Review article

Lupus and other autoimmune diseases: Epidemiology in the population of African ancestry and diagnostic and management challenges in Africa

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Autoimmune diseases are prevalent among people of African ancestry living outside Africa. However, the burden of autoimmune diseases in Africa is not well understood. This article provides a global overview of the current burden of autoimmune diseases in individuals of African descent. It also discusses the major factors contributing to autoimmune diseases in this population group, as well as the challenges involved in diagnosing and managing autoimmune diseases in Africa. (J Allergy Clin Immunol Global 2024;3:100288.)

Key words: *Lupus, autoimmune diseases, epidemiology, challenges, Africa*

Abbreviations used
AD: Autoimmune disease
ADMT: Autoimmune disease–modifying therapy
ANA: Antinuclear antibody
DEI: Diversity, equity, and inclusion
ID: Infectious disease
SAD: Systemic autoimmune disease

Autoimmune diseases (ADs) are chronic diseases resulting primarily from the breakdown of tolerance against self-antigens.[1,2] Besides the classic autoantibody-positive and seronegative AD subtypes,[1,3,4] there are "borderline ADs" that exhibit both autoimmune (eg, autoantibodies in a subset of patients) and autoinflammatory (eg, excess pattern recognition receptor signaling) features.[4,5] Examples of "borderline ADs" include sarcoidosis, inflammatory bowel disease, and psoriatic disease.[6-8] Most ADs are treated with the same types of AD-modifying therapies (ADMTs) because they arise from a combination of similar patient-related and environmental factors driving similar pathogenetic and clinical features.[5,9] This phenomenon is referred to as the "autoimmune tautology."[9] However, some ADs are mendelian disorders.[5]

The estimated prevalence of ADs outside Africa is 5% to 10%, with people of African ancestry being particularly affected.[10-12] From 1965 (when ADs were officially recognized) to the 2000s, they were rarely reported in Africa.[13-16] Given the high burden of infectious diseases (IDs) in Africa,[17] the "hygiene" or "old friend" hypothesis, which suggests an inverse association between IDs and ADs,[18,19] was used to justify the scarcity of AD documentation in Africa. The scarcity of lupus (a prototypical AD) reports from West Africa in the 20th century and the high frequency of lupus in West African immigrants in America and Europe gave rise to the "lupus gradient" hypothesis.[20-22] This hypothesis suggests that lupus is rare in West Africa owing to the high prevalence of malaria, which is the most common ID.[20,23-25] However, infectious agents have been identified as risk factors for ADs.[5,26] Current lupus data from Nigeria[27] are inconsistent with data from the 20th century.[21] Additionally, a universal definition of a "rare disease" is still awaited.[28,29]

In this article, we have narratively synthesized up-to-date epidemiologic data on lupus and other ADs in people of African ancestry. We also discuss potential determinants of the high burden of ADs in this population group and the challenges faced when it comes to managing ADs in Africa.

EPIDEMIOLOGY

Frequency

The combined frequency of organ-specific lupus and systemic lupus erythematosus (SLE) is unknown. The frequency of diagnosed SLE is highest in the United States (US) and Barbados (the Caribbean).[30] The US Centers for Disease Control and Prevention estimated the prevalence of diagnosed SLE in the US at 72.8 of 100,000 in the general population, 230.9 of 100,000 in females of African ancestry, and 26.7 of 100,000 in males of African ancestry (vs 14.6 for all US males).[31] In Barbados, the incidence of diagnosed SLE is 10.37 per 100,000 person years.[30] Among low- and middle-income countries, the World Health Organization-Africa region had the second-highest (after American countries) standardized prevalence (60 per 100,000 of individuals with diagnosed SLE [95% CI = 40-1300]).[32] Population-based prevalence data on SLE in Sub-Saharan Africa

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are scarce,[32] but SLE was diagnosed in 1.7% of 28,175 natives in Sub-Saharan African internal medicine and rheumatology settings between 1987 and 2014 (95% CI = 0.8-2.9).[15] It is also estimated that 9% of the global population of individuals who are younger than 16 years and have SLE reside in Eastern Africa.[33]

Scleroderma, type 1 diabetes mellitus, and sarcoidosis are more commonly diagnosed in individuals of African descent than in individuals of other ancestries.[34-36] However, the frequencies of these conditions in Africa are not well known.[37-39] Between 2004 and 2012, dermatomyositis and polymyositis were diagnosed in US Medicaid beneficiaries of African ancestry more frequently than in White people.[40] In Africa,[41] the frequency of diagnosed autoimmune inflammatory myopathy mirrors the frequency reported elsewhere.[42] The incidence rates of dermatomyositis per 1,000,000 person years were approximately 7.5 and 1.2 in South Africa[43] and Botswana,[44] respectively. The incidence of polymyositis was 8.8 per 1,000,000 person years in Lybia,[45] and its prevalence was 11 per 100,000 persons in Egypt (95% CI = 0-32).[46] Data from Southern California in the US suggest that multiple sclerosis could be more prevalent among young people of African ancestry than among young people of other ancestries.[47] In Africa, the prevalence of diagnosed multiple sclerosis saw a 59% increase from 2013 to 2020, with the latest prevalence rate at 8.76 per 100,000 individuals.[48]

Further research is needed to determine across age groups the magnitude of other ADs in individuals of African descent versus in individuals of other ancestries.[15,33,49-60]

Phenotype, severity and case fatality rate

In general, individuals of African ancestry tend to develop ADs earlier in life[15,16,37,41,61-63]; in addition, ADs have a higher female predilection[16,54,64] and are characterized by more symptoms in individuals of African ancestry[15,16,65] than in those of other ancestries.

Patients with SLE who are of African descent are more likely to develop lupus nephritis (the most severe manifestation of lupus), have higher disease activity scores, and experience more organ damage accrual than White patients do.[65-69] It has been estimated that people of African ancestry have a higher frequency of biomarkers indicating severe SLE than people of other ancestries do.[70,71] For example, the frequency of checked anti-Smith autoantibodies in Sub-Saharan Africans with SLE was found to be 53.5% (95% CI = 40.4%-66.2%),[72] whereas the frequency of anti-Smith autoantibodies in the general population of people with SLE varies between 5% and 30%.[73]

Survival has improved markedly in patients with SLE who are living in developed countries.[74,75] However, US Medicaid beneficiaries of African ancestry who had SLE had a 21% higher risk of death than did White people in 2000-2006 (hazard ratio = 1.21 [95% CI = 1.10-1.33),[76] and SLE ranked fifth and sixth among causes of death in women of African descent aged 15 to 24 and 25 to 34 years compared with 10th and 14th in age-matched White American women.[77] In Sub-Saharan Africa, SLE inpatient case fatality rate reaches as high as 43.1% (95% CI = 29.3%-57.8%), with death often occurring shortly after the diagnosis.[72] IDs are the main causes of death.[15,76,78] However, kidney disease is also a major cause of death in Sub-Saharan Africa.[15,79] More robust studies are needed to clearly determine the burden of juvenile-onset SLE in individuals of African ancestry versus in individuals of other ancestries.[80-83]

Individuals of African descent who have various ADs tend to have worse disease activity and outcomes than patients of other ancestries do. Examples of these ADs include, but are not limited to, type 1 diabetes mellitus, multiple sclerosis, autoimmune hepatitis, myasthenia gravis, autoimmune inflammatory myopathy, rheumatoid arthritis, and scleroderma.[38,40,41,47,49,52,64,84-86] Scleroderma and SLE are 2 of the deadliest systemic ADs (SADs),[87] with the highest case fatality rates found in individuals of African descent.[76,85] In the US, the age-adjusted case fatality rate of scleroderma in people of African ancestry is 1.5 times higher than that in White individuals.[85] In a study in South Africa, a 9% case fatality rate was reported among individuals with scleroderma who were receiving medical care.[88] These high death rates are due mainly to scleroderma-associated interstitial lung disease.[37,85,88]

Determinants

There are cross-sectional studies linking ADs with sociologic and genetic factors in people of African ancestry. However, other environmental factors may also play a crucial role (Fig 1).[5,89-126]

Sociologic determinants. Sociologic determinants refer mainly to psychosocial stressors that can increase the likelihood and severity of ADs.[92-95] They do this by affecting the body, increasing environmental exposures, and limiting access to optimal care.[92,107-109,127,128] An important bodily consequence of these factors is the change in a person's genetic makeup, resulting in the development of "social epigenetic factors."[127] Additionally, these factors stimulate brain regions responsible for threat vigilance and emotions such as the amygdala, and the hypothalamic-pituitary-adrenal axis, leading to the sustained production of stress hormones and proinflammatory cytokines.[107-109,128-131]

Low socioeconomic status. The socioeconomic status of an individual refers to his or her relative position in the hierarchic structure of society based on access to control over wealth, prestige, and power.[92] The socioeconomic stratification of individuals has substantial effects on their self-perception and well-being.[92,129,132,133] Socioeconomic stratification also affects mating behavior, which in turn affects the genetic diversity of populations.[134]

Low socioeconomic status is a psychosocial stressor that can lead to various diseases, including ADs, given that "health" encompasses physical, mental, and social well-being.[92,107-109,132,135] Socioeconomic status is determined mainly by income, educational level and quality, occupational class, social class, and ancestry.[92] This entails low socioeconomic status being associated with poverty and low educational level and quality, other psychosocial stressors, and driving factors of low self-health perception and unadvisable habits that have been associated with ADs in people of African descent.[38,52,64,65,92-95,136-138] For instance, in Nigeria, most individuals diagnosed with SLE between 2017 and 2020 had a monthly income below $100 US.[27]

Individuals with low socioeconomic status may be subject to neglect and social abuses such as rape and emotional abuse.[82,111,112,130,139,140] They may also have limited social support and fewer opportunities to take advantage of available resources.[111,112,132,138] This can help explain why patients of African ancestry, who experience multiple adverse life events and insecurities such as food and housing insecurity and low access to health care, are more likely to experience SLE severity and organ damage.[65,79,82,93,111,112,130,139-141]

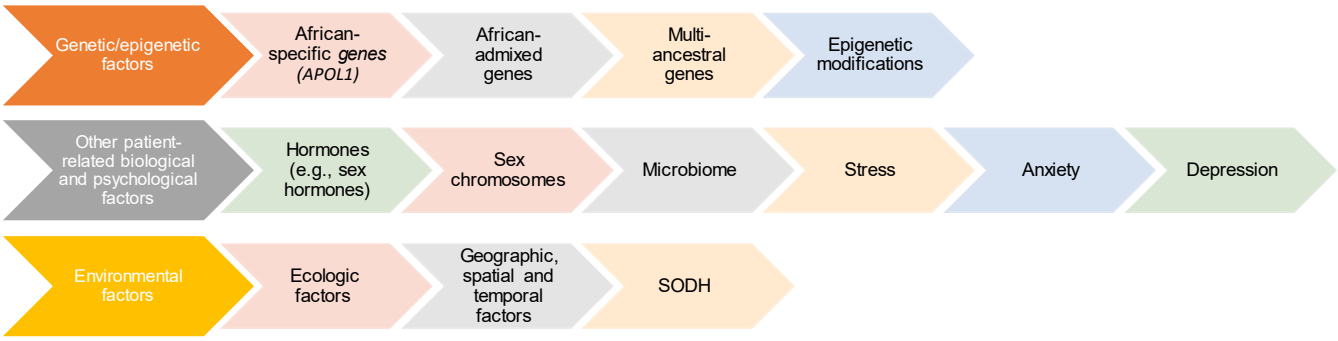

FIG 1. Factors driving susceptibility and severity of multigenic ADs in individuals of African ancestry.[5,89-126] There is a specific gene in people of African descent that is strongly associated with ADs. This gene is called *Apolipoprotein 1* (*APOL1*), and its G1 and G2 variants are linked to lupus nephritis and lupus-related end-stage kidney disease in people of African descent.[89-91] Social determinants of health (SODHs) including low socioeconomic status and Diversity-Equity-Inclusion (DEI)-related matters are the best-known AD determinants in people of African ancestry.[65,66,92-95] Considering data from the general population with ADs, ecologic factors commonly found in neighborhoods of individuals of African ancestry (eg, infectious agents and pollutants) and other lifestyle-related SODHs such as food environments could also be major environmental AD determinants in the population of African ancestry.[5,96-126] Geographic, spatial, and temporal factors, including latitude, longitude, altitude, temperature, precipitation, and humidity, may contribute as well.[119]

Low socioeconomic status is associated with increased susceptibility, severity, and worse outcomes of ADs,[65,92-95,142] as well as with comorbidities[111,112,142] such as infections, depression, and cardiovascular diseases, especially among individuals of African descent with SLE.[65,92,93,142]

Bias in implementation of the principles of DEI. The concept of "diversity, equity, and inclusion" (DEI) was developed in the US in the 1960s to close the socioeconomic gap across ancestral groups (see Beavers D.[143]). An unbiased implementation of DEI principles would have been helpful for people of African ancestry with ADs or predisposing factors had all forms of racial discrimination been eliminated in 1965, as was expected by the United Nations.[93,121,137,139,140,143-146] According to the 2015-2017 BeWell (Black Women's Experiences Living with Lupus) study, perceived racist microaggressions and vicarious racism (unintended exposure to a racist act inflicted on someone else) were associated with SLE disease activity and organ damage, as well as with comorbid obesity.[139,140] Racism is a psychosocial stressor.[128] Its impact on susceptibility and outcomes of ADs could also be understood through the lens of low socioeconomic status, as racism amplifies stigma and common discriminations against people with ADs (eg, sex discrimination, ableism, and job discrimination).[92,93,95,111,112,128,147,148]

Besides interpersonal and structural racism outside the health system, people of African ancestry with ADs are being discriminated against within the health system.[82,93,142,147] The provision of care for SADs is often influenced by rheumatologists, who may or may not have received formal training in internal medicine. This could be due to a reduced interest in autoimmunity among general internal medicine physicians, and there is no formal autoimmunology medical specialty.[149-152] African-specific phenotypes, such as skin and hematologic manifestations, data, and voices, are substantially underrepresented in rheumatology training, research, and management materials and frameworks.[16,153-161] This leads to unequal opportunity for timely diagnosis and management of SADs in people of African descent versus in people of other ancestries.[16,153,154] This inequality also limits the understanding of ADs, as data from individuals of African ancestry may have important educational value.[153,154,162]

Patients of African ancestry have often been underrepresented in studies on the effectiveness of ADMTs.[16,163] This is due partly to inadequate counseling that does not take cultural sensitivities into consideration.[163] It is also observed in Western rheumatology settings during treatment of patients of African ancestry with biologic ADMTs.[164] However, providing appropriate counseling and ensuring equitable inclusion in clinical trials is crucial for patients of African ancestry with ADs.[163,165] These patients may respond differently to ADMTs such as azathioprine and belimumab than do patients of other ancestries.[166,167] It is important to note that African Americans carry the memories of historical injustices in medicine, such as the 1932-1972 Tuskegee syphilis experiment, during which drug treatment was withheld from males of African ancestry.[163,168] Moreover, appropriate counseling has the potential to mitigate the increased risk of IDs in individuals of African ancestry treated with biologic ADMTs.[65,67,169]

Infectious agents. Infectious agents are well-known AD determinants.[5,162,170] All types of pathogens can trigger autoimmunity through mechanisms such as molecular mimicry, epitope spreading, and bystander activation of autoreactive B and T lymphocytes.[26,171-174] According to epidemiologic and molecular data, the most incriminated infectious agents are viruses, particularly Epstein Barr Virus (EBV) and severe acute respiratory syndrome coronavirus 2.[5,26,175-178] A study conducted in Tunisia found that lymphocytic EBV infection was present in the salivary glands of patients with Sjögren disease but not in the control group.[113] However, the reported absence of a significant association between EBV infection and Sjögren disease in that study was potentially due to a low statistical power.[51] EBV, Human Immunodeficiency Virus, and *Mycobacterium* spp are potential major determinants of inflammatory bowel disease in people of African descent.[17,26,57,58,179,180] *Mycobacterium* spp. are part of the "sarcoidosis autoantigenome," although their pathogenetic mechanisms in this context are unclear.[181,182] In the rest of this section, we take a look at the link between human malaria and human ADs.

Overlapping characteristics	Malaria	Lupus
	Susceptibility and severity factors	
Genetic	○ Classes I and II HLA genes ○ Outside the HLA system: *IL23R*, *IL12RB2*, *FCGR2A*, *IL 10*, *FcyR,* and IFN and TNF genes ○ Kidney involvement-specific gene: *APOL1*	
Epigenetic	DNA modifications	
Microbiome dysbiosis	Yes	
Psychological	Yes	
Hormonal	Yes	
Ecologic and sociologic	Geo-spatial factors, climatic factors, poor housing conditions, low economic status	
	Soluble and tissular immunopathologic features	
Autoantibody-producing cell	$CD11c^{+}T\text{-}bet^{+}ABC$	
Autoantibodies	○ ANA ○ Against membrane antigens: aPL ○ Against the Fc fragment of IgG: RF	
Other cells and biomarkers	○ Cells: Tfh, $CD8^{+}$ T, gamma-delta T, monocytes, macrophages and dendritic cells ○ PRRs: TLR7, TLR9 ○ Major cytokines: IFN markers and interleukins	
	Epidemiology	
Most vulnerable populations	○ Women of reproductive age ○ Children under five years	○ Women of reproductive age ○ Pediatric age group
	Organ involvement and treatment	
Most frequent end-organ/systemic complications	○ CNS involvement, Kidney involvement/ESKD ○ Thrombocytopenia, autoimmune hemolytic anemia, leukopenia, and lymphopenia	
AM treatment effectiveness	Yes	

FIG 2. The "malaria-lupus tautology."[23,25,70,80,89-93,96,102-109,157,158,171,186-190,194,196-199,207-246] The malaria features described refer mostly to *P falciparum* malaria, and the lupus features described refer mostly to SLE. Malaria and lupus share disease determinants and features and antimalarials (AMs) are used to treat both malaria and lupus.[23,25,70,80,89-93,96,102-109,157,158,171,186-190,194,196-199,207-246] Shared genetic factors include Human Leukocyte Antigen (HLA)[96,194,207,208] and non-HLA[89-91,96,194,208-211] genes. Deoxyribonucleic acid (DNA) modification is an epigenetic mechanism incriminated in the pathogenesis of both malaria and lupus.[212,213] Age-associated B cells (ABCs) responsible for the production of autoantibodies in individuals with malaria and those with lupus have been found to exhibit the following common characteristics: derivation from an interferon (IFN)-γ -mediated differentiation of naive B cells, identical autoimmune segment (VH4-34), and similar transcriptional profiles.[23,197] The following ANAs have been found in individuals with malaria: IgG/IgM/IgA anti-single and double stranded deoxyribonucleic acid, and anti-Smith/ribonucleoprotein.[171,186-188] Women of reproductive age at highest risk of contracting malaria are pregnant women,[25] and available data do not highlight a striking risk difference for lupus across the age spectrum in the paediatric population.[80] Whilst all AMs used in clinical practice have been shown to be variably effective in individuals with malaria,[242] hydroxychloroquine and chloroquine have been the most tested and proven effective AMs in lupus patients.[243-245] *aPL,* Antiphospholipid antibody; *CNS,* central nervous system; *ESKD,* end-stage kidney disease; *PRRs*, pattern recognition receptors; *RF*, rheumatoid factor; *Tfh*, T follicular helper cell; *TLR*, Toll-like receptor; *TNF*, tumor necrosis factor.

Does malaria trigger polygenic ADs? It has been observed in and outside Africa that *Plasmodium falciparum* triggers the production of numerous autoantibodies (eg, anti-smooth muscle antibody, anti-parietal cell antibody, anti-red blood cell antibody, antilymphocyte antibody, rheumatoid factor, antiphosphatidylserine, and antinuclear antibodies [ANAs]) in people with chronic and acute malaria.[171,183-190] A study published in 2004 found that 27% of 124 West African immigrants in Italy who had previously experienced repetitive episodes of malaria and diarrhea in Africa were seropositive for ANAs. In comparison, only 3.5% of 375 White Italian relatives of patients with SLE were seropositive for ANAs. The data from West Africans were further divided on the basis of their duration of stay in Italy. The ANA frequencies were 35%, 19%, and 14% for groups having stayed in Italy since 3 months to 7 years, 8 to 14 years, and 15 to 21 years, respectively.[191] IgM and IgG antiphosphatidylserine autoantibodies were also found in sera of people with *Plasmodium malariae, Plasmodium knowlesi,* and *Plasmodium vivax* malaria in Asia.[192] A study from Nigeria suggested NETosis could be a mechanism for the production of the ANA termed anti-deoxyribonucleic acid in people with *P falciparum* malaria,[185] as commonly observed in individuals with SLE.[193] The development of cell-mediated immune responses has also been reported in individuals with malaria.[188,194]

Could malaria-induced autoimmunity be pathogenic?[171,183] It has been reportd that human malaria autoantibodies are produced by a segment of age-associated B cell known as the autoantibody-producing segment (VH4-34), which is involved in the pathogenesis of ADs.[23,195-199] IgG and IgM antiphosphatidylserine serum autoantibodies were correlated with anemia in Ugandans and Malaysians with malaria.[188] Anti-deoxyribonucleic acid autoantibodies were correlated with

TABLE I. Barriers to the diagnosis of ADs in Africa

Level of barrier	Barrier		Comment
Patient- and public-related	Low awareness for ADs and discrimination against individuals with ADs in the society		Limited awareness among patients, patients' families/guardians, and the public: source of discrimination against, stigmatization and isolation of patients with consequential reluctance to attend to health care settings in some cases[15,16,41,51,58,111,112,273,274]
	Unadvisable health-seeking behaviors		- Low self-health perception[138] - Prioritization of self-management strategies (eg, use of over-the-counter glucocorticoids to relieve arthritis pain) and care from informal medicine retailers (street drug sellers, spiritual leaders) at the expense of hospital care[136]
	Low income		Unaffordability of autoimmunity diagnostic tests often carried out through subcontracting between local and international health laboratories[15,16,27] For example, the estimation of detailed ANA assessment at 53.3 € through health laboratory subcontracting in Gabon[275] provides a glimpse of the burden imposed on people with low income[27] by costs of autoimmunity assessment in sub-Saharan Africa
Disease-related	- Overlap of disease features between ADs and other diseases highly prevalent on the continent such as IDs and SCD - Insidious forms of disease (eg, in AIH)[60] - Possibility for spontaneous disease remission (eg, in a subset of patients with sarcoidosis)[182]		- Overlap of clinical features: barrier to the diagnosis of ADs when autoimmunity testing cannot be done[22,27,58,256] - Overlap of autoantibodies: a barrier to the diagnosis of ADs in individuals with chronic IDs such as HCV infection, previous repetitive episodes of IDs (eg, malaria), or SCD[22,23,58,256] - Overlap of morphologic (endoscopic/histopathologic) lesions: barrier to the attribution of an AD etiology to morphologic lesions (eg, tuberculous colitis and CD) and source of delayed diagnosis made only after the failure of a course of antibiotic (eg, antituberculosis) treatment[58]
Health system related: limited diagnostic capacity	Limited diagnostic offer due to health care setting deficiencies	Precarity of ambulatory, home and street based models of care	Limiting factor for a timely diagnosis of ADs in disabled patients unable to attend to health care settings[276]
		Deficiencies in public and private health laboratories	- Limited availability of tests for autoimmunity and histopathologic (eg, renal and salivary gland) analyses[15,16,51] - Limited availability of equipments for autoimmunity assessment[15,16,38,272,277]
	Limited diagnostic offer due to providers' biases		- Shortage of health care professionals, especially physicians who represented 9% of 3.6 million health care professionals across 47 countries in Africa between 2018 and 2019[278] - Limited awareness for ADs among health care professionals[15,16,58,136,272,279] - Limited interest in ADs among aware health care professionals[272,274] - Few local opportunities for training on autoimmunity and limited possibility for interested health care professionals to take advantage of online opportunities of training owing to low incomes[58] and reduced access to optimal internet connexion[280]

AIH, Autoimmune hepatitis; *CD*, Crohn disease; *HCV*, hepatitis C virus; *SCD*, sickle cell disease.

anemia in Ugandan children with severe *P falciparum* malaria.[200] It has also been reported that *P falciparum* malaria–related anti-deoxyribonucleic acid autoantibodies are nephritogenic,[171,183,188,200] and both IgM and IgG anti-deoxyribonucleic acid autoantibodies that were found in people with a recent or past episode of malaria[187] have been incriminated in SLE pathogenesis.[2] In a study conducted in Brazil, the authors reported a destruction of infected red blood cells by cytotoxic CD8 T cells in people with *P vivax* malaria.[201] Because of the cross-sectional nature of these studies, it remains unclear whether malaria autoantibodies are causally associated with the clinical expression of autoimmunity. However, it has been observed that antimalarial drugs are highly effective in reducing antiphospholipid antibody levels and associated thromboembolic events in antiphospholipid antibody–seropositive adults during follow-up.[202] Additionally, there was an inverse association between maternal use of hydroxychloroquine during pregnancy and cardiac neonatal lupus in infants born to anti-Ro–seropositive women.[203] These findings[202,203] highlight the need for longitudinal studies assessing whether there is a causal link between malaria and the clinical expression of autoimmunity in humans.[202-204]

ANA seropositivity following malaria exposure could raise the suspicion for ADs[5,158,205] such as scleroderma[206] and lupus (Fig 2).[23,25,70,80,89-93,96,102-109,157,158,171,186-190,194,196-199,207-246] In a cross-sectional study involving 1297 unhealthy people, including individuals with unspecified IDs and 188 individuals with SLE, the specificity of ANAs for the SLE diagnosis increased with ANA titer and number of positive autoantibodies against nuclear antigens.[247] Accordingly, future studies of

Box 1. Suggested research pillars to improve the understanding of ADs in people of African descent[5,51,92,99,110,114-119,127,134,144,162,170,194,220,264,265,300-308]

General notes
- ❖ Data disaggregation by ancestry subtype (i.e., African only versus African admixed), age, sex, birthplace, country of origin, tribe, geographic location (including the area (urban versus rural) and the migration status), and any other nondiscriminatory social identity deemed relevant
- ❖ Scrutiny of the causal relationships existing between different ADs
- ❖ Epidemiologic assessment of the autoimmune lymphoproliferative syndrome in the global population of African ancestry
- ❖ Tracing the history of ADs in Africa and people of African ancestry living outside Africa, including any information about a possible historical link with other diseases commonly found in Afro-descendants

Patient-related biologic determinants
- ❖ Priority for studies of African genomic, "phenomic" (including blood groups), and "microbiomics" biomarkers of autoimmunity, including classic and "dark matter" biomarkers

Infectious agents
- ❖ Scrutinity of the pathogenicity or protective effect of different autoantibodies from each *Plasmodium* spp. across different age groups, and if necessary, identification of predictors of transition from preclinical to clinically manifest AD → need for (1) long-term prospective cohort studies specifying the titers of autoantibodies in participants with acute, repetitive episodes, chronic ADs, or history of malaria; (2) studies tracking *Plasmodium* spp. in people with newly diagnosed ADs before the commencement of antimalarial drugs in and outside Africa
- ❖ Scrutiny of the possible interactions between *Plasmodium* spp. and typical AD determinants such as EBV infection and pollutants, as well as the putative independent role of other infectious agents
- ❖ Assessment of the long-term effect of malaria and other vaccines on the incidence of ADs in Africa
- ❖ Data disaggregation by antimalarial-resistance strain of *Plasmodium*

Pollutants and climate hazards
- ❖ Long-term prospective cohort/case-control studies of the effect of different pollutants from forest fires, wood smoke, charcoal, fugitive dust, silica, and other sources on different ADs
- ❖ Studies of seasonal variations of ADs and the impact of climatic factors on ADs in Africa

Psychosociologic factors
- ❖ Robust data on the association between psychological factors and ADs in people of African ancestry around the world
- ❖ Cohort/case-control studies on individual and intersecting sociologic determinants of different ADs in Africans around the world
- ❖ Scrutiny of the impact of sociologic factors on the African autoimmune genome and epigenome

To allow more accurate conclusions and comparisons of data from Afro-descendants within and across different regions of the world, future studies will need to consistently report patients' ancestry. This is important given that the terms *race* and *ethnicity* are social constructs whose classification varies with the geographic region and is expected to evolve with the advancement of societies' knowledge.[307,308] Accordingly, the use of the terms *race* and *ethnicity* may lead to selection, analysis and interpretation biases in studies.[144,307] Indeed, the population of African ancestry is a multiracial population, and even the dark skin tone most commonly found in sub-Saharan Africa is found in all regions of the world because of the settlement of some sub-Saharan Africans outside Africa during the transatlantic slave trade.[308] The specification "African ancestry/descent/heritage" together with clear information about individuals' geographic locations, will also help to uncover the specific effects of settling environments in the pathogenesis of ADs in people of African descent.[144]

malaria should specify participants' autoantibody titers to help guide the follow-up of malaria patients seropositive for ANAs.

Can malaria indirectly trigger ADs? It is common for humans to contract malaria alongside other endemic infections caused by intracellular pathogens known as triggers of ADs (eg, EBV infection).[26,248] This is mainly because once in the human body, *Plasmodium* parasites can reactivate latent infectious agents with which they have phylogenetic links.[248,249] In this light, *Plasmodium* parasites could be viewed as indirect triggers of ADs commonly triggered by EBV including lupus, Sjögren disease, rheumatoid arthritis, and multiple sclerosis.[179,250-252] Besides their potential indirect triggering role, *Plasmodium* parasites may enhance AD activity directly by eliciting the production of anti-Smith autoantibodies, or indirectly through the reactivation of EBV with consequential production of anti-Smith autoantibodies.[70-73,179,248-251] Another mechanism through which *Plasmodium* parasites could enhance AD activity is by upregulating interferon signaling directly[232,233] and/or through the reactivation of viral infections.[162,179,248,249]

Have ADs evolved from Plasmodium*-mediated genetic selection?* It has been reported that human ADs have evolved from the selection pressure of pathogens on the human genome.[170,253] One such pathogen could be the *Plasmodium* parasite, which has been a major threat to human health for thousands of years across the globe, particularly in Africa.[25,194,207,208,254] *Plasmodium* parasites have been dsecribed as the most powerful known pressure forces on the human genome.[194,208] They reportedly have selected genes responsible for immune dysregulation and inflammation in humans.[194,208] However, the specific human immune-related conditions shaped by malaria have not been clearly elucidated.[194,208] Interestingly, shared immune response gene variants between human malaria and human lupus have been described, as shown in Fig 2. This suggests that human lupus, which has been known since the Middle Ages,[255] could have resulted from selection pressure of *Plasmodium* on the human genome. Findings of an overlap of autoimmune pathways[256] between lupus and sickle cell disease, which is a mendelian disease known to have resulted from selection pressure of *Plasmodium* on the human genome,[194,207,208] may be an additional indirect argument for a potential evolutionary link between malaria and lupus.

Besides lupus, other ADs could have evolved from the selection pressure of *Plasmodium* parasites on the human genome, given the potential association between multiple ADs and

Activity	Comment	Health care professionals involved	Potential partners
Diagnosis and management 	Diagnosis and management of ADs based on task-shifting and task-sharing models as observed with other non-communicable diseases in Africa	Autoimmunologists/immunologists/internal medicine physicians, other relevant medical/surgical specialists, and allied health care professionals working in and outside autoimmunity centers of excellence	• Ministries of Health in Africa • Other local public and private health care settings • Local centers of organ/tissue/cell replacement/transfert therapy • Local centers of assisted reproduction and women's health • Autoimmunity centers of excellence outside Africa • Non-governmental organisations
Education 	• Coordination of training of autoimmunologists • Acceleration and fine-tuning of the education of non-specialist health care professionals and the public on ADs in Africa	Autoimmunologists, immunologists, internal medicine physicians, and other relevant medical specialists (e.g., Rheumatologists, Dermatologists, Nephrologists, Neurologists, Cardiologists, Gastroenterologists, Haematologists, Endocrinologists)	• Ministries of Health in Africa • Scientific and professional societies/organizations of health care professionals involved in the management of and research on ADs in and outside Africa • Patient organizations in and outside Africa
Research 	Epidemiologic and basic science studies on all aspects of ADs in Africa, including the efficacy of African herbal medicines	All health care professionals/scientists/researchers with interest on ADs in individuals of African ancestry	• Autoimmunity centers of excellence outside Africa • H3Africa/Other African genomic consortia • ID research centers • African herbal medicine research centers • AD funding bodies • Non-governmental organisations

FIG 3. Proposed road map for establishing an autoimmunity center of excellence with integrated digital clinic in Africa.[15,16,38,58,136,151,152,280,288,289,309,310,324,325] The Human Heredity and Health in Africa (H3Africa) consortium involves a collaboration between the NIH and Wellcome Trust and the African Society of Human Genetics that has successfully led to a better understanding of genetic determinants of many diseases highly prevalent in individuals of African descent.[325]

glucose-6-phosphate dehydrogenase deficiency,[257,258] which is another mendelian disease shaped by malaria.[194,207,208] The causal role of lupus in other ADs, such as primary biliary cirrhosis,[259] the "autoimmune tautology" phenomenon,[9,220] and the overlap of clinicopathogenetic features between ADs and other immune-related conditions (eg, autoinflammatory diseases,[4] primary immunodeficiencies,[260,261] and atopic and Kikuchi-Fujimoto diseases[2,262,263]), are additional indirect arguments suggesting that if human malaria has shaped human lupus, then it has also potentially contributed to shaping other human ADs, autoimmune lymphoproliferative syndrome,[264,265] and other diseases in immunology/allergy and hematology/oncology.[266,267] In the same vein, malaria could have contributed to the shaping of multiple myeloma[268,269]; the bases for this assumption are data highlighting features of autoimmunity[270] and a predominance of the African ancestry[269] among patients with multiple myeloma, as well as the spectacular improvement of multiple myeloma manifestations which was observed in some patients following antimalarial drug treatment.[267,271]

DIAGNOSTIC AND THERAPEUTIC CHALLENGES IN AFRICA

ADs are underdiagnosed and underreported in Africa.[15,16,58,272] Table I provides a summary of the challenges in diagnosing ADs in Africa, especially sub-Saharan Africa.[15,16,22,23,27,38,41,51,58,60,111,112,136,138,182,272-280]

Three main factors hinder the management of diagnosed ADs in Africa. These factors are deficiencies in the management framework, the huge burden of highly immunosuppressive IDs such as tuberculosis and Human Immunodeficiency Virus infection,[17,281] and unadvisable patient behaviors such as low adherence to standard-of-care management and combination of multiple models of care (eg, ADMT-traditional African medicine combination) at the discretion of health care providers.[16,136,272]

An important deficiency in the management framework is the limited understanding of African-specific AD features, which reduces the possibility to implement personalized ADMTs.[15,16,41,51,282] Additionally, there are few robust data on the effectiveness of current immunosuppressive and immunomodulatory ADMTs[283] in Africa,[16,41,51,58,169] as well as limited availability and affordability of those ADMTs.[15,16,41,51,169]

The limited availability of ADMTs in sub-Saharan Africa could be due to a limited universal health insurance coverage and implementation of the World Health Organization's essential medicines list for ADs (clearly considered for the first time in 2023) in many countries in Africa.[284-286] Consequently, synthetic ADMTs are often available intermittently at unaffordable costs for many patients in private pharmacies surrounding the health care settings where those ADMTs are most frequently prescribed.

Biologic ADMTs are prescribed infrequently because of their original compounds' limited availability and the prohibitive cost of their biosimilar products.[169] Regarding instrumental ADMTs, kidney dialysis is the most accessible.[287] Transplantation[288] (including chimeric antigen receptor T-cell therapy[289]) and apheresis[290] are not readily available in many countries in sub-Saharan Africa.

DISCUSSION

Current data highlight a growing number of individuals diagnosed with ADs and allergic diseases[291,292] in Africa. There is also an association between IDs and ADs, which suggests that we need to reconsider the "hygiene" and "lupus gradient" hypotheses, which are supported primarily by animal data.[23,293-296] Furthermore, these data highlight the need for extensive confirmation bias–free[297] research on the burden, determinants, and mechanisms of the more than 100 organ-specific ADs and SADs[298,299] in individuals of African ancestry around the world (Box 1).[5,51,92,99,110,114-119,127,134,144,162,170,194,220,264,265,300-308]

There is also an urgent need for improved AD care in people of African descent worldwide. To provide holistic management of ADs in this population group, complementary and alternative therapies[309] valued by patients of African ancestry (eg, spirituality and African herbal medicines[111,136,310]) need to be tested in robust randomized clinical trials including those patients, and implemented if definitely proven effective.[310,311] Proactive social care measures also need to be implemented for patients with low socioeconomic status.[141,312] This requires an unbiased implementation of DEI to ensure that health care professionals and patient representatives of African ancestry, who are more aware of cultural sensitivities of and challenges faced by people of African ancestry, are equitably represented on the boards of AD clinician and patient organizations and funding bodies.[313-315] In Africa, where there are competing health interests,[15,16,41] the best approach to AD care provision would require a paradigm shift that regards all ADs as a single disease landscape[151,152] in existing noncommunicable disease programs.[15] When grouped, ADs that broadly require similar management and biopathologic diagnostic procedures[5,9,151,152,220] are not infrequent.[10-12] The capitalization on ID programs, which are the most important health programs in Africa,[17,25] could also help provide some resources for AD care on the continent.

Regulatory implementation of advances in the artificial intelligence and Internet landscape are expected to help improve the management of and research on ADs in Africa.[316,317] These advances provide a great opportunity for distance learning, which can further increase awareness and interest in ADs among health care professionals on the continent.[280] Additionally, they have the potential to facilitate networking, advocacy, and telemedicine activities among enthusiasts of autoimmunology in people of African descent around the world.[318,319] Finally, these advances enable accurate data recording.[29,320] This means that governments in Africa need to extend the supply of a symmetric Internet bandwidth across the continent and further develop electronic health record systems for an accurate record of data on ADs in Africa.[11,12,29,280,320,321]

Good partnerships between public health care facilities and private autoimmunity centers of excellence[322,323] (Fig 3)[15,16,38,58,136,151,152,280,288,289,309,310,324,325] are also expected to contribute to improving research on and care provision for ADs in Africa, as seen with other diseases on the continent[326] and with ADs on other continents.[152,318,322]

CONCLUSION

There are increasing numbers of individuals diagnosed with ADs in the global population of African ancestry, especially those of sub-Saharan African ancestry whose data represent the bulk of data highlighted in this review. The increasing magnitude of ADs in this population could be due to genetic and sociologic factors, as well as to exposure to IDs and pollutants. To improve care and research on ADs in people of African descent, it is important that DEI be embraced and that governments in Africa harness the power of digital health. This will enable timely diagnosis and proper management of ADs in all persons of African ancestry, regardless of their geographic location, or social identity, just as is done for any other person of any other ancestral group.

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