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Commentary

Characteristics of Humoral Autoimmunity against Nucleus Components

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DESCRIPTION

Systemic Lupus Erythematosus (SLE) is a multi-system autoimmune illness characterized by humoral autoimmunity against nucleus components, notwithstanding its various presentations. In SLE, autoantibodies attach to cognate autoantigens, causing immune complex accumulation and tissue destruction. Because of the wide variety of clinical symptoms, ideal standards for SLE classification, as well as the question of whether SLE reflects numerous diseases with a final common pathway leading to disease or an aggregation of pathophysiologically connected disorders, are still debated. Furthermore, because many SLE patients (up to 40%) have antiphospholipid antibodies or overlap autoimmune disease syndromes, the most common of which is secondary Sjogren's syndrome (up to 25%), overlap with other associated disorders is frequently part of SLE categorization. Given the variety of possible syndromic combinations that could satisfy SLE categorization criteria, one could expect genetic investigations to be unrevealing. Yet, early studies of genetic linkage in multiplex lupus families, as well as more recent genome-wide genetic association studies, have shown a plethora of genetic risk factors for the development of SLE and specific clinical presentations. These investigations demonstrate a spectrum of genetic components, including monogenic routes to disease, which are more common in childhood-onset SLE, and polygenic plus environmental routes, which are more common in adult-onset SLE.

It has been found that SLE risk and clinical manifestation differ by race, geography, and sex at birth, with females from numerous non-European derived populations having the highest prevalence and severity. For example, according to a recent cause of death analysis, SLE is the 10th major cause of mortality in females aged 15-24 in the United States, whereas it is the 5th highest cause of death in females aged 15-24 in both African-American and Hispanic populations. It's tempting to infer that these disparities are the result of genetic divergence related to the continental ancestral distinctions that distinguish these populations. It is worth noting that population-level genetic variations caused by divergence are estimated to account for no more than 16% of genetic variability in human groups, with the majority of genetic variance caused by within-population differences. The tendency to attribute population-level incidence and severity disparities to divergent genetic variables stems from examples of genetic variants having disproportionate effects in other disease states (i.e. APOL1 variants associated with risk for ESRD and protection from African sleeping sickness or SLC16A11 and risk for type 2 diabetes in Mexican Americans).

Illness risk alleles like these, which are only found in one continental ancestral population, are thought to be the result of random chance and historical founder or bottleneck effects. It is crucial to note, however, that concordance rates of SLE for monozygotic twins are predicted to be around 25% based on twin studies. While there are significant population-level differences in SLE risk and severity, these genetic risk variables must be carefully positioned within the broader risk environment in which they function. That is, to truly understand the causes of observed population-level differences in prevalence and severity, longitudinal studies comparing the relative contribution of systemic structural factors, environmental exposures, access to healthcare, and population-level genetic differences to both SLE risk and disease severity will be required, as previously outlined. It is not fair nor reasonable to infer that population-level differences are caused by underlying genetic differences. While either case may eventually be proven true or partially true (that is, if population-level genetic differences are discovered to explain some of the increased risk of SLE), care must be taken to avoid perpetuating population-level health disparities by incorrectly attributing or incorrectly not attributing population-level differences to genetic causes.

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