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D-dimer level in systemic lupus erythematosus and its correlation with disease activity, a systematic review and meta-analysis

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ABSTRACT

In systemic lupus erythematosus (SLE), thrombotic events represent a high-risk manifestation associated with autoantibody-induced damage. This underscores the necessity of utilizing biomarkers to assess thrombotic risk and monitor the thrombotic status of patients. D-dimer is a widely used and inexpensive marker routinely-used to assess coagulation disorders. A systematic literature search was conducted from 1990-2024 using the PubMed, Scopus, and Web of Science databases. Included articles reported D-dimer serum levels in both SLE patients and healthy individuals. Data were analyzed using standard mean difference (SMD) with a 95% confidence interval using random effect models. Heterogeneity was assessed based on the Cochran chi-square and I^2 statistic. Article quality was assessed via the Newcastle-Ottawa scale. This study incorporated data from 14 studies, encompassing individuals diagnosed with SLE and 1785 healthy participants serving as controls. The results indicated a significant increase in serum D-dimer level in the SLE group compared to the control group (SMD= 0.74, 95% CI 0.49, 1.00, $p < 0.001$; $I^2 = 87.47\%$, $p < 0.001$). Moreover, patients with high SLE disease activity had higher D-dimer levels compared to those with low SLE disease activity (SMD=0.78, 95% CI 0.46, 1.11; $I^2 = 0$, $p = 0.77$). Moreover, a significant positive correlation was found between SLE disease activity and D-dimer concentrations ($r = 0.45$, 95% CI 0.34, 0.55, $p < 0.0001$). D-dimer may serve as an indicator of thrombotic status and disease severity in SLE patients. Establishing D-dimer as a reliable biomarker for tracking SLE activity will require well-designed prospective studies that consider standardized assays, control for confounding factors, and incorporate predictive modelling.

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Introduction

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease resulting from the dysregulation of both innate and adaptive immune responses which can impact various organs throughout the body, including kidney, blood, heart, skin (1). Over recent decades, survival rate for patients with SLE has improved. However, the mortality rate remains higher than that of the general population. Disease activity, infection, and thrombotic events have been reported as the leading causes of death among these patients (2). SLE patients have a 2- to 10- fold higher risk of thrombotic events, including both arterial and venous thrombosis, which contribute significantly to morbidity and mortality (3, 4). The presence of anti-phospholipid antibodies, which bind to phospholipids on endothelial cells, active tissue factors, and induce platelet aggregations, has been recognized as a key contributor to the increased risk of thrombosis in these patients (5, 6). The identification and evaluation of biomarkers are essential for monitoring patients with thrombosis activities, which in turn facilitates the prevention of thromboembolic events and their associated complications. D-dimer is a fibrin degradation product generated during the breakdown of cross-linked fibrin, serving as an indicator of both thrombus formation and fibrinolysis. Thus, D-dimer reflects not only thrombotic activity but also systemic inflammation and fibrin turnover (7). Persistently high D-dimer concentrations after cessation of anticoagulation therapy are associated with increased risk of recurrent venous thromboembolism (8). Clinically, D-dimer testing is commonly employed in evaluating patients suspected of having disseminated intravascular coagulation and is gaining prominence as a preliminary diagnostic tool for venous thromboembolism (9).

However, the utility of D-dimer testing for screening thrombosis and assessing disease activity in SLE patients has not been comprehensively evaluated. This study aimed to conduct a systematic review and meta-analysis to investigate D-dimer level differences between SLE patients and healthy controls, and to examine the correlation between D-dimer concentrations and disease activity in SLE. By integrating data from current studies, we are looking to provide a comprehensive assessment of D-dimer's potential role as a biomarker for disease activity in

SLE, ultimately contributing to improved clinical management of this complex disease.

Methods

Study design

This study was registered in PROSPERO on October 9, 2024 (CRD42024591152) and its results were reported according to the PRISMA guidelines.

Search strategy

An extensive literature search was carried out across PubMed, Web of Sciences, and Scopus databases from 1990 to September 2024. The search queries were selected based on MESH including "Systemic Lupus Erythematosus" OR "systemic lupus erythematoses" OR "systemic lupus erythematosus" OR "systemic lupus erythematosus" OR "SLE" AND "D-dimer fibrin" OR "D-dimer fragments" AND "D-dimer" OR "fibrin fragment D1 dimer" OR "fibrin fragment DD" OR "fibrin fragment D-dimer" OR "fibrin degradation product d dimer" OR "D dimer".

Inclusion and exclusion criteria

Two independent researchers (ABS and FN) selected eligible articles based on the following criteria. The inclusion criteria encompassed case-control, cross-sectional, and cohort studies published in English, 2- studies reported mean \pm standard deviation (SD) of D-dimer levels ($\mu\text{g/ml}$) in patients with SLE and healthy controls, 3- studies reported the mean \pm SD of D-dimer levels ($\mu\text{g/ml}$) in groups with high and low SLE disease activity, categorized using the SLE disease activity index (SLEDAI). A SLEDAI score ≤ 4 was considered indicative of low disease activity, while a score >4 was classified as high disease activity. The exclusion criteria comprised: 1-review, clinical trial, and editorial articles, 2-studies lacking a healthy control group, duplicate publications, studies with potentially erroneous D-dimer values, and animal studies. Moreover, to avoid reprint bias, the article results were completely examined, and any duplicates were excluded.

Quality assessment

In this study, the quality of the selected articles was independently assessed by two authors (ABS and FN) using the Newcastle-Ottawa Scale (NOS) checklist for case-control, cross-sectional, and cohort studies. This

scale evaluates studies based on the three broad criteria: selection of study groups, comparability of groups, and ascertainment of either the exposure or outcome of interest. Each study can be awarded a maximum of nine stars. The studies included in our meta-analysis met the criteria for acceptable quality.

Statistical analysis

In this study heterogeneity was assessed using the Cochran chi-square and I² statistic. Due to the significant heterogeneity, the Laird random effect model was used to combine the results of the studies.

A Forest plot of mean \pm SD D-dimer levels was generated to assess the difference in D-dimer concentrations between SLE patients and healthy controls. The robustness of the estimated overall effect size was determined by excluding each study using the Leave-One-Out method. A subgroup analysis was conducted to evaluate the association between effect size and specific parameters under investigation, including publication year, study type, quality assessment score, and geographic region. All analysis was conducted using Stata version 17.

Results

Study selection

From the initial 435 documents identified through the systematic search, after screening and eligibility, 14 studies met the eligibility criteria and were ultimately included.

An outline of the study selection workflow is depicted in Figure 1. The 14 selected studies, assessed the D-dimer in 1236 SLE patients (mean age 39.96 years, 1124 (90.9%) and 1785 healthy controls (mean age 40.60 years, 1416 (81.6%) females). The continents where the studies were conducted included East Asia (n= 7), Europe (n= 4), and for each the continents of North America, South America and Australia (n=1). 11 studies were case-control studies, 2 studies were cross-sectional, and there was only one cohort study. Mean disease duration, as reported in 8 study groups, ranged between 2.15 and 15.5 years. The characteristics of the selected articles are detailed in Table 1.

Main results

The forest plot analysis revealed that individuals with SLE exhibited markedly elevated D-dimer levels in comparison to healthy controls (SMD = 0.74, 95%

CI 0.49-1.00, $p < 0.001$; I² = 87.47%, $p < 0.001$; Figure 2). The pooled SMD of the D-dimer levels in the high active group compared to the low active group showed a statistically significant difference (SMD=0.78, 95% CI 0.46-1.11; I² = 0, $p=0.77$) (Figure 3). The pooled SMD of D-Dimer levels in both high and low activity SLE groups was significantly higher compared to the control group (SMD=1.32, 95% CI 0.6-2.03; I² = 75.87, $p=0.02$) and (SMD= 0.77, 95% CI 0.46-1.08; I² = 0.0, $p=0.96$), respectively. Moreover, there was a positive and statistically significant correlation between SLE disease activity and D-dimer concentrations [$r = 0.45$, 95% CI 0.34-0.55, $p < 0.0001$; with no observed heterogeneity (I²=0, $p=0.530$) (Figure 4).

Sensitivity analysis and Publication bias

Sensitivity analysis confirmed the robustness of the pooled SMD, as exclusion of individual studies did not materially alter the overall effect estimate (Figure 5). Neither Begg's test ($p= 0.274$) nor Egger's test ($p= 0.484$), and the funnel plot, revealed significant evidence of publication bias (Figure 6).

Subgroup analysis

In the subgroup analyses stratified by publication year, study type, geographical region, and quality assessment score, the SMD in serum D-dimer levels remained statistically significant for studies published both before 2010 (SMD=0.79, 95% CI 0.51-1.07; I² = 87.92, $p < 0.001$) and after 2010 (SMD=0.59, 95% CI 0.01, 1.18; I² = 85.5, $P < 0.001$). Studies employing latex agglutination assays yielded a statistically significant increase D-dimer levels (SMD= 0.93, 95% CI 0.42, 1.43; I² = 74.94, $p= 0.019$), whereas ELISA-based studies did not show a significant difference (SMD= 0.68, 95% CI -0.09, 1.47; I² = 87.02, $p= 0.001$).

The SMDs were significant in cohort studies (SMD= 0.95, 95% CI 0.82-1.09), case-control studies (SMD= 0.66, 95% CI 0.36, 0.97; I² = 86.5, $p < 0.001$), and cross-sectional studies (SMD= 1.05, 95% CI 0.56, 1.5; I² = 47.39, $p=0.168$). Based on continental subgroup analysis, the SMD was statistically significant in studies conducted in the America (SMD= 0.72, 95% CI 0.39, 1.04; I²=0.00, $P=0.494$), and East Asia (SMD= 0.82, 95% CI 0.54, 1.09; I²= 81.78%, $p= 0.002$), but not in Europe (SMD=0.67, 95% CI -0.24, 1.57; I²=92.98, $p < 0.001$).

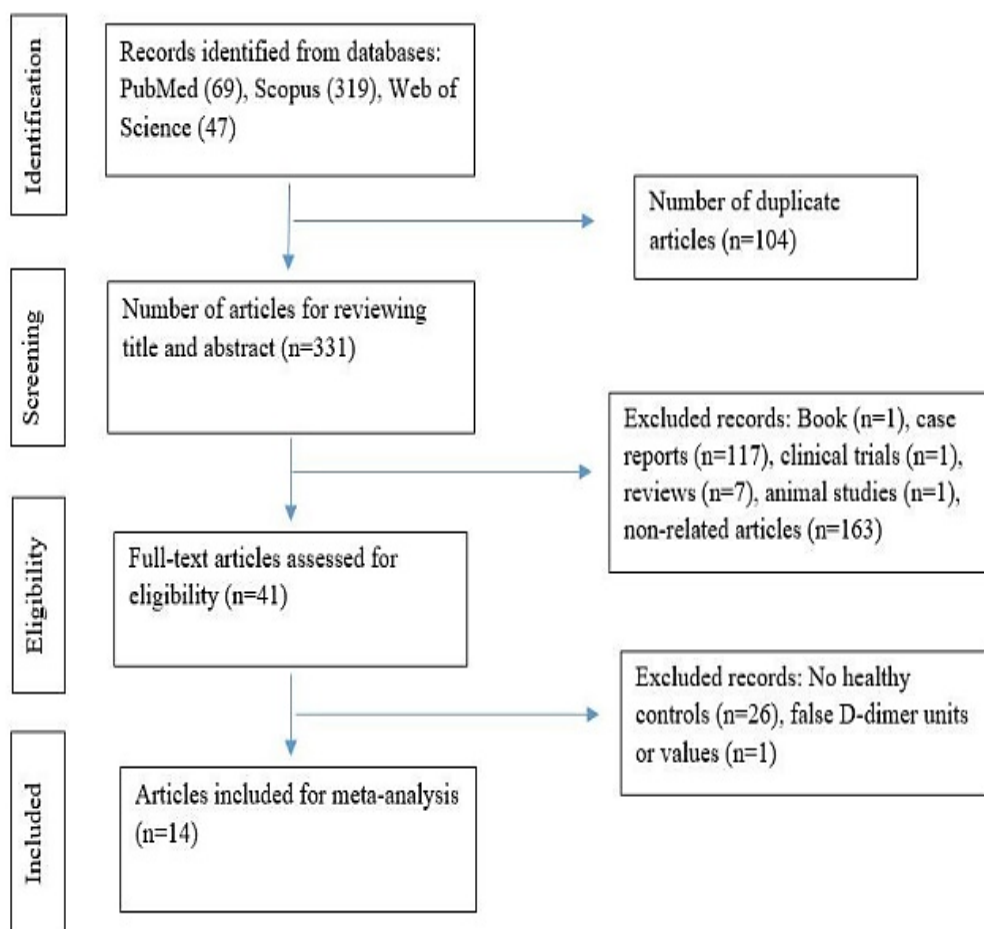


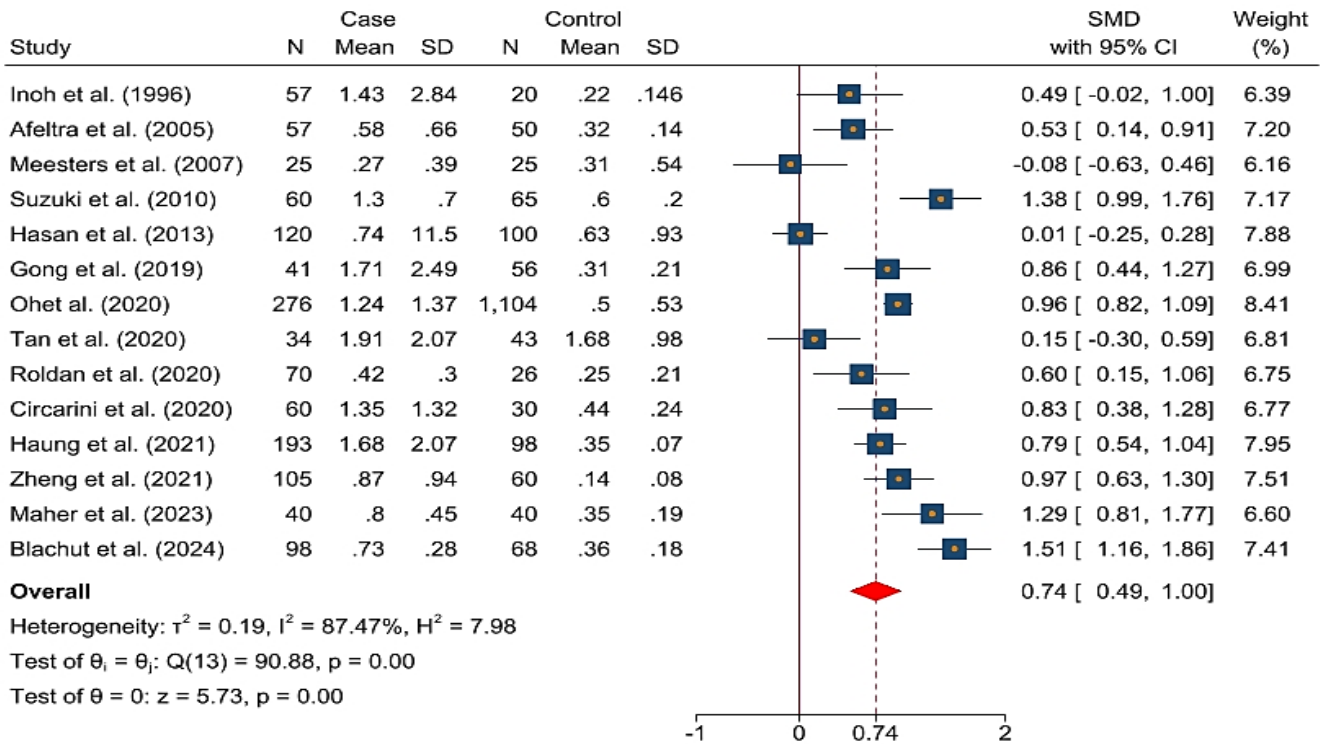
Figure 1. A summary overview of the search and selection process in the meta-analysis

Table 1. Characteristics of the included studies

Author	Country	Year	Study type	Quality assessment	SLEDAI-2K	Study test	SLE patients				Healthy controls			
							Size	Gender (F/M)	Age	D-dimer (µg/ml)	Size	Gender (F/M)	Age	D-dimer (µg/ml)
Inoh (10)	Japan	1996	Case control	5	-	Latex Agglutination	57	57/0	30.61±9.89	1.39 ± 2.93	20	20/0	30.5±5.07	0.22±0.146
Afeltra (11)	Italian	2005	Case control	8	-	-	57	49/8	59.89 ± 11.47	0.73 ± 0.28	50	matched	matched	0.32±0.14
Meesters (12)	Netherlands	2007	Case control	7	-	ELISA	25	25/0	41.7 ± 14.0	0.27±0.39	25	25/0	41.4 ± 11.7	0.31±0.53
SUZUKI (13)	Japan	2010	Case control	6	2.5 (0-10)	Latex Agglutination	60	57/3	15.5 ± 9.9	1.3 ± 0.7	43	25/18	28	0.6 ± 0.2
Zheng (14)	China	2021	Case control	7		-	105	95/10	42	0.87±0.94	60	54/6	39	0.14±0.08
Circarini (15)	Brazil	2020	Cross sectional	6	4.50 (0-18)	ELISA	60	60/0	40.13±13.65	1.35±0.19	30	30/0	matched	0.44 ± 0.24

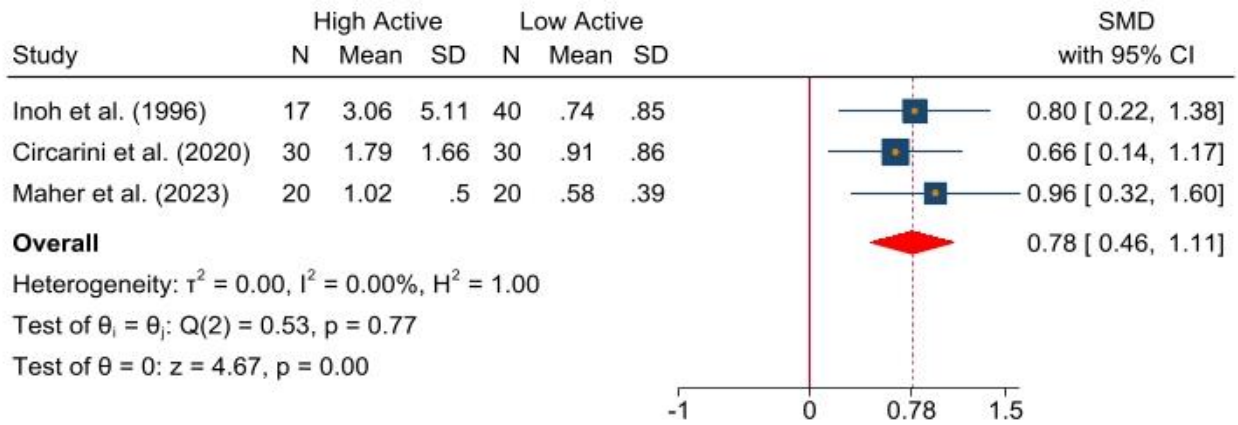
Author	Country	Year	Study type	Quality assessment	SLEDAI-2K	Study test	SLE patients				Healthy controls			
							Size	Gender (F/M)	Age	D-dimer (µg/ml)	Size	Gender (F/M)	Age	D-dimer (µg/ml)
Oh (10)	Korea	2020	Retrospective cohort	9	-	-	276	241/35	36.7 ± 13.6	1.24 ± 1.37	1104	932/172	38.0 ± 12.6	0.5 ± 0.53
Tan (16)	China	2020	Case control	8		latex immunoturbidimetry	34	20/14	62 ± 14	1.91 ± 2.07	43	21/22	67 ± 10	1.68 ± 0.98
Roldan (17)	USA	2020	Case control	8	11.68 ± 9.40	-	70	64/6	36.0	4.17 ± 3.03	26	22/4	31.5	2.47 ± 2.12
Haug (18)	China	2021	Case control	8	9.52 ± 4.05	-	193	183/10	37.19 ± 12.01	1.68 ± 2.07	98	78/20	39.35 ± 10.09	0.35 ± 0.07
Maher (19)	Egypt	2023	Cross sectional	6		ELISA	40	40/0		0.8 ± 0.05	40	40/0	12.3	0.35 ± 0.19
Blachut (20)	Poland	2024	Case control	6	8.18 ± 7.78	-	98	87/11	59.89 ± 11.47	0.73 ± 0.28	68	60/8	54.86 ± 10.87	0.36 ± 0.18
Gong (21)	China	2019	Case control	5	5.55 (0-17)	Latex Agglutination	41	36/5	43.4	1.71 ± 2.49	56	10/46	63.2	0.31 ± 0.21
Hassan (22)	Egypt	2013	Case control	7		-	120	110/10	32 ± 7.75	0.74 ± 11.5	100	90/10	30 ± 8.25	0.63 ± 9.3

SLEDAI-2K, systemic lupus erythematosus disease activity index 2000



Random-effects empirical Bayes model
Sorted by: Year

Figure 2. Forest plot of studies analyzing D-dimer concentration in patients with SLE compared to the control group



Random-effects empirical Bayes model
 Sorted by: Year

Figure 3. Comparison of D-dimer concentration between SLE patients with high and low activity disease state

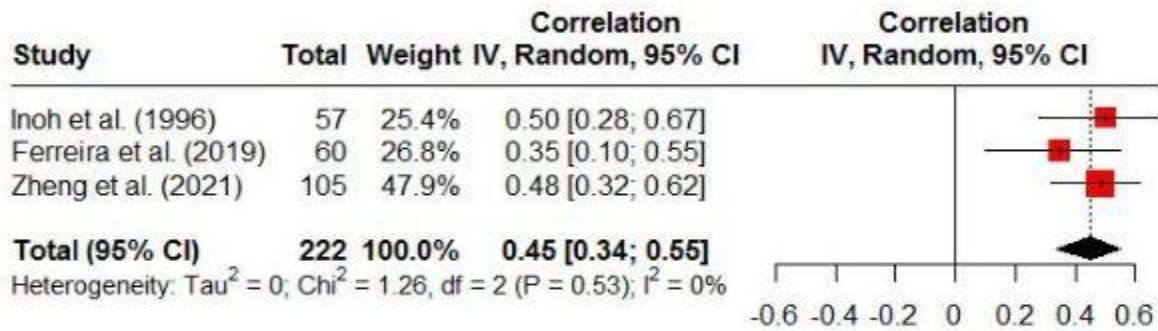


Figure 4. Forest plot of studies investigating the correlation between D-dimer levels and SLE diseases activity

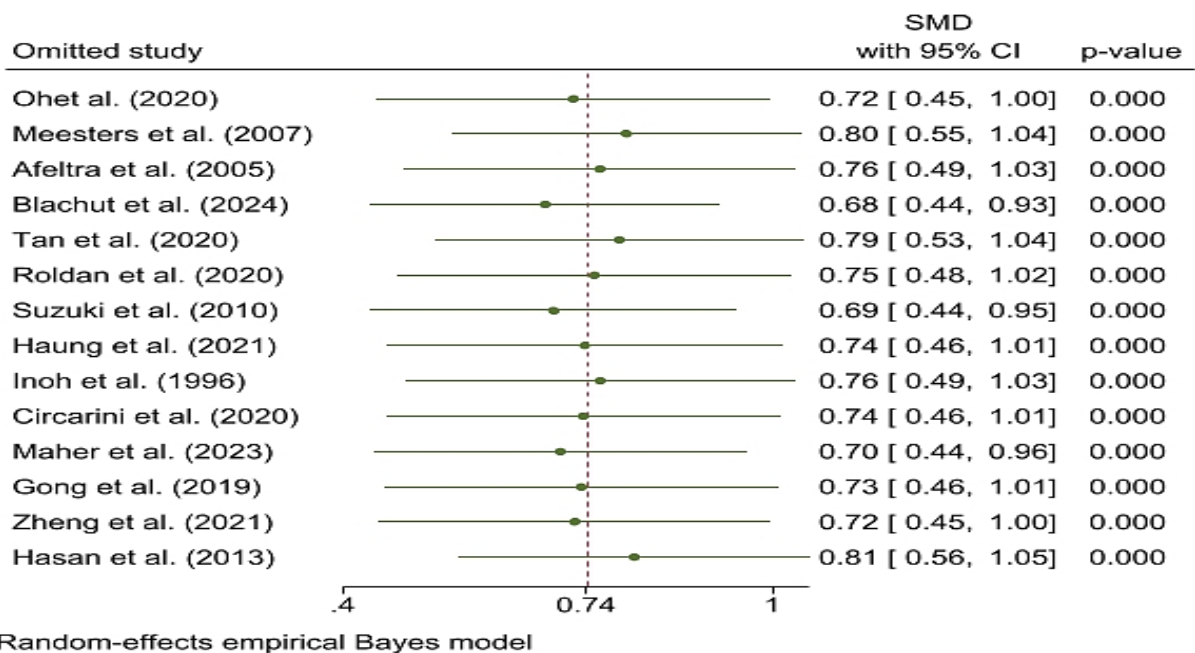


Figure 5. Sensitivity analysis results

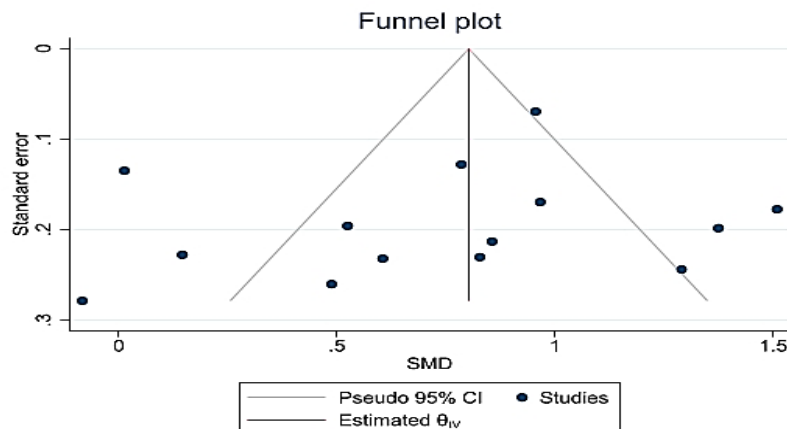


Figure 6. Funnel plot analysis of the included studies

Conversely, in Africa, the pooled SMD value showed no statistically significant difference between African patients and healthy control (SMD=0.63, 95% CI -0.62 to 1.88; $I^2=95.24$, $p<0.001$). Based on the NOS scale, the pooled SMD for both high and moderate quality studies was statistically significant (SMD= 0.52, 95% CI 0.23-0.81; $I^2= 86.27$, $P<0.0001$), and (SMD= 1.08, 95% CI 0.77-1.39; $I^2= 69.03$, $p=0.0007$), respectively (Table 2). Moreover, significantly elevated levels of CRP, ESR, and fibrinogen levels were observed in SLE patients compared to controls with SMD of 0.54 (95% CI 0.02-1.06, $p < 0.001$; $I^2 = 92.03$, $p < 0.001$), 0.99 (95% CI 0.55-1.43, $p < 0.001$; $I^2 = 57.74$, $p = 0.12$), and 0.78

(95% CI 0.50-1.05, $p < 0.001$; $I^2 = 73.53$, $p < 0.001$). Conversely, PLT levels showed the opposite results (SMD= -0.84, 95% CI -0.67 to -1.02, $p < 0.001$; $I^2 = 18.07$, $p=0.33$).

Meta-regression analysis

According to the meta-analysis regression in Table 3, the effect size was not associated with the female-to-male ratio ($\beta=-0.012$, $p=0.812$), age ($\beta=-0.002$, $p=0.879$), and mean disease duration ($\beta=0.056$, $p=0.263$). Similarly, no significant association was observed with publication year (Figure 7), different geographic regions, type of studies, fibrinogen, platelet, and CRP ($P>0.05$).

Table 2. Sub-group analysis of D-dimer levels based on publication year, study type, geographic region, and quality assessment

Variable Subgroup (number of datasets)	SLE patients (n)	Healthy controls (n)	SMD % (95% CI)	I^2	Q	p-value
Publication year						
Before 2010 (10)	1037	1625	0.79 (0.51, 1.07)	87.92	68.77	<0.001
After 2010 (4)	199	160	0.59 (0.01, 1.18)	85.5	21	<0.001
Type of studies						
Cohort (1)	276	1104	0.95 (0.82, 1.09)	-	-	-
cross-sectional (2)	100	70	1.05 (0.56, 1.5)	47.39	1.9	0.168
Case-control (11)	860	611	0.66 (0.36, 0.97)	86.5	77.04	<0.001
Geographic regions						
Global (14)	1236	1785	0.74 (0.49, 0.99)	87.47	90.88	<0.001
America (2)	130	56	0.72 (0.39, 1.04)	0.00	0.47	0.494
USA (1)	70	26	0.6 (0.15, 1.06)	-	-	-
Brazil (1)	60	30	0.83 (0.38, 1.28)	-	-	-
East Asia (7)	766	1446	0.82 (0.54, 1.09)	81.78	20.84	0.002
China (4)	373	257	0.71 (0.36, 1.05)	74.05	8.98	0.03
Japan (2)	117	85	0.95 (0.8, 1.82)	86.42	7.36	0.007
Korea (1)	276	1104	0.96 (0.82, 1.09)	-	-	-
Europe (3)	180	143	0.67 (-0.24, 1.57)	92.98	27.74	<0.001
Italian (1)	57	50	0.52 (0.14, 0.91)	-	-	-

Poland (1)	98	68	1.51 (1.6, 1.86)	-	-	-
Netherland (1)	25	25	-0.08(-0.63, 0.46)	-	-	-
Africa (2)	160	140	0.63 (-0.62, 1.88)	95.24	21.01	<0.001
Egypt (2)	160	140	0.63 (-0.62, 1.88)	95.24	21.01	<0.001
Quality assessment score						
Moderate (6)	356	279	1.08 (0.77, 1.39)	69.03	15.98	0.007
High (8)	880	1506	0.52 (0.23, 0.81)	86.27	57.06	<0.001

Table3. Meta-regression analysis of factors associated with D-dimer standardized mean difference (SMD)

Variables	Coefficient	95% CI	P-value	R-squared
Publication year	0.027	(-0.004,0.057)	0.091	0.16
Geographic regions				0.26
America	0.109	(-0.970,1.189)	0.843	-
East Asia	0.199	(-0.653, 1.053)	0.646	-
Europe	0.079	(-0.900, 1.058)	0.874	-
Africa	Ref.			-
Type of studies				0.21
Cohort	0.290	(-0.665, 1.245)	0.552	-
cross-sectional	0.391	(-0.383, 1.165)	0.322	-
Case-control	Ref.			-
Age	0.584	(-1.998, 3.166)	0.657	0.23
CRP	0.052	(-0.114, 0.220)	0.536	0.35
Fibrinogen	-0.187	(-0.771, 0.397)	0.530	0.23
platelets	-3.096	(-9.253, 3.059)	0.324	0.16
Sample Size	-0.044	(-0.411, 0.321)	0.811	0.21

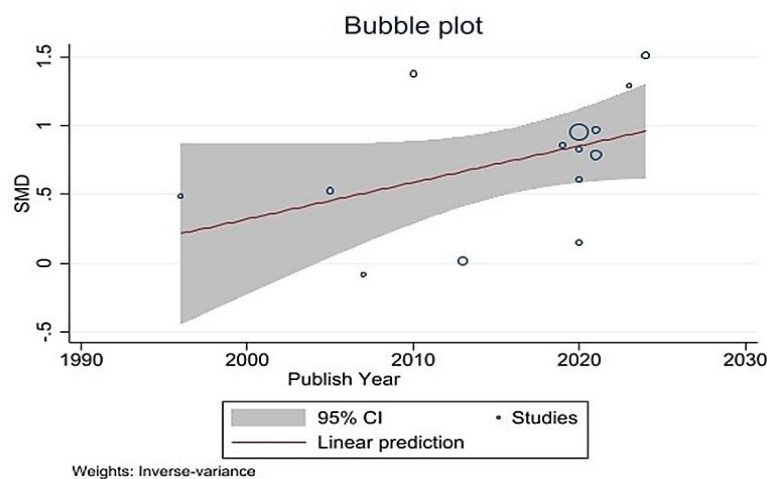


Figure 7. Meta-Regression Bubble Plot of D-dimer Standardized Mean Difference (SMD) by Publication Year.

Discussion

D-dimer measurement, as an indicative of fibrin clot formation and thrombosis, has been correlated with severity and outcome in various autoimmune and inflammatory conditions (23). In this meta-analysis,

we merged data on serum D-dimer levels from 1236 SLE patients and 1785 healthy controls. Our findings revealed a significant elevation in serum D-dimer levels among SLE patients compared to normal subjects. Moreover, patients with higher disease

activity scores had greater D-dimer levels than those with lower disease activity.

This observation aligns with previous individual studies reporting a positive correlation between elevated D-dimer levels and severity of lupus disease (24). Wu et al. reported that regular monitoring of D-dimer concentrations in individuals with SLE may serve as a predictive marker for thrombotic events. Patients with normal D-dimer levels had a low risk of thrombosis, whereas those with D-dimer levels between 0.5-2 and above 2 µg/ml experienced a higher risk of thrombosis (25). Besides, SLE patients with thrombosis exhibited a remarkable elevation in the SLEDAI compared to those without thrombosis (26). Thus, D-dimer may function as a valuable indicator for identifying SLE patients with an increased risk for aggressive thrombotic events and high disease activity.

Sub-group analysis by assay revealed notable heterogeneity across both ELISA-and latex agglutination-based assays, suggesting that methodological differences may substantially influence the magnitude and consistency of D-dimer measurements. This variability between assays is attributable to differences in analytical sensitivity, calibration standards, and unit reporting.

Substantial geographic heterogeneity may reflect differences in diagnostic criteria, healthcare infrastructure, and population-level risk factors across countries. Moreover, the heterogeneity observed across study designs may be attributed to variations in methodological quality, participant selection criteria and outcome.

The limitations of this meta-analysis include heterogeneity in the units used to report D-dimer levels, which necessitated the use of the SMD for consistent reporting. This led to a lack of precise and reliable data for clinical assessments. Additionally, most of the included studies, did not consider the use of drugs that interfere with coagulation pathways, the dose of glucocorticoids, markers related to inflammation and dyslipidemia. D-dimer levels can also be influenced in the presence of anti-phospholipid antibodies or anti-phospholipid syndrome (APS), active infections, and pregnancy. Moreover, only the study by Oh, et al. conducted a receiver operating characteristic (ROC) curve analysis for serum D-dimer levels. The results indicated that D-dimer has low specificity in evaluating thrombosis in SLE patient compared to healthy individuals (10). Another

drawback was the small number of eligible studies, which reduced the precision and strength of the conclusions regarding the role of D-dimer as a reliable diagnostic and prognostic marker for monitoring thrombosis-related complications in patients with SLE.

The findings of this analysis indicate that patients with SLE exhibit elevated D-dimer levels, which show a significant correlation with disease activity. To confirm D-dimer as a valuable biomarker for longitudinal monitoring of disease activity, future research should focus on prospective cohort studies employing standardized D-dimer assays, adjusted analyses, and robust diagnostic and prognostic modeling approaches.

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