DISCUSSION

Given that psoriasis is increasingly recognized to be associated with comorbid conditions of obesity, diabetes, metabolic dysregulation, and cardiovascular diseases that may be related to inflammation in skin (Davidovici *et al.*, 2010), it is of interest that associated functional pathways were also identified through IPA, including metabolic disease and cardiovascular disease (Supplementary Figure S1b online), leading to two potential mechanisms for increased association between psoriasis and metabolic and cardiovascular comorbidities.First, a product made in psoriatic plaques could produce diffusible hormone-like proteins that influence the biology of distant cells/tissues (e.g., renin, vascular endothelial growth factor, and monocyte chemoattractant 9 protein-1 (CCL2); **Table 4**) **and IL-17 A (Supplementary Figure S2 online)**.

MATERIALS AND METHODS

Serum protein profiling

A 92-protein vendor-defined multiplex Luminex-based panel (Human Map 1.6 plus IL-17 and IL-23; Rules Based Medicine, Austin, TX) was used to profile differential serum protein expression from healthy volunteers ($n = 162$) and patients with psoriasis ($n = 149$). The complete list of analytes in the Human Map 1.6 can be found at http://rulesbasedmedicine.com/products-services/humanmap-services/human-discoverymap.

A more sensitive immunoassay, an IL-17A single-plex assay (Singulex, CA) was used to quantify IL-17A in serum from healthy and psoriatic subjects ($n = 10$ in each group).

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The affiliations in this meeting report are incorrect. The affiliations should be stated as follows:

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Acute Inhibition of MEK Suppresses Congenital Melanocytic Nevus Syndrome in a Murine Model Driven by Activated NRAS and Wnt Signaling

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