time ($F(4,141) = 0.712, p = 0.585$). Figure 1 shows the estimated trajectories of DAS28-ESR scores over 12 months time for the three health literacy groups. Patients with “several health literacy limitations” were prescribed prednisolone significantly more often (52.4%) than patients with “some health literacy limitations” (21.2%) or “good health literacy” (22.2%) over time ($p = 0.019$). Patients with “some health literacy limitations” were prescribed conventional DMARDs more often (72.7%) than patients with “good health literacy” (38.9%; $p = 0.008$). There was no difference in biological DMARDs use between the health literacy groups.

Figure 1. Mean (standard error) DAS28-ESR scores of health literacy (HL) groups over a 12-month time period.

Conclusion: We found that among patients with RA, those with several health literacy limitations have higher disease activity scores over time and use prednisolone significantly more often than patients with higher health literacy levels.

No difference was observed in biological DMARD use. These results grant more nisolone significantly more often than patients with higher health literacy levels.

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drome and anti-phospholipid syndrome


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None declared

Disclosure of Interests:

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Systemic lupus erythematosus, Sjogren’s syndrome and anti-phospholipid syndrome

POS0114

COMPUTATIONAL IDENTIFICATION OF SLE PATIENT RECORDS USING DATA-DRIVEN CLINICAL FINGERPRINTS

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Background: Systemic lupus erythematosus (SLE) is a chronic relapsing auto-immune disorder that is challenging to diagnose due to its heterogeneous presentation of a combination of non-specific clinical manifestations and laboratory findings. This heterogeneity and lack of granularity is a major cause of treatment failure. With the advent of precision medicine, there is a need for accurate methods to identify cases of SLE in large clinical databases, and to recognize constellations of pathological characteristics (or fingerprints) that allow a better understanding of the disease. Computational phenotype discovery aims to disentangle the fingerprints of disease that hide within the noisy, sparse and incomplete electronic health record data.

Objectives: To generate data-driven phenotypic fingerprints of SLE and validate them by assessing their ability to distinguish records of patients with SLE vs. ‘near miss’ patients without the disease.

Methods: Records of 716 patients that published, expert-curated algorithms [1] indicated as likely SLE patients were reviewed by 3 clinicians and labeled as positive (490 records), negative (261), or indeterminate (55). Those labeled positive by an algorithm but negative by clinician chart review were considered ‘near misses’ and should be among the more difficult cases to classify. We trained an ElasticNet (regularized logistic regression) model to distinguish the true positive records from the near misses and evaluated it under 10x cross validation. Inputs to the predictive model were the projections of each patient record into the space of 2000 latent variables. These features had been inferred separately by an unsupervised machine-learning algorithm from a much larger dataset of 646,716 randomly sampled temporal cross sections of 63,775 longitudinal records of patients with an antinuclear antibody test. Each of the 2000 variables constitutes the phenotypic fingerprint of an unbiased, independent potential disease mechanism. Formally, they represent linear combinations of demographic data, laboratory test results, billing code intensities, and medication exposures [2].

Results: Our predictive model achieved an area under the Receiver Operating Characteristic Curve of 0.90, 95% CI: [0.879, 0.922], an area under the Precision-Recall Curve of 0.94, 95% CI: [0.926, 0.954], and an Integrated Calibration Index of 0.074, 95% CI: [0.067, 0.080].

The model selected 61 of the 2000 potential latent mechanisms to distinguish positive SLE records from near-misses. All of the phenotypes selected to be predictive exhibit high face-validity from a clinical interpretation perspective. They include recognizable patterns of variables representing different clinical features of SLE (Figure 1).

Figure 1. Source Fingerprint S-1497

SLE plasmocytopenic syndrome (x13.31)
Systemic lupus erythematosus (x1.333)
Kidney disease (x11.149)
DNA double strand Ab (x2.824)
ANA titer (x15.14)
Proteinuria (x1.073)
Chronic plasmocytopenic (x0.537)
Complement C3 (x3.528)
Collagen disease (x0.058)
Systemic lupus erythematosus with... (x0.064)

Systemic lupus erythematosus is a chronic relapsing autoimmune disorder that is challenging to diagnose due to its heterogeneous presentation of a combination of non-specific clinical manifestations and laboratory findings. This heterogeneity and lack of granularity is a major cause of treatment failure. With the advent of precision medicine, there is a need for accurate methods to identify cases of SLE in large clinical databases, and to recognize constellations of pathological characteristics (or fingerprints) that allow a better understanding of the disease. Computational phenotype discovery aims to disentangle the fingerprints of disease that hide within the noisy, sparse and incomplete electronic health record data.

Objectives: To study the effect of serum PCSK9 on major cardiovascular adverse events (MACEs) in Chinese patients with systemic lupus erythematosus (SLE).

Methods: Consecutive patients who fulfilled ≥4 1997 ACR criteria for SLE and consented for a biomarker study between 2009 and 2012 were included. Stored serum samples from these patients were assayed for the levels of PCSK9 using a commercial ELISA kit (OKBB00903, Lot# 1344, Aviva Systems Biology, San Diego, US). New MACEs (acute coronary syndrome, ischemic stroke, peripheral vascular disease) documented by imaging and angiographic studies over time was evaluated. Patients were stratified into high/low PCSK9 groups according to the best cut-off level by ROC analysis for the prediction of these events. The cumulative incidence of new MACEs and mortality over time was studied by Kaplan-Meier’s analysis and compared between the high and low PCSK9 subgroups. Cox regression was performed to study the effect of the PCSK9 subgroups on new MACEs and mortality, adjusted for other confounding factors.

Results: 539 SLE patients were studied (93% women, age 41.9±14.0 years; disease duration 106±90.4 months at entry). The mean PCSK level at baseline was 828 Scientific Abstracts