



Editorial

The effect of being African American on atopic dermatitis



Atopic Dermatitis is a complex pruritic inflammatory skin disease that is associated with significant impairment of quality of life and high socio-economic burden.^{1,2} Recent studies reinforce the challenges of managing patients with severe AD as they often suffer from uncontrollable scratching, sleep disturbance, depression and systemic comorbidities.^{3,4} It is often associated with the atopic march⁵ and can be complicated by serious infection.⁶

Understanding mechanisms underlying the various phenotypes and endotypes of AD is critical to the development of a precision medicine approach for this common skin disease. Several of these subgroups include the distinction between severe vs mild AD, highly allergic vs non-allergic AD, transient vs persistent AD as well as early onset vs adult onset AD. The key driving forces shaping AD phenotypes are linked to a combination of skin epithelial dysfunction and polarized immune pathways that may drive the different endotypes.⁷ Adding to the complexity, race is emerging as another factor to consider, e.g. Asians with AD have an increased IL17 related immune response layered on top of the Type 2 immune response seen in all forms of AD. This may account for the increased psoriasiform appearance to eczema in Asian patients.

Molecular profiling of skin is increasingly being used to identify new immune pathways which will benefit treatment of AD subgroups. Most studies have focused on European Americans adults with AD. African Americans with AD have been understudied despite the fact that they may have more severe AD. Therefore there is an unmet need to identify the immune activation pathways in African American patients with AD. A new study published in the current issue of The Annals of Allergy, Asthma and Immunology by Guttman-Yassky and her colleagues⁸ have evaluated potential differences in the molecular profile of skin from African Americans with AD compared to the skin of European Americans with AD with the goal of determining whether there are differences that might improve treatment options for African Americans. Their results indicate that the Type 2 and Th22 immune responses in African Americans with AD are similar to European Americans but African Americans have lower levels of interferon gamma and IL17 expression than European Americans. This immune imbalance may increase the atopic propensity of African Americans with AD. The relative pathogenic contributions of these immune axes in African American patients with AD will need to be established in future clinical trials with immune pathway antagonists or treatment with gamma interferon which has had variable effects in AD.

The current publication complements The AD yardstick, published in The Annals at the beginning of 2018 provided the most recent management guidelines for treatment of AD by the practicing allergist.⁹ A novel aspect of the yardstick was the inclusion of decision-making algorithm incorporating the use of biologics in treating moderate to severe AD. As we move toward more targeted therapies, we need to appreciate the complex heterogeneity of AD that include race-related effects on AD, acquired and genetic skin barrier dysfunction, and different immune pathways that are activated in the various AD subtypes. Because these biologics are expensive, studies are needed which identify biomarkers that predict the patients likely to benefit most from these targeted biologics.¹⁰ Despite these challenges, the future prospects for novel and effective therapies in AD that are further refined by potential differences in various racial groups are exciting.

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Disclosures: Dr. Leung is the Chair of the Data Safety Monitoring Committee; has received fees from Aimmune; consulting fees from AbbVie, Regeneron, Sanofi-Aventis Pharma; and research grants from Inctye Corp and Pfizer.

Funding Sources: None.

<https://doi.org/10.1016/j.anai.2018.09.454>

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