



# The whole lupus: Articulating biosocial interplay in systemic lupus erythematosus epidemiology and population disparities



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## ABSTRACT

Systemic lupus erythematosus (SLE), commonly known simply as lupus, is an autoimmune disease in which the body's immune system attacks healthy tissue and organs. Characteristic of the disease is a disproportionate effect on women and communities of color, both in terms of prevalence and severity of symptoms. Lupus is also both genetically driven and subject to external environmental conditions, many with place based corollaries. Thus, lupus presents a series of complex and intersecting biosocial questions regarding its origin and treatment, questions which transdisciplinary approaches are uniquely suited to address. In this paper, we propose a framework, incorporating critical approaches to the production of embodied formations of race and gender as well as new understandings of the impact of environmental conditions and lived experience at the genetic level, that can direct future research into lupus that is both more inclusive of a range of influences and more precise in its ability to treat and diagnose the disease.

## 1. Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease, specifically an inflammatory disease in which the body creates antibodies that attack its own tissues and organs. Effecting an estimated 1.5 million Americans and 5 million people worldwide<sup>1</sup> (Lupus Foundation of America, 2013), SLE typically develops between the teen and adult years, ranging from ages 15–44. SLE is characterized by chronic pain and inflammation across numerous systems, including the kidneys, brain, blood cells, joints, skin, lungs, and heart (Schur and Gladman, 2011). There are currently recognized a number of pathognomonic antibodies in lupus that correlate with or predispose to certain manifestations, some more severe than others. Given these multiple autoantibodies, lupus presents a host of symptoms such as fatigue, fever, joint and chest pain, confusion and memory loss, skin lesions, and most characteristically, a butterfly shaped rash across the nose and cheeks. These symptoms can appear suddenly or develop slowly, depending on the specific cause and which body systems are affected. Most cases of lupus are character-

ized by flares, with the disease worsening for a time before improving (Schur and Gladman, 2011).

People of all genders, sexes, races, ancestral backgrounds, and ethnic groups can develop lupus, however its impacts are not evenly distributed amongst those groups. 90% of patients who are diagnosed with lupus are women. It is believed that this disparity is caused by hormones, given that women begin estrogen production in the same years that lupus begins to develop. Additionally, SLE is two to three times more prevalent in all women of color (e.g. African American, Latina, Asian, Native American, Pacific Islander) than it is for white women (Danchenko et al., 2006). Again, the reasons for this discrepancy are unknown, but research has shown that high interferon-alpha (IFN $\alpha$ ) levels are associated with the manifestation of lupus.<sup>2</sup> Blacks and Latinos have higher levels of IFN $\alpha$  than white Americans (Weckerle et al., 2011). While there are hundreds of related genes involved in the production of interferon pathways, currently six different genes are recognized as most representative of this process, each with distinct differences between African Americans and whites (Niewold et al., 2008).

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<sup>1</sup> Estimates of the incidence and prevalence of SLE vary. Prevalence in the US has been estimated at 24–150 per 100,000 and estimates of incidence in the US range from 2.2 to 5.6 per 100,000 (Feldman et al., 2013).

<sup>2</sup> Interferon-alpha is one of a large subgroup of interferons, or signaling proteins, produced by white blood cells and released by host cells in response to the perceived presence of pathogens (De Andrea et al., 2002). Typically, a virus-infected cell will release interferons, resulting in nearby cells heightening their anti-viral defenses.

Though the exact causes of lupus are not fully known or understood, there is general agreement that both genetic and environmental factors contribute to the development of the disease (Demas and Costenbader, 2009). While the genetic correlates in lupus prevalence would suggest a biological origin, current research also identifies key environmental and social dimensions. Exposure to toxins in the built environment and through occupational contact, along with less tangible but still impactful psychosocial experiences, point to a constellation of external factors that contribute not only to the disease's prevalence, but also to variations in the lived experience of those suffering from SLE (Cooper, 2010; Finckh et al., 2006; Chae et al., 2015). The inequities in terms of prevalence and experience of SLE forces an examination of the disease as more than biological and more than social; we label this approach *biosocial*, following Mansfield (2008), and see it as crucial for interdisciplinary attempts to understand and treat lupus.

Historically, mainstream epidemiology has tended towards an understanding of social categories as static and fixed, with disparities across racial and ethnic lines attributed to specific genetic differences (Krieger, 2011; Gravlee, 2009; Lock, 2007). Social scientists, including feminist and critical race theorists, have challenged a naturalized, biological notion of racial and gender identity, favoring instead an understanding of these categories as socially produced; lenses through which reality is perceived and interpreted, with very real effects on the material well-being of individuals (Gravlee, 2009; Gilbert, 2000). Importantly, these constructs are understood as *constantly emergent* processes subject to shifting social and material conditions (Omni and Winant, 1994; Butler, 2011). Recently, some social scholars have attempted to follow the emergence of race and gender back into biology. Put concretely, while race and gender may indeed be social constructs, they are also *dialectically* biological concepts that produce distinct yet shifting and evolving lived experiences which inform the health and well-being of all human bodies living within racialized and gendered systems (Levins and Lewontin, 1985). The social realities of difference (for example, in the form of systemic sexism or racism) are seen to have biological consequences for marginalized groups (Gravlee, 2009). While conventional epidemiology has moved away from reductionist conceptions of disease distribution, embracing social determinants of health as an organizing principle, more work is needed to understand the fluid and historically and socially contingent natures of social difference and inequity and their relationship to individual and population health. Given that SLE has been understood as a disease that *targets* certain groups, it is worth considering how the material consequences of race and gender as concepts may *become* biological through SLE in ways quite distinct from static (i.e., genetic) interpretations.

In this paper, we seek to continue the work of theorists (Krieger, 1999, 2012; Gravlee, 2009; Lock et al., 2015; Mansfield, 2012; Springer et al., 2012) who suggest that neither static, naturalized notions of race and gender nor the notion of these identities as biologically irrelevant, can duly account for the health disparities observable across racial, ethnic, and gender lines. In this sense, we understand bodies and the occurrence of SLE to be biosocial, that is, the result of a co-constitutive process between so-called biological and so-called social elements of human life. Rather than uncritically attributing difference to biological origins or social determinants, we conceive of biological and social aspects of life as relational and ontologically indistinguishable (c.f. Springer et al., 2012). By examining the interrelation of biology and biography in order to better understand health and disease, we hope to promote research on SLE and other complex diseases that innovatively conceptualizes and attends to the dense interactions between ostensibly distinct factors/features of disease (c.f. Horwitz et al., 2017).

The challenge remains to conceive of biographical and biological factors simultaneously, and to articulate the interrelation between these factors as life unfolds and bodies develop. Recently, social

scholars interested in understanding the human body as biosocial have begun to latch on to biological concepts that describe distinct mechanisms through which the biological and the social are known to interrelate (e.g., Guthman and Mansfield, 2013; Meloni, 2014, 2016; Landecker and Panofsky, 2013). Our work on SLE advances this approach through a discussion of two such concepts: epigenetics and allostatic load. We also include a holistic conception of “disease” itself, following Mol and Law (2004), which allows us to theorize SLE in terms of unequal prevalence, experience, and care simultaneously. By creating a conceptual model in which the co-constitutive, biosocial nature of lupus can be considered, we open up a space in which novel treatments and approaches can be developed.

## 2. Building a new framework

We propose a model that understands Systemic lupus erythematosus (SLE) holistically and relationally. Our model articulates the interrelation between biological and social factors, accounts for patients lived experiences and care, and emphasizes the emergent and temporal nature of the disease. In order to best illustrate why the proposed model is crucial in the understanding and treatment of SLE, we compare our model with other dominant models derived from epidemiology.

In recent years, public health and epidemiology as disciplines have shifted their theoretical orientation towards an emphasis on how social structures and political institutions influence health (Krieger, 2011), chiefly characterized by the rise of social determinants of health (SDOH) as a key framing. SDOH understands health disparities not as the result of the innate biology of a given body, but as the cumulative effect of social factors such as early childhood development, socioeconomic status, stress, nutrition and employment (WHO CSDH, 2008). Within a SDOH framing, the social may positively or negatively impact the biological, but this movement is unidirectional. Moreover, experiential, psychosocial impacts of social phenomena on bodies are largely left out of a SDOH formulation (Krieger, 2011).

By contrast, theorists drawing from ecological perspectives, have proposed a number of models (McMichael, 2002; Susser and Susser, 1996) that seek an integrative, multilevel and dynamic means of understanding disease occurrence and health disparities. Most notable amongst these is Krieger's “ecosocial theory of disease distribution” (Krieger, 1994), with its emphasis on the embodiment of lived experience, structural inequities, and the interplay of risk, susceptibility and exposure across spaces and life courses (Krieger, 2011). Recently, as hinted above, others have echoed this more integrative take on health and disease, emphasizing the need to understand so-called biological and social aspects of life as ontologically united (e.g. Mansfield 2012, Landecker 2016, Meloni et al., 2018). Importantly, much of this work has highlighted the role of social difference – especially race and ethnicity, sex/gender, and class – in a similarly integrative manner, examining not simply population-level statistics on racial or gender disparities in health, but rather how race and gender are rematerialized in ways that have distinct (yet not pre-determined) impacts in human bodies. Such an approach to social difference has been considered not only ethically necessary, but also essential for understanding many health phenomena, such as SLE, that are already seen as gendered or racialized in the eyes of medical professionals (Roberts, 2017; Springer et al., 2012).

Aligned with these integrative and relational approaches, and recognizing the importance of disease specific models, we sought to re-conceptualize SLE to account for both the interaction between biological and social factors, as well as the dynamic origin of these factors (Fig. 1). Such a model is particularly important because it allows us to insist that biological factors are rarely “pure” but are rather always already biographical – that is, having to do with the life experience of the body in question. Disease risk, experience, and care and treatment outcomes are a result of the dynamic interplay between biological and social factors, which are always interrelated, even prior to birth.

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