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Higher Rates and Clustering of Abnormal Lipids, Obesity, and Diabetes in Psoriatic Arthritis Compared with Rheumatoid Arthritis

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Abstract

Objective—We compared the prevalence and the clustering of the Metabolic Syndrome (MetS) components: obese body mass index ($\text{BMI} \geq 30 \text{ kg/m}^2$), hypertriglyceridemia, low high-density lipids, hypertension and diabetes, in patients with psoriatic arthritis (PsA) and rheumatoid arthritis (RA) in the Consortium of Rheumatology Researchers of North America (CORRONA) registry.

Methods—We included CORRONA participants with the rheumatologist-confirmed clinical diagnoses of PsA and RA with complete data. We used a modified definition of MetS that did not include insulin resistance, waist circumference or blood pressure measurements. Logistic regression models were adjusted for age, sex and race.

Results—In the overall CORRONA population, the rates of diabetes and obesity were significantly higher in PsA compared with RA. In 294 PsA and 1162 RA participants who had lipids measured, the overall prevalence of MetS in PsA vs. RA was 27% vs. 19%. The odds ratio (OR) of MetS in PsA vs. RA was 1.44 (95% confidence interval (CI) 1.05 to 1.96), $p=0.02$. The prevalence of hypertriglyceridemia was higher in PsA compared with RA, 38% vs. 28%, OR 1.51 (95% CI 1.15 to 1.98), $p=0.003$. The prevalence of type II diabetes was also higher in PsA compared with RA (15% vs. 11%), OR 1.56 (95% CI 1.07 to 2.28), $p=0.02$, in the adjusted model. Similarly, higher rates of hypertriglyceridemia and diabetes were observed in the subgroup of PsA and RA patients with obese BMI.

Conclusion—PsA is associated with the higher rates of obesity, diabetes, and hypertriglyceridemia, compared with RA.

Over the last decade, high frequencies of cardiovascular disease (CVD) and events have been reported in chronic inflammatory arthritides. Numerous studies have documented an increased burden of CVD in patients with rheumatoid arthritis (RA) (1–5), including a meta-analysis of observational studies reporting a 50% increase in all-cause CVD mortality compared to the general population (6). The magnitude of cardiovascular (CV) burden in RA may in fact equal the CV risk in patients with type II diabetes (7, 8). Similarly, in a retrospective analysis of a large healthcare claims database in the United States (US), Han et al reported that the age and sex adjusted prevalence ratios of ischemic heart disease, peripheral vascular disease, congestive heart failure, and cerebrovascular disease were higher in psoriatic arthritis (PsA) patients than matched controls (9). Furthermore, a

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prospective Canadian study demonstrated a two- to three- fold increase in standardized prevalence ratios for myocardial infarction and angina in PsA (10). However, in 2010 the European League Against Rheumatism (EULAR) found compelling evidence to recommend CV risk screening and management for RA and PsA patients, but noted that the evidence about the magnitude of the CV risk in PsA was less robust (11).

These EULAR guidelines highlight that our understanding of CV disease in PsA has often been extrapolated from RA patients. While systemic inflammation and persistent elevations of pro-inflammatory cytokines are implicated in the increased CV risk observed in both diseases (12, 13), PsA is a distinct entity that includes features of axial disease, dactylitis, enthesitis, and skin and nail disease not seen in RA (14). When considering that PsA is characterized by inflammation of both skin and joints, we may in fact be underestimating CV risk in PsA.

Metabolic Syndrome (MetS) is a clustering of classical and modifiable cardiovascular risk factors, including insulin resistance, central obesity, elevated blood pressure, high triglyceride levels and low levels of high-density lipoprotein (15, 16). The importance of MetS is that it identifies individuals who are at high risk of CV disease, and may confer a CV risk which is higher than the sum of the individual components (17, 18). In chronic inflammatory conditions, such as RA (19–22) and psoriasis (PsO) (23–25), previous studies have shown that MetS and its components are highly prevalent with rates up to three times those reported in the general population. Although individual components of the MetS such as obesity (26–28), hypertension (9, 26–28), diabetes mellitus (9, 26, 28), and dyslipidemia (9, 27–29) have been reported with higher frequency in PsA, there are limited data regarding the prevalence of MetS in PsA (26, 30), and in comparison with RA and PsO patients (31, 32). A recent study conducted in Hong Kong by Mok et al. reported a higher prevalence of MetS in PsA compared with RA and ankylosing spondylitis (33). Furthermore, Eder et al. demonstrated that the prevalence of MetS, insulin resistance, and adipose tissue biomarkers (leptin and adiponectin) was higher in PsA compared with PsO (34).

We hypothesized that the prevalence of MetS and its components is higher in PsA than in RA. Thus, we compared the prevalence of MetS and its components among patients with PsA and RA using data from a large US-based cohort of inflammatory arthritis patients.

Patients and Methods

Study Design and Population

We conducted a cross-sectional study of PsA and RA patients enrolled in the Consortium of Rheumatology Researchers of North American (CORRONA) registry. The CORRONA registry is a prospective observational cohort of patients with either RA or PsA who are enrolled by participating rheumatologists in both academic and private practice sites within the USA (35). The CORRONA registry was approved by the institutional review boards of participating academic sites and a central institutional review board for community-based private sites, and all patients signed informed consent before participation.

We included CORRONA participants enrolled between October 2001 and October 2010 with a rheumatologist-confirmed clinical diagnosis of PsA and RA. The comparison of the MetS was restricted to PsA and RA participants who had at least one visit where serum cholesterol levels were available for analysis, as lipid measurements are not mandated in the registry, and lipid information was not gathered before June 2008. All of the data elements required to determine whether patients met formal diagnostic criteria were not available.

Measures and Data Collection

Data were collected from both patients and their treating rheumatologists using clinical research forms at enrollment and at follow-up visits at the time of a routine clinical encounter. Data elements collected in the registry included demographic information and disease specific variables such as disease duration, medications, laboratory parameters, tender and swollen joint counts, presence of enthesitis and sausage digits, as well as various measures of disease activity by physicians and patients (36, 37).

Data on cardiovascular risk factors and lipid parameters were also recorded by the rheumatologist using CORRONA questionnaires. The data were collected in the same manner for PsA and RA groups.

Definition of metabolic syndrome

The most recent definition of MetS is devised by the International Diabetes Federation (IDF) (38). These criteria define the presence of MetS if there is central obesity (defined as waist circumference ≥ 40 inches (102 cm) in men and ≥ 35 inches (88 cm) in women or if BMI ≥ 30 kg/m²) AND any two of the following: (1) hypertriglyceridemia, TG > 150 mg/dL (1.7 mmol/L), (2) low high-density lipid, HDL < 40 mg/dL (1.0 mmol/L) in males; HDL < 50 mg/dL (1.3 mmol/L) in females, (3) elevated blood pressure (BP), systolic BP ≥ 130 mm Hg or diastolic BP ≥ 85 mm Hg or treatment of previously diagnosed hypertension, or (4) elevated fasting plasma glucose, ≥ 100 mg/dL (5.6 mmol/L) or previously diagnosed type II diabetes. Since waist circumference, blood pressure measurements and fasting glucose were not uniformly available throughout the CORRONA data set, we used the following components to define MetS: (1) BMI ≥ 30 kg/m² as our designation of central obesity, (2) a diagnosis of type 2 diabetes mellitus, and (3) a diagnosis of hypertension. To explore if our findings were independent of the criteria used, we also performed additional analyses using 2 other validated and widely used definitions: World Health Organization (WHO) definition and Adult Treatment Panel III (ATP III) definition (39). The modified WHO criteria was defined as history of diabetes plus at least 2 of the following: (1) BMI ≥ 30 kg/m², (2) triglycerides (TG) of 150mg/dl or greater, and/or high density lipoprotein (HDL)-cholesterol < 40 mg/dl in men and < 50 mg/dl in women, (3) history of hypertension. The modified ATP III criteria was defined as any three of the following: (1) BMI ≥ 30 kg/m² (2) TG of 150mg/dl or greater, (2) HDL-cholesterol < 40 mg/dl in men and < 50 mg/dl in women, (3) history of hypertension, (4) history of diabetes. The strength of this approach is that the criteria we used to define MetS are readily available to rheumatologists in clinical practice.

Statistical Analysis

First, we compared the prevalence of MetS and its individual components: obese BMI (BMI ≥ 30 kg/m²), hypertriglyceridemia, low HDL, hypertension and diabetes, analyzed as dichotomous variables (yes/no) between PsA and RA participants in the CORRONA registry who had complete information about MetS components. We also compared lipid profiles of PsA and RA participants, using the following cut-offs based on previously established definitions for CVD risk: high TC (TC > 200 mg/dL), low HDL (HDL < 40 mg/dL in males and HDL < 50 mg/dL in females), high LDL (LDL > 100 mg/dL), and hypertriglyceridemia (TG > 150 mg/dL), and elevated TC/HDL ratio, TC/HDL > 3.5 (40–42). In addition, we compared the rates of diabetes, hypertension, and obesity in a larger subset of PsA and RA participants who had these data available, but did not have lipid profiles reported.

We used the student's t-test (or its non-parametric alternative, Wilcoxon rank sum test) to evaluate the differences between distributions of continuous variables, and chi-square (or Fisher's exact test when appropriate) to evaluate the association between categorical

variables. Differences were considered statistically significant for $p < 0.05$ (two-tailed). Continuous variables were reported as mean $\pm$ SD, and categorical variables were reported as frequencies. We used multivariable logistic regression to adjust for potential confounders and to check for interactions. Since the frequency of MetS increases with age and BMI and varies with sex and race (43), we adjusted for these variables in our analyses. We decided a priori not to adjust our models for medication use, disease activity, prior history of MI, stroke or congestive heart failure, because it is not possible to establish whether these variables were a part of a causal pathway, given the cross-sectional design of this study. Since BMI ≥ 30 kg/m² was a necessary condition for MetS, we performed a subgroup analysis of the participants with obese BMI. All analyses were performed using STATA version 10.1.

Results

There were 4,015 PsA patients and 25,976 RA patients enrolled in CORRONA during the study period. There were 3,132 PsA patients (78% PsA) and 18,778 RA patients (72% RA) for whom BMI, hypertension and diabetes data were available. There were 294 patients with PsA (9.4% PsA) and 1,662 patients with RA (8.9% RA) who had complete information about MetS components.

Metabolic Syndrome Analysis

Baseline characteristics of the 294 PsA and 1662 RA participants who had all of the components of the MetS measured are reported in Table 1. The majority of the patients were White in both groups, but there were lower percentages of Black and Hispanic patients in the PsA group. PsA participants were younger, included more men, were more frequently prescribed TNF- α inhibitors, were less likely to be on prednisone, and had lower disease activity, as represented by several disease activity measures. PsA and RA patients were similar in regards to mean disease duration.

Cardiovascular risk factors comparisons

The prevalence of CV risk factors is reported in Table 1. PsA patients were less likely to be current tobacco users (9% vs. 14%), $p = 0.02$, but more likely to use alcohol (57% vs. 51%), $p = 0.02$, compared to RA patients. The mean BMI of the PsA group was higher than in RA, 30.6 ± 6.8 vs. 29.3 ± 6.9 , $p = 0.004$. In addition, PsA patients had significantly higher rates of an obese BMI compared with RA, 45% vs. 39%. The frequency of previous CV events was less than 10% in both groups. There were no differences with respect to the history of myocardial infarcts, strokes, or coronary artery disease between PsA and RA groups.

Prevalence of Metabolic Syndrome and its components

Overall, the prevalence of the MetS was significantly higher in PsA group compared to the RA group: 27% of PsA and 19% of RA patients had MetS, OR 1.44 (95% CI 1.05 to 1.96), $p = 0.02$, adjusted for age, sex, and race (Table 2). In regards to the individual components of the MetS definition, higher frequency of hypertriglyceridemia (38% vs. 28%) was observed in PsA patients compared to RA patients, with an OR 1.51 (95% CI 1.15 to 1.98), $p = 0.003$, adjusted for age, sex, and race. PsA patients were also more likely to have type II diabetes than RA patients (15% vs. 11%), OR 1.56 (95% CI 1.07 to 2.28), $p = 0.02$, in the adjusted model. PsA patients were more likely to be obese, BMI ≥ 30 kg/m² (45% vs. 39%), however the difference was not statistically significant after adjusting for age, sex, and race. No difference was found in the frequency of hypertension or low HDL between the two groups.

The lipid parameters of the PsA and RA patients in the study are compared in Table 3, adjusted for BMI, age, sex and race. There was no difference observed in the prevalence of

high TC, low HDL, high LDL, and TC/HDL ratio between the two groups. However, a greater percentage of PsA patients had hypertriglyceridemia, OR 1.51 (95% CI 1.14 to 1.98), $p=0.003$. Furthermore, patients with PsA had significantly higher mean TG levels (139.28 ± 69.9 mg/dL vs. 128.06 ± 72.1 mg/dL, $p=0.01$) and lower mean HDL levels (50.8 ± 17.0 mg/dL vs. 56.3 ± 18.7 mg/dL, $p<0.001$) than RA patients, respectively. No difference in the percentage of patients using a statin was observed between the two groups.

Obese subgroup analysis

As obese BMI was a necessary condition for MetS, we analyzed the subgroup of 133 PsA and 457 RA with BMI ≥ 30 kg/m² who had lipids measured (Table 4), adjusting for age, sex, and race. Fifty-nine percent of obese PsA patients had MetS compared with 49% of obese RA patients. The OR of MetS in obese PsA compared to RA adjusted for BMI as a continuous measure was 1.46 (95% CI 0.98 to 2.20), $p=0.06$. In addition, after adjusting for age, gender, race, smoking and alcohol status, education, statin use and family history of heart disease the OR became statistically significant, OR 1.57 (95% CI 1.03 to 2.37, $p=0.03$). Hypertriglyceridemia was more prevalent in the obese PsA patients than in obese RA patients, (51% vs. 39%, $p=0.02$), with OR of 1.63 (95% CI 1.09 to 2.42), $p=0.02$. Diabetes was also more prevalent in the obese PsA patients (25% vs. 18%) with OR 1.62 (95% CI 1.01 to 2.59), $p=0.05$. No difference was observed between the frequency of low HDL or hypertension in obese PsA and RA patients.

Sensitivity analyses

We explored whether there was a selection bias in PsA and RA groups with respect to checking lipids, as lipid measurements are not mandated in the registry. We compared the 1956 CORRONA participants with lipid data and 19,733 CORRONA participants who never had lipid values reported. The comparisons for BMI, hypertension, diabetes, age and gender between participants with lipids measured and participants without lipids measured are reported in Table 5. Participants with lipids measured were slightly older, and had slightly higher rates of diabetes and hypertension.

We then compared lipid screening patterns among PsA patients and among RA patients. BMIs were similar in PsA patients with and without lipid measurements, and in RA patients with and without lipid measurements. In addition, in both PsA and RA groups people who had lipids tested were more likely to be slightly older, more educated, less likely to smoke, more likely to have co-morbidities such as diabetes and hypertension, slightly longer disease duration, slightly lower disease activity measures, and more likely to be on statins. There was some differential selection between PsA and RA lipid groups with respect to female gender and alcohol use. Therefore, we constructed an additional model adjusting for age, gender, race, smoking, alcohol use, education, statin use, and family history of heart disease. We found that the higher odds of MetS in PsA remained statistically significant in this adjusted model: OR 1.4 (95% CI 1.05 to 1.99), $p=0.02$.

We next explored whether our results from the subset of CORRONA patients who had lipid data available were generalizable to the larger CORRONA sub-group of 3,132 PsA and 18,778 RA patients for whom hypertension, diabetes and BMI data were available. We found that in this larger CORRONA subgroup, the prevalence of obese BMI was significantly higher in PsA compared with RA, 48% and 37% respectively, OR 1.65 (95% CI 1.52 to 1.80) $p<0.001$. The prevalence of diabetes was also higher in PsA compared with RA, 12% and 8% respectively, OR 1.72 (95% CI 1.51 to 1.97), $p<0.001$. The prevalence of hypertension was 32% in both PsA and RA groups, however, PsA patients were 1.5 times more likely (95% CI 1.36 to 1.65), $p<0.001$ to have hypertension after adjusting for age, sex

and race. These results suggested that in the overall CORRONA population, PsA was associated with the higher rates of cardiovascular risk factors compared with RA.

We also estimated the percentages and the odds of MetS using modified WHO and modified ATPIII criteria. The odds ratios were very similar to the odds ratios obtained by using the modified IDF criteria, OR 1.79 (95% CI 1.17 to 2.74), 0.02, and OR 1.44 (95% CI 1.07 to 1.95), $p=0.01$), respectively.

Finally, we explored whether medication use, prior cardiovascular history, family history of cardiovascular disease, and smoking may confound the association of MetS and PsA. We conducted additional analyses adjusting our models for the following variables: statin use, smoking, family history of cardiovascular disease, and prior history of cardiovascular disease. Adjusting for these variables did not significantly alter odds ratios, p -values, or study conclusions for any of the outcome variables (data not shown).

Discussion

Our results show that MetS was more prevalent in PsA compared with RA in the CORRONA cohort, even after adjustments for age, sex, race and BMI. Overall, individuals with PsA had higher rates of obesity and diabetes compared with RA. PsA patients who had lipids measured showed a higher frequency for the presence of hypertriglyceridemia and diabetes compared to RA patients. Thus, our study contributes to the growing body of literature highlighting an association of PsA with MetS and obesity (44, 45). However, we also observed a higher prevalence of hypertriglyceridemia and diabetes in the subgroup of obese PsA compared to obese RA CORRONA patients, suggesting that hypertriglyceridemia was associated with PsA independent of BMI.

Increased cardiovascular morbidity and mortality have been observed in both PsO and RA, but data on CV disease and risk factors in PsA are limited. A recent study by Husted et al demonstrated that the prevalence of CVD risks was significantly greater in PsA patients than in PsO (28), although the evidence is controversial (32). In our study, we have found that the prevalence of MetS is higher in PsA than RA patients. Therefore, CV risk in PsA cannot be simply added to or extrapolated from PsO or RA. The combination of skin and joint inflammation in PsA may be associated with elevations of inflammatory cytokines such as TNF- α and Interleukin 6 (IL-6) (46), and consequentially increase the frequency of MetS in PsA. Moreover, it is possible that the sometimes insidious onset of PsA may extend the period of unchecked inflammatory burden in patients with PsA, thereby putting them at greater risk for MetS and CV disease.

Similar to our study, two previous studies showed that PsA was associated with a high prevalence of MetS and hypertriglyceridemia (30, 33). However, a recent systematic review by Jamnitski et al. concluded that the CV risks were comparable in PsA and RA (47).

We would like to acknowledge several limitations of our study. Since the PsA and RA diagnoses in CORRONA are rheumatologist - confirmed (either clinically or using the established criteria), there is a possibility of misclassification of RA and PsA patients in our study. However, since the diagnoses of RA and PsA are based on the expertise of rheumatology subspecialty physician, disease misclassification is less likely compared to other diagnostic criteria applied in epidemiologic studies (e.g. health insurance billing codes, patient-reported diagnoses). While this may limit the ability to generalize our findings to PsA and RA groups with criteria-proven disease, our study population reflects the “real world” experience and our results suggest that physicians should consider screening for MetS and its components in both PsA and RA.

Due to the cross-sectional design, we were unable to evaluate causality and the relationship between medications, disease activity and MetS. Since the majority of patients were White, our findings may not be generalizable to other racial/ethnic groups. The definition of metabolic syndrome (MetS) is controversial and has been defined differently according to several societies (39). Because IDF definition includes the measurement of waist-circumference, more individuals are captured via the IDF definition, leading to an increase in prevalence of MetS compared with other definitions (39). Indeed, there are differences in MetS prevalence estimates due to the varying components of the MetS emphasized by the different criteria, whether it be insulin resistance, as with the World Health Organization (WHO) criteria, or obesity, as with the IDF criteria (39, 48). Despite of the above difference, these definitions likely encapsulate the same clinical syndrome (39, 49). Furthermore, it has been shown that each definition is comparable in terms of their ability to predict cardiovascular disease risk and events in the general population (50, 51). It has not been studied whether these definitions accurately predict cardiovascular disease in individuals with inflammatory arthropathies. However, we obtained similar results irrespective of the definitions used further adding to the validity and robustness of our findings and conclusions.

One of the main limitations of our study is that waist circumference, BP measurements or glucose intolerance data were unavailable in the CORRONA registry. Therefore, the prevalence of MetS may have been underestimated in our study for both RA and PsA. For example, according to the IDF definition, central obesity (the necessary condition for the presence of MetS) is defined as either BMI>30kg/m² or abnormal waist circumference. Since some people with normal BMI may have abnormal waist circumference, excluding these individuals in our MetS definition may underestimate the prevalence of MetS in both PsA and RA (39). Based on the results of the study by Mok et al. individuals with PsA have higher rates of abnormal waist circumference and insulin resistance compared to RA (33). Therefore, we may be underestimating the odds of MetS in PsA compared with RA in our study. However, we included the MetS measures that are readily available to rheumatologists in clinical practice. Therefore, our findings may alert rheumatologists to initiate further screening for the presence of MetS in PsA and RA.

Finally, since only a fraction of patients in each group had lipids measured, there is a possibility of selection bias, despite adjusting for potential confounders. However, while there was some selection bias with respect to lipid testing, we performed several sensitivity analyses and demonstrated that our conclusions and findings were robust and generalizable to the larger CORRONA population.

In conclusion, our study is the first to compare the prevalence of abnormal lipids, obesity and diabetes, as well as clustering of these factors in PsA and RA, in a large US cohort. Although individual components of the MetS have been reported in PsA (9, 26, 27, 29, 52), currently there is limited data regarding the prevalence of MetS in PsA, and how it compares to RA. Because MetS confers a risk that is higher than the sum of its individual components, it is important to study in the PsA population. Although both diseases have underlying inflammatory mechanisms, the differences in the prevalence of MetS, triglycerides, and diabetes between PsA and RA suggest that PsA may differ from RA in terms of disease mechanisms, cytokine profile, and lipid metabolism. Further studies are needed to shed light on these differences. Most importantly, our findings highlight the importance of aggressive screening of MetS and modifiable CV risk factors in patients with both RA and PsA. Further studies are needed to determine if controlling for these risk factors reduces cardiovascular morbidity and mortality in both PsA and RA.

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Significance and Innovation

- To date, there are limited data regarding the prevalence of metabolic syndrome and its components in psoriatic arthritis, and in comparison with rheumatoid arthritis.
- In our study, psoriatic arthritis was associated with significantly higher rates of hypertriglyceridemia, obese body mass index, and diabetes.
- The overall prevalence of metabolic syndrome and its components in psoriatic arthritis was higher than in rheumatoid arthritis.
- The implications of these associations for cardiovascular risk and disease mechanisms of disease in psoriatic arthritis need to be explored further.

Table 1

Bivariate comparisons of baseline characteristics between PsA and RA

	PsA (n = 294)	RA (n = 1662)	p-value
Age, years (mean $\pm$ SD)	55.7 $\pm$ 11.9	61.6 $\pm$ 12.2	< 0.001
Female gender, N (%)	135 (46)	1279 (77)	<0.001
Race/ethnicity, N(%)			
White	269 (92)	1323 (80)	
Black	5 (2)	169 (10)	< 0.001
Hispanic	10 (3)	100 (6)	
DISEASE ACTIVITY MEASURES			
Disease duration, years (mean $\pm$ SD)	11.1 $\pm$ 10.44	12.0 $\pm$ 10.3	0.17
CDAI (mean $\pm$ SD)	7.7 $\pm$ 8.2	9.9 $\pm$ 10.2	0.001
Enthesitis or Sausage Digits, N (%)	71 (24)	0 (0)	N/A
MEDICATIONS			
Prednisone, N (%)	25 (9)	453 (27)	< 0.001
Methotrexate, N (%)	130 (44)	1091 (66)	< 0.001
Any non-Biologic DMARD, N (%)	152 (52)	1377 (83)	< 0.001
Anti-TNF- α use, N (%)	171 (58)	598 (36)	< 0.001
CARDIOVASCULAR RISK FACTORS			
Smoker, N (%)	25 (9)	216 (14)	0.02
Alcohol, N (%)	167(58) (58.2%)	799 (51)	0.02
Family History of Heart Disease, N(%) (%)	152 (52)	872 (53)	0.02
BMI, kg/m ² (mean $\pm$ SD)	30.6 $\pm$ 6.8	29.3 $\pm$ 6.9	0.004
BMI categories, N(%)			
Normal ($18.5 \leq BMI < 25$)	56 (19)	454 (28)	
Overweight ($25 \leq BMI < 30$)	104 (36)	534 (36)	0.007
Obese ($BMI \geq 30$)	133 (45)	654 (39)	
History of Cardiovascular Disease	27 (9)	171 (10)	0.60
History of Myocardial Infarction	7 (2)	59 (4)	0.38
History of Coronary Artery Disease	4 (1)	46 (3)	0.23

CDAI = clinical disease activity index; DMARD = disease-modifying antirheumatic drug; TNF- α = tumor-necrosis factor alpha; BMI = body mass index.

Table 2

Odds ratios for MetS and its components among PsA and RA patients in the CORRONA registry, adjusted for age^{*}, gender, and race.

	PsA (n = 294)	RA (n = 1662)	OR (95% CI)	p-value
Metabolic Syndrome, %	27	19	1.44 (1.05–1.96)	0.02
Obesity, BMI ≥ 30 kg/m ² , %	45	39	1.19 (0.90–1.57)	0.22
Hypertriglyceridemia, % TG >150 mg/dL	38	28	1.51 (1.14–1.98)	0.003
Diabetes, %	15	11	1.56 (1.07–2.28)	0.02
Hypertension, %	36	40	1.09 (0.81–1.47)	0.56
Low HDL, % < 40 mg/dL for males, < 50 mg/dL for females	36	33	1.00 (0.75–1.32)	0.98

OR = odds ratio; 95% CI = 95% confidence interval; BMI = body mass index; HDL = high-density lipoprotein.

*
in 10 year units

Table 3

Odds ratios for lipid parameters in PsA compared with RA patients in the CORRONA registry, adjusted for BMI, age^{*}, gender, and race.

	PsA (n = 294)	RA (n = 1662)	OR (95% CI)	p-value
TC > 200 mg/dL, %	35	37	1.09 (0.82–1.45)	0.56
Low HDL, % < 40 mg/dL for males, < 50 mg/dL for females	36	33	1.25 (0.94–0.71)	0.67
LDL > 100 mg/dL, %	60	57	1.10 (0.84–1.45)	0.48
TG > 150 mg/dL, %	38	28	1.51 (1.14–1.98)	0.003
TC/HDL > 3.5, %	60	45	1.23 (0.92–1.64)	0.15
Statin Use, %	26	23	1.29 (0.93–1.78)	0.13

OR = odds ratio; BMI = body mass index; 95% CI = 95% confidence interval; TC = total cholesterol; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglycerides

*
in 10 year units

Table 4

Odds ratios for MetS and its components in obese PsA and obese RA patients in the CORRONA registry, adjusted for age*, sex, and race.

	Obese PsA (n = 133)	Obese RA (n = 654)	OR (95% CI)	p-value
Metabolic Syndrome, %	59	49	1.46 (0.98–2.2)	0.06
Hypertriglyceridemia, % TG >150 mg/dL	51	39	1.63 (1.09–2.42)	0.02
Diabetes, %	25	18	1.62 (1.01–2.59)	0.05
Low HDL, % < 40 mg/dL for males, < 50 mg/dL for females	49	44	0.97 (0.65–1.44)	0.87
Hypertension, %	48	52	0.96 (0.62–1.48)	0.85

OR = odds ratio; 95% CI = 95% confidence interval; TG = triglycerides; HDL = high-density lipoprotein

*
in 10 year units

Table 5

Baseline characteristics of patients with and without lipids available in the CORRONA registry

	Without lipid values (n=19,733)	With lipid values (n=1956)	p-value
Age	59.16 ± 13.9	61.21 ± 12.3	<0.001
Female (%)	14,378 (73.3)	1414/1954 (72.4)	0.38
BMI kg/m ²	29.36±7.2	29.58±6.9	0.20
History of Diabetes (%)	1645 (8.3)	233 (11.9)	<0.001
History of Hypertension (%)	6018 (30.5)	786(40.2)	<0.001