17.3) years (see Figure 1). Lupus nephritis was diagnosed in 36 of 126 (28.6%) at onset of SLE. The prevalence of SLE on December 31st 2021 was 64.5 per 100 000 inhabitants (87% females); higher in females (110.7 per 100 000) compared to males (17.4 per 100 000). The mean age was 55.9 (SD 16.7) years. Age at diagnosis and disease activity measures (SLEDAI-2K and the Physician's Global Assessment) increased ($p<0.05$) over the time period, but none of the laboratory items changed significantly. Lupus nephritis, as well as involvement of other organ systems (e.g., fulfilled classification criteria), at disease onset did not vary significantly.

Conclusions In Östergötland County, SLE incidence and prevalence estimates were constant during the 14 years of follow-up. Whereas the prevalence of SLE was almost identical to what has previously been reported from Southern Sweden (Ståhl-Hallengren C, et al. J Rheumatol 2000;27:685–91; Ingvarsson RF, et al. Lupus 2016;25:772–80), we obtained slightly lower incidence figures. In addition, our data indicate that SLE is diagnosed also among older individuals with a more even female-to-male ratio. Disease phenotypes observed in patients at onset of SLE were similar over the time period.

PO.7.163 EPIDEMIOLOGY OF SYSTEMIC LUPUS ERYTHEMATOSUS AMONG BLACK AFRICANS LIVING IN AFRICA: A POOLED ANALYSIS OF DATA FROM 896 SUBJECTS

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10.1136/lupus-2022-elm2022.183

Background This was the first systematic review and meta-analysis on the epidemiology of systemic lupus erythematosus (SLE) among Black Africans living in Africa.

Methods We queried PubMed, EMBASE, Web of Science, African Journals Online, and Global Index Medicus to select studies published in the period 01/01/2008–7/10/2018, and addressing SLE among Black Africans living in Africa. Results were pooled through narrative review and random-effects model, and the review protocol was registered with PROSPERO (CRD42019139226).

Results Of 1502 records, we included 15 hospital-based studies. There was no incidence data. The pooled prevalence of SLE in Rheumatology and Internal Medicine departments was 1700 per 100,000 persons (800–2900). The mean age at diagnosis ranged from 28.8 to 39.2 years, and the female proportion from 88% to 100%. The commonest SLE features were rheumatological (5.1%-99.9%), mucocutaneous (4.3%-100%) and hematological (1.4–86.9%). Patients had a high seroprevalence for anti-ribonucleoprotein 57.9% (36.4–77.9), anti-Smith 53.5% (40.4–66.2), anti-Sjogren syndrome antigen A 45.6% (19.2–73.4) and anti-Sjogren syndrome antigen B 33.7% (13.6–57.6) autoantibodies. Mean SLEDAI score (from one study) was 9.8±8.6. There was no measure of damage accrual. The pooled mortality rate was 10.3% (3.3–20.6), and main death causes were infections, kidney and central nervous system involvement.

Conclusions Over the last three decades, the epidemiology of SLE among Black Africans living in Africa shared many similarities with data from Black Africans living in the diaspora.

Acknowledgements None.

Friday 07 October 2022 from 13:00 to 14:10

PO.8 E- poster session 8: skin manifestations, SLE and infections, fertility and pregnancy, imaging

PO.8.164 MELANODERMA INDUCED BY LONG-TERM USE OF HYDOXYCHLOROQUINE IN SLE

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10.1136/lupus-2022-elm2022.184

Introduction Hydroxychloroquine (HC) remains a standard treatment in many systemic diseases, including systemic lupus erythematosus (SLE), rheumatoid arthritis and many others, but these multiple side effects are often overlooked. The best known of its adverse effects are retinitis pigmentosa, digestive disorders and disturbances in liver function tests, unlike the mucocutaneous effects.

Objective To draw attention to melanoderma as a mucocutaneous side effect of long -term use of HC.

Observation We report the case 55 years old woman witch was diagnosed and monitored for SLE 15 yeras ago. She is currently being treated with HC 400mg/d with prednisone at 10mg/d. This patiente was in prolonged remission from her disease and declares that she is satisfied with her treatment until diffuse melanodermal lesions appear on her body, bothersome and above all worrying the patient. These melanodermal spots are located on the upper and lower extremities, abdomen and oral cavity.

Discussion After ruling out all of the other causes of melanoderma, in particular slow adrenal insufficiency and paraneoplastic syndrome, and considering the long-term intake of HC likely to cause such a side effect, we confirmed the iatrogenic causality link. The patiente was informed and the causal drug was stopped with narrow monitoring of the lupus disease. The prolonged duration of drug exposure could ensure a sufficient cumulative dose allowing for a therapeutic window. The reintroduction of HC was estimated possible after total disappearance of the melanoderma. However, this must be gradual and as late as possible.

Conclusion In addition to the known side effects of HC, melanoderma is not uncommon and must be taken into consideration without disturbing the management of the disease treated by this molecule recognized by antiinflammatory, immunomodulatory and antithrombogenic actions.

PO.8.165 WHOLE-BLOOD DNA METHYLATION ANALYSIS REVEALS RESPIRATORY ENVIRONMENTAL TRAITS INVOLVED IN COVID-19 SEVERITY FOLLOWING SARS-COV-2 INFECTION

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10.1136/lupus-2022-elm2022.185

Purpose SARS-CoV-2 causes a severe inflammatory syndrome (COVID-19) leading, in many cases, to bilateral pneumonia, severe dyspnea and in ~5% of these, death. DNA methylation is known to play an important role in the regulation of the immune processes behind COVID-19 progression, however it has not been studied in depth. In this study, we aim to evaluate the implication of DNA methylation in COVID-19 progression by means of a genome-wide DNA methylation analysis combined with DNA genotyping.

Methods We integrated genetic and blood DNA methylation information, analysed them together with clinical information in a sample of ~100 healthy controls and ~470 SARS-CoV-2 PCR tested patients recruited from two different clinical centers and compared with seven different systemic autoimmune diseases (SADs) from the PRECISESADS cohort.

Results The EWAS revealed widespread epigenetic variation associated with COVID-19 phenotypes, which were enriched in molecular pathways related with SADs and differed between mild and severe COVID19 cases. Among them, interferon, FCGR mediated phagocytosis and CD209 signatures in severe and mild COVID-19 were shared with seven different SADs (including systemic lupus erythematosus). Environmental trait-related CpG sites were found to be specifically hypermethylated in mild SARS-CoV-2 positive cases. These sites were enriched in key regulators of inflammatory cytokine gene expression known to be part of the cytokine storm described in the most severe outcomes of the disease. Additionally, these DNA methylation changes were found to be differentially regulated by genetic variants and associated with different regulatory mechanisms.

Conclusions The results reveal the existence of epigenomic regulation of functional pathways associated with COVID-19 progression and mediated by genetic loci. Our work reveals pathways involved in COVID19 pathogenesis both shared and not shared with SADs, novel risk variants with epigenetic downstream effects, and illustrate how the genetic architecture of DNA methylation depends on the infection status and severity of COVID19. In addition, the analyses suggest that an interaction between environment, genetics and epigenetics might be playing a role in triggering the cytokine storm described in the most severe cases.

PO.8.167 INTERCURRENT INFECTION AS A RISK FACTOR FOR DISEASE FLARES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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10.1136/lupus-2022-elm2022.186

Background Intercurrent infections are presumed potential triggers of systemic lupus erythematosus (SLE) disease flares.[1] However, most of the evidence is obtained from a limited number of observational studies, and the results of these studies are conflicting.[2,3]

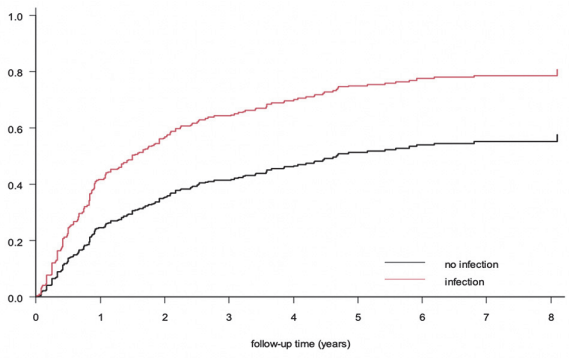
Purpose To determine whether intercurrent infections are a risk factor for disease flares in SLE.

Methods Demographic and clinical characteristics of 203 SLE patients from the Amsterdam SLE cohort were collected at baseline, and at the start of an intercurrent infection that either was or was not followed by a flare within three months. Relevant data regarding infections and flares were collected systematically by chart review. Major and minor infections were defined as, respectively: infections for which hospital admission or intravenous antibiotic therapy was required, and infections (proven or not proven, but highly likely based on clinical symptoms and/or response to therapy) for which hospital admission was not warranted. SLE flares were defined as an increase in disease activity requiring intensification of immunosuppressive therapy. Flares were categorized as major or minor depending on fulfillment of a predefined set of criteria.[4] Incidence rates for infections, flares, and infections followed by a flare within three months were calculated using Poisson regression. Descriptive analyses were performed where appropriate. Proportional hazard models with recurrent events and time-varying covariates were used to estimate the hazard ratio of SLE flares.

Results Table 1 shows the demographic and clinical characteristics of all 203 SLE patients. Fifty-six major infections occurred in 1060 patient years, and 670 minor infections occurred in 1048 patient years. The incidence rates of major and minor infections were 5.3 per 100 patient years (95% CI: 4.1–6.9) and 63.9 per 100 patient years (95% CI: 59.3–69.0), respectively. In total, 198 flares occurred within 1060 patient years. The incidence rate of flares is 18.7 per 100 patient years (95% CI: 16.3–21.5), 3.6 per 100 patient years (95% CI: 2.6–4.9) for major flares and 15.1 per 100 patient years (95% CI: 12.9–17.6) for minor flares.

Abstract PO.8.167 Table 1 Baseline demographic and clinical characteristics

Variables	SLE patients (n = 203)
Sex, Female, n (%)	184 (91)
Age, years (median (IQR))	40.0 (32.0 – 47.0)
Caucasian ethnicity, n (%)	137 (68)
SLICC/ACR damage index (median (IQR))	1 (0 – 2)
SELENA-SLEDAI2k (median (IQR))	4 (2 – 6)
Disease duration, years (median (IQR))	6 (1 – 11)
History of:	
Biopsy proven lupus nephritis, n (%)	38 (19)
Renal insufficiency (eGFR < 45), n (%)	6 (3)
Diabetes mellitus, n (%)	7 (3)
Malignancy, n (%)	5 (3)
Stroke, n (%)	12 (6)
Asplenia, n (%)	5 (3)
Treatment variables:	
Glucocorticoids, n (%)	106 (52)
Antimalarials, n (%)	151 (74)
Immunosuppressants, n (%)	75 (37)
NSAIDs, n (%)	67 (33)



Abstract PO.8.167 Figure 1 Estimated cumulative incidence of SLE flares (major and minor) following an intercurrent infection within three months