SCORE multiplied by a factor of 1.5) as proposed by the EULAR group.²⁶ The study included 146 Chinese PsA patients, with 42.5% displaying carotid atheromatous plaque. These patients with carotid plaques were notably older, with higher systolic blood pressure values and higher LDL-cholesterol levels. FRS, QRISK2, ASCVD and mSCORE algorithms had moderate discrimination in recognising PsA patients with and without atheromatous plaque in the carotid artery. Notably, the predetermined high-risk cut-off values (FRS>10%, QRISK2>20%, SCORE>5% and ASCVD>7.5%) for all indices underestimated the risk by failing to identify atheromatous plaques.²⁶ These findings differ from our results, likely due to differences in the number of patients included in the study and possibly to differences in the genetic background within both studies. More importantly, Shen *et al* employed carotid atheromatous plaque as a surrogate marker for CV risk, whereas our study focused on evaluating the scales' performance in identifying CV events.

Navarini *et al*¹⁹ conducted another study in patients diagnosed with PsA according to the CASPAR criteria.²⁴ These authors performed a retrospective analysis using prospectively collected data from two Italian rheumatology centres. The data set, comprising 155 PsA patients with no history of CV events at baseline in November 2007, was reviewed and analysis was performed in November 2017. Over the follow-up period, 21 patients with PsA experienced a CV event. The study evaluated

the performance of five risk algorithms: FRS, SCORE, QRISK2, Reynolds risk scores (RRS) and the Italian individual index Progetto CUORE. The findings revealed that all five algorithms demonstrated relatively good discrimination between PsA patients with and without CV events, with areas under the ROC curves ranging from 0.7183 for RRS to 0.8660 for QRISK2. However, the calibration of these algorithms ranged from poor to moderate. Notably, CUORE, SCORE and RRS models exhibited a poor fit, as the distribution of observed events significantly differed from the predicted values. In contrast, FRS and QRISK2 showed a more aligned distribution. Moreover, the adaptation according to EULAR recommendations performing multiplication by the factor of 1.5 of CV risk algorithms in patients with inflammatory arthritis did not enhance the algorithms' discriminative ability or calibration.^{19 27}

There some possible explanations for the apparent contradiction between the results by Navarini *et al* and the results of the CARMA cohort after 7.5 years of follow-up. In this respect, Navarini *et al* confirmed that the persistence of elevated C reactive protein (CRP) and high disease activity may be considered predictive factors of CV disease in patients with axial spondyloarthritis.²⁸ These authors also assessed 295 patients with axial spondyloarthritis without personal history of CV disease and observed that persistency of increased CRP levels at each visit and high values of clinical parameters of disease activity help to identify patients with axial spondyloarthritis at higher risk of CV disease.²⁸ We entirely agree with this fact as in our experience disease activity influenced the reclassification of patients with RA into the category of very high CV risk.²⁹

Baseline data of the PsA included in the CARMA cohort showed that 42.5% of them were receiving biological DMARDs and 74% conventional DMARDs at the time of recruitment. The baseline median CRP of the PsA from the CARMA cohort was 3 mg/L. Moreover, the median Health Assessment Questionnaire, considered as the functionality score, was <0.5 (median 0.4 with an IQR between 0.0 and 0.9). These results indicate that patients with PsA included in the CARMA registry and followed prospectively at rheumatology units underwent tight control of the disease and consequently had low disease activity. Therefore, most of them achieved some of the objectives proposed by Coates *et al* of minimal disease activity of the disease.³⁰

In previous studies on carotid ultrasound in patients with PsA without CV risk factors or clinically evident CV disease, we disclosed a high prevalence of macrovascular disease compared with ethnically matched controls.⁶ Yet, when examining patients with RA, Karpouzias *et al* disclosed that the utilisation of biological therapy correlated with a decrease in CV disease among these patients.³¹ Thus, this intervention played a preventive role in CV events by inhibiting coronary plaque formation and stabilising high-risk coronary lesions.³¹ Additionally, Karpouzias *et al* showed that the administration

of statins altered the influence of inflammation on the development of new coronary plaque. This intervention also predicted both regression and calcification of non-calcified lesions in patients with RA, leading to a long-term reduction in CV risk for these individuals.³² In this context, as highlighted in our previous discussion on mortality after a 5-year follow-up,³³ individuals within the CARMA cohort were closely followed at tertiary referral centres with special attention to CV risk factors. Consequently, the tight control of the disease, incorporating frequent use of biological therapy and providing control of traditional CV risk factors, may have contributed to a diminished atherogenic burden among our patients with PsA. This could potentially render them somewhat comparable in terms of CV risk to individuals in the general population.

In our study, the follow-up of CARMA cohort was 7.5 years, which is a limitation to evaluate CV score estimations as these scores were based on 10 years follow-up period. In this sense, we do not know if the fact that the follow-up was 7.5 years compared with the 10 years as applies to the CV risk equations could have contributed to finding fewer CV events than predicted. Nevertheless, our study has several strengths, primarily attributed to its prospective design. Notably, we examined a substantial PsA patient cohort undergoing regular follow-up visits, ensuring standardised data collection and minimising the likelihood of data loss. Moreover, we wish to highlight the potential significance of using two scores in clinical practice. From our perspective, this approach may be clinically relevant for the patients and it is also feasible. Combining two risk chart algorithms may assist in more effectively identifying PsA patients at a high risk of CV events. This information could empower us to proactively implement measures for preventing future CV events in these individuals.

In conclusion, in PsA patients under close observation in rheumatology units included in the prospective CARMA project, risk chart algorithms prove highly valuable for distinguishing individuals at low and high CV risk. The integration of QRISK3 and SCORE2 in a comprehensive model demonstrated an optimal combination, leveraging QRISK3's discrimination capability and SCORE2's calibration accuracy.

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REFERENCES

- Coates LC, Helliwell PS. Psoriatic arthritis: state of the art review. *Clin Med (Lond)* 2017;17:65–70.
- Ocampo D V, Gladman D. Psoriatic arthritis. *F1000Res* 2019;8.
- Papagoras C, Voulgari PV, Drosos AA. Atherosclerosis and cardiovascular disease in the spondyloarthritides, particularly ankylosing spondylitis and psoriatic arthritis. *Clin Exp Rheumatol* 2013;31:612–20.
- Urruticoechea-Arana A, Castañeda S, Otón T, et al. Prevalence of metabolic syndrome in psoriatic arthritis: systematic literature review and results from the CARMA cohort. *J Clin Rheumatol* 2022;28:e388–96.

- 5 Gonzalez-Juanatey C, Llorca J, Miranda-Filloo JA, *et al.* Endothelial dysfunction in psoriatic arthritis patients without clinically evident cardiovascular disease or classic atherosclerosis risk factors. *Arthritis Rheum* 2007;57:287–93.
- 6 Gonzalez-Juanatey C, Llorca J, Amigo-Diaz E, *et al.* High prevalence of subclinical atherosclerosis in psoriatic arthritis patients without clinically evident cardiovascular disease or classic atherosclerosis risk factors. *Arthritis Rheum* 2007;57:1074–80.
- 7 Agca R, Heslinga SC, Rollefstad S, *et al.* EULAR recommendations for cardiovascular disease risk management in patients with rheumatoid arthritis and other forms of inflammatory joint disorders: 2015/2016 update. *Ann Rheum Dis* 2017;76:17–28.
- 8 Peters MJ, van der Horst-Bruinsma IE, Dijkmans BA, *et al.* Cardiovascular risk profile of patients with spondylarthropathies, particularly ankylosing spondylitis and psoriatic arthritis. *Semin Arthritis Rheum* 2004;34:585–92.
- 9 Polachek A, Touma Z, Anderson M, *et al.* Risk of cardiovascular morbidity in patients with psoriatic arthritis: a meta-analysis of observational studies. *Arthritis Care Res (Hoboken)* 2017;69:67–74.
- 10 Castañeda S, Nurmohamed MT, González-Gay MA. Cardiovascular disease in inflammatory rheumatic diseases. *Best Pract Res Clin Rheumatol* 2016;30:851–69.
- 11 Peters MJ, Symmons DPM, McCarey D, *et al.* EULAR evidence-based recommendations for cardiovascular risk management in patients with rheumatoid arthritis and other forms of inflammatory arthritis. *Ann Rheum Dis* 2010;69:325–31.
- 12 Conroy RM, Pyörälä K, Fitzgerald AP, *et al.* Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project. *Eur Heart J* 2003;24:987–1003.
- 13 Perk J, De Backer G, Gohlke H, *et al.* European guidelines on cardiovascular disease prevention in clinical practice (version 2012). the fifth joint task force of the European society of cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of nine societies and by invited experts). *Eur Heart J* 2012;33:1635–701.
- 14 SCORE2 working group and ESC Cardiovascular risk collaboration. SCORE2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in Europe. *Eur Heart J* 2021;42:2439–54.
- 15 Hippisley-Cox J, Coupland C, Brindle P. Development and validation of QRISK3 risk prediction algorithms to estimate future risk of cardiovascular disease: prospective cohort study. *BMJ* 2017;357:j2099.
- 16 Corrales A, González-Juanatey C, Peiró ME, *et al.* Carotid ultrasound is useful for the cardiovascular risk stratification of patients with rheumatoid arthritis: results of a population-based study. *Ann Rheum Dis* 2014;73:722–7.
- 17 Ionescu M, Ionescu P, Suceveanu AP, *et al.* Cardiovascular risk estimation in young patients with ankylosing spondylitis: a new model based on a prospective study in constanta county, romania. *Exp Ther Med* 2021;21:529.
- 18 Galarza-Delgado DA, Azpiri-Lopez JR, Colunga-Pedraza IJ, *et al.* Cardiovascular risk reclassification according to six cardiovascular risk algorithms and carotid ultrasound in Psoriatic arthritis patients. *Clin Rheumatol* 2022;41:1413–20.
- 19 Navarini L, Margiotta DPE, Caso F, *et al.* Performances of five risk algorithms in predicting cardiovascular events in patients with psoriatic arthritis: an Italian bicentric study. *PLoS One* 2018;13:e0205506.
- 20 Corrales A, Vegas-Revenga N, Atienza-Mateo B, *et al.* Combined use of QRISK3 and SCORE as predictors of carotid plaques in patients with rheumatoid arthritis. *Rheumatology (Oxford)* 2021;60:2801–7.
- 21 Corrales A, Vegas-Revenga N, Rueda-Gotor J, *et al.* Carotid plaques as predictors of cardiovascular events in patients with rheumatoid arthritis. Results from a 5-year-prospective follow-up study. *Semin Arthritis Rheum* 2020;50:1333–8.
- 22 Castañeda S, Martín-Martínez MA, González-Juanatey C, *et al.* Cardiovascular morbidity and associated risk factors in Spanish patients with chronic inflammatory rheumatic diseases attending rheumatology clinics: baseline data of the CARMA project. *Semin Arthritis Rheum* 2015;44:618–26.
- 23 Moll JM, Wright V. Psoriatic arthritis. *Semin Arthritis Rheum* 1973;3:55–78.
- 24 Taylor W, Gladman D, Helliwell P, *et al.* Classification criteria for psoriatic arthritis: development of new criteria from a large international study. *Arthritis Rheum* 2006;54:2665–73.
- 25 Visseren FLJ, Mach F, Smulders YM, *et al.* 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. *Eur Heart J* 2021;42:3227–337.
- 26 Shen J, Lam SH, Shang Q, *et al.* Underestimation of risk of carotid Subclinical atherosclerosis by cardiovascular risk scores in patients with psoriatic arthritis. *J Rheumatol* 2018;45:218–26.
- 27 Navarini L, Margiotta DPE, Costa L, *et al.* Performance and calibration of the algorithm ASSIGN in predicting cardiovascular disease in Italian patients with psoriatic arthritis. *Clin Rheumatol* 2019;38:971–6.
- 28 Taylor W, Currado D, Marino A, *et al.* Persistence of C-reactive protein increased levels and high disease activity are predictors of cardiovascular disease in patients with axial spondyloarthritis. *Sci Rep* 2022;12:7498.
- 29 Ferraz-Amaro I, Corrales A, Quevedo-Abeledo JC, *et al.* Disease activity influences the reclassification of rheumatoid arthritis into very high cardiovascular risk. *Arthritis Res Ther* 2021;23:162.
- 30 Coates LC, Fransen J, Helliwell PS. Defining minimal disease activity in Psoriatic arthritis: a proposed objective target for treatment. *Ann Rheum Dis* 2010;69:48–53.
- 31 Karpouzas GA, Ormseth SR, Hernandez E, *et al.* Biologics may prevent cardiovascular events in rheumatoid arthritis by inhibiting coronary plaque formation and stabilizing high-risk lesions. *Arthritis Rheumatol* 2020;72:1467–75.
- 32 Karpouzas GA, Ormseth SR, Hernandez E, *et al.* The impact of Statins on coronary Atherosclerosis progression and long-term cardiovascular disease risk in rheumatoid arthritis. *Rheumatology (Oxford)* 2022;61:1857–66.
- 33 Martín-Martínez MA, Castañeda S, Sánchez-Alonso F, *et al.* Cardiovascular mortality and cardiovascular event rates in patients with inflammatory rheumatic diseases in the cardiovascular in rheumatology (CARMA) prospective study—results at 5 years of follow-up. *Rheumatology (Oxford)* 2021;60:2906–15.