(4.14 days to 1.49 days, \( t = 0.95; P = .37 \)) or clinic capacity, defined as the sum of appointment slots allocated to a given clinic or location (1612.6 to 1722.8, \( t = -1.139; P = .29 \)). There was a significant correlation between the quantity of SFT consultations completed and the percentage of new patients being seen at the MDC within 30 days (\( R^2 = 0.88; P < .05 \)), new patient wait times (\( R^2 = 0.95; P < .001 \)), and percentage of no-shows (\( R^2 = 0.74; P = .001 \)), but not with established patient wait times (\( R^2 = 0.10; P = .36 \)).

Discussion | In previous studies, SFT decreased the time for dermatologic intervention and unnecessary consultations.1-4 A recent literature review revealed programs that had 2 of 4 critical factors (effective preselection, high-quality photographic images, high-quality dermoscopic images if pigmented lesions are evaluated, and supportive infrastructure and culture) had filtering percentages near 50%.5 Our analysis suggests that SFT may improve patient access to the MDC by decreasing the percentage of no-shows and the average wait time for new patients. Interestingly, the quantity of SFT consultations completed was directly associated with an increase in the percentage of new patients being seen at the MDC within 30 days, a decrease in the rate of no-shows, and a decrease in new patient wait times. To determine if these effects were confounded by a change in capacity, we analyzed capacity for both new patient wait times. To determine if these effects were confounded by a change in capacity, we analyzed capacity for both time intervals and found no statistical difference. Our study is limited by the observational pre-post study design, lack of a control group, relatively small number of patients, short follow-up, and veteran population being studied. The differences in the results of the pre-post study outcomes may not be causally related to the selected SFT intervention and may be related to one or a number of other factors entirely. Additional studies will be needed to establish the clinical significance of our observation.

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Accepted for Publication: November 30, 2014.

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Author Contributions: Dr Fabri and Mr Bezalel had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: All authors.
Acquisition, analysis, or interpretation of data: Bezalel, Fabri.
Drafting of the manuscript: Bezalel, Park.
Critical revision of the manuscript for important intellectual content: All authors.
Statistical analysis: Bezalel, Fabri.
Administrative, technical, or material support: Bezalel, Park.
Study supervision: Fabri, Park.

Conflict of Interest Disclosures: Dr Park is an attending physician in the Dermatology Section and Deputy Associate Chief of Staff for Education, James A. Haley Veterans’ Hospital and Clinics, Tampa, Florida. Dr Fabri is an attending physician in the Surgical Service, James A. Haley Veterans’ Hospital and Clinics. No other disclosures were reported.

Funding/Support: This study was supported in part by the resources and the use of facilities at the James A. Haley Veterans’ Hospital, Tampa, Florida.

Role of the Funder/Sponsor: The funding source had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Disclaimer: The contents of this article do not represent the views of the US Department of Veterans Affairs or the United States government.

Additional Contributions: We are indebted to Suzette M. Maynard, James A. Haley Veterans’ Hospital, for her time and help with data extraction. She was not compensated for her contribution.


Low Filaggrin Monomer Repeats in African American Pediatric Patients With Moderate to Severe Atopic Dermatitis

The severity of atopic dermatitis (AD) and intragenic filaggrin (FLG; OMIM 135940) copy number variant (CNV) genotypes were assessed in African American pediatric patients, a health disparities group that is disproportionately affected with AD.1

Methods | The study was approved by Washington University School of Medicine’s institutional review board. Eligibility criteria for recruited pediatric patients were (1) age 3 months to 18 years, (2) United Kingdom Working Party’s Diagnostic Criteria for Atopic Dermatitis,2 (3) African American ethnicity (self-reported), (4) moderate to severe AD (SCORAD index, ≥25),3 and (5) written informed assent or consent. Common European FLG R501X and 2282del4 mutations and intragenic FLG CNV (3 alleles of either 10, 11, or 12 FLG monomer repeats), upon high-quality DNA assessment, were genotyped4 and correlated with AD severity.

Results | Thirty-nine pediatric African American patients with AD were recruited with a mean (range) age of 6.7 (0-4-15) years (Table 1). Thirty-five patients reported a first-degree family member with atopy, and 30 patients reported AD onset before age 2 years. Of the 31 patients who were 4 years or older at the time of visit, a history of asthma and allergic rhinitis and/or hay fever was reported in 24 (77%) and 16 (52%), respectively. Food allergies were reported as well (51% [n = 20]), most commonly peanut (n = 10) and fish and/or shellfish (n = 10) that were not coincident. All but 1 were either being treated with or had been prescribed topical triamcinolone ointment, 0.1%.
The mean (range) SCORAD of the participants was 58.5 (28.0-94.1) (Table 1) with severe pruritis (mean, 7.8) and moderate sleep loss (mean, 5.7) (both scales, 0-10). Twenty-four patients (62%) exhibited severe AD. The mean lesional body extent was 44% and mean lesional intensity was 10 (scale, 0-15; 15 = worst). Lichenification and dryness (both means, 2.2; scale, 0-3; 3 = worst) contributed the most to the lesional intensity (Figure, A).

We sought to explain the associated severe nonlesional skin dryness in our participants by genotyping a previously described European dose-dependent risk factor for AD, intragenic FLG repeats, or CNV. Excluding 2 FLG R501X heterozygous patients (no FLG 2282del4 mutations identified), 16 cases (43%) were homozygous for the FLG CNV 10 allele, thus totaling 20 filaggrin monomers (Table 2). Of the total 74 FLG alleles in our cases (n = 37), the FLG CNV 10 allele made up 64% (Table 1). Patients with a total of either 20 or 21 FLG CNV exhibited higher SCORAD (mean, 63.7) and hence severe AD compared with those participants with 22, 23, and 24 FLG CNV (mean SCORAD, 48.5) (P = .01) (Figure, B). Moreover, we found that individuals with 20 total filaggrin monomers are 1.9 times more likely to have severe AD (Table 2). However, this was not statistically significant (P = .33).

### Discussion

Despite epidemiological data supporting a marked increase in AD in African American children, to our knowledge, a quantitative measure of AD severity and an investigation of FLG CNV in this health disparities group have not been reported until now. We identify a significant difference between low FLG CNV (20 or 21) with severe AD vs FLG CNVOF of 22, 23, or 24 with moderate AD (P = .01). Each FLG repeat encodes 1 posttranslationally modified active filaggrin monomer that is further degraded to metabolites such as urocanic acid that makes up part of the skin’s natural moisturizing factor (NMF). Addition of each FLG monomer decreases the odds ratio of disease risk of AD by 0.88. A reduction of NMF metabolites was also observed in skin of South African patients with AD. Although NMF metabolites were not assessed in this study, the parallels between our study and that of Brown et al with respect to low FLG CNV and AD suggest a reduction in filaggrin metabolites contributing to our patients’ skin dryness. Observed low if not absent frequencies of FLG and/or CNV alleles, R501X excluded, No. (%) 16 (52)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, No. (%)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>18 (46)</td>
</tr>
<tr>
<td>Male</td>
<td>21 (54)</td>
</tr>
<tr>
<td>Age, mean (range), y</td>
<td>6.7 (0.4-15)</td>
</tr>
<tr>
<td>Atopy in immediate family, No. (%)</td>
<td>35 (90)</td>
</tr>
<tr>
<td>Onset age, No. (%), y</td>
<td></td>
</tr>
<tr>
<td>&lt;2</td>
<td>30 (77)</td>
</tr>
<tr>
<td>≥2</td>
<td>9 (23)</td>
</tr>
<tr>
<td>Asthma at age ≥4 y, No. (%)</td>
<td>24 (77)</td>
</tr>
<tr>
<td>Hay fever at age ≥4 y, No. (%)</td>
<td>16 (52)</td>
</tr>
<tr>
<td>Allergy</td>
<td></td>
</tr>
<tr>
<td>Food</td>
<td>20 (51)</td>
</tr>
<tr>
<td>Peanut</td>
<td>10 (26)</td>
</tr>
<tr>
<td>Fish or shellfish</td>
<td>10 (26)</td>
</tr>
<tr>
<td>Quality of life disruption, mean (range)</td>
<td>7.9 (1-10)</td>
</tr>
<tr>
<td>Triamcinolone ointment, 0.1%, treatment, No. (%)</td>
<td>38 (97)</td>
</tr>
<tr>
<td>SCORAD Index, mean (range)</td>
<td>58.5 (28.0-94.1)</td>
</tr>
<tr>
<td>Moderate AD (SCORAD, ≥25-49), No. (%)</td>
<td>15 (38)</td>
</tr>
<tr>
<td>Severe AD (SCORAD, ≥50), No. (%)</td>
<td>24 (62)</td>
</tr>
<tr>
<td>FLG CNV allele, R501X excluded, No. (%)</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>47 (64)</td>
</tr>
<tr>
<td>11</td>
<td>13 (18)</td>
</tr>
<tr>
<td>12</td>
<td>14 (19)</td>
</tr>
</tbody>
</table>

Abbreviation: CNV, copy number variant.

*Scale of 0 to 10.

**Scale of 0 to 103.

### Figure

**A.** Mean Intensity Item Values and Total FLG Repeats vs SCORAD Index

**B.** Distribution of total FLG repeats with respect to SCORAD. The horizontal line in the middle of each box indicates the mean, while the top and bottom borders of the box mark 1 standard deviation above and below the mean. The whiskers above and below the box mark the maximum and minimum values, respectively. Statistics, 2-sided Wilcoxon rank-sum test.
**FLG-2** stop-gain mutations in African Americans and Africans suggest decreased likelihoods for these mutations in AD risk specific to this ancestry. Future case-control studies specific to this health disparities group are warranted to more fully elucidate the genetics of AD.

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