the HPV cohort. This could have introduced an ascertainment bias for HPV detection and may explain our lower overall rate of identified HPV. However, it is clear that post-HSCT, the cohort without HPV disease had markedly improved NK cell engraftment when compared with their counterparts with HPV disease.

Investigators have proposed that in γc receptor deficiency, dendritic cells may remain dysfunctional post-HSCT and thus permissive to HPV infection despite donor T cell engraftment.[3] No specific data supports this theory. Our study revealed a lower incidence of HPV disease than in other cohorts, likely related to the lower mean age of our HPV-negative cohort and excellent engraftment of T, B, and NK cells in our entire cohort. We propose that poor NK cell engraftment and function represents a more likely contributor to the development of severe HPV disease in our 4 patients. It is possible that pre-HSCT myeloablation improved NK cell engraftment and function, allowing for improved response to HPV exposure in later life and thus disease limitation. With improvements in early diagnosis and curative treatment in SCID, it is imperative to review and determine the etiology of long-term adverse events like severe HPV disease following transplant.

We would like to thank our patients and their families for their photographs and participation.

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This work was supported by National Institutes of Health (AI067946 to J.S.O.).

Disclosure of potential conflict of interest: J. S. Orange has received consultancy fees from Baxter, CSL Behring, Octapharma, Atlantic Research, Grifols, Bio Products Laboratory, and AmerisourceBergen; has provided expert testimony for the State of Arizona; has received research support from CSL Behring; has received lecture fees from Baxter; and has received royalties from UpToDate. The rest of the authors declare that they have no relevant conflicts of interest.

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Available online August 23, 2014.
http://dx.doi.org/10.1016/j.jaci.2014.07.009

Dry roasting enhances peanut-induced allergic sensitization across mucosal and cutaneous routes in mice

To the Editor:

The reasons for the disproportionate contribution of peanuts to prevalent and severe cases of food allergy in the Western world are unclear. Emerging statistics from East Asia generally match the overall common food allergies in the West, with the striking exception of peanuts, which are consumed equivalently in both regions.[1,2] Differences in peanut preparation, roasted and dry roasted (DR) in the West versus raw, boiled, or fried in the East, have been proposed to contribute to this trend[1-3] and are supported by serological studies.[1,4] Results obtained with other proteins have highlighted the potential immunomodulatory properties of advanced glycation end products (AGEs),[5] which are extensively formed during the high-temperature dry roasting of peanuts.[6] However, despite this information, the immunogenicity and allergenicity of raw compared with DR peanuts has not been characterized *in vivo*.

To address this, we first primed BALB/c mice subcutaneously with endotoxin-depleted soluble fractions of peanut protein extract (PPE) from raw or DR peanuts in PBS without adjuvant (see Figs E1, *A*; E2; and E3, *A*, and the Methods section in this article's Online Repository at www.jacionline.org). This resulted in enhanced peanut-specific IgG titers in DR-primed groups across a range of doses reactive against both raw and DR peanut extracts and with a dominant IgG_1/IgG_{2a} bias (see Fig E4, *A-C*, in this article's Online Repository at www.jacionline.org). After 3 subsequent intragastric gavages of endotoxin-undetectable DR crude peanut homogenate (CPH; see Figs E2 and E3, *A*), anti–raw peanut IgG titers increased by an average of 100-fold more in the DR group compared with those in the raw PPE group and were associated with significant titers of anti–raw peanut IgE, which was functional in basophil degranulation (Fig 1, *A* and *B*). Mesenteric lymph node cells from DR but not raw PPE–primed mice proliferated robustly in response to raw and DR PPE, and cytokines produced were dominated by IL-4 and IL-5 but not IFN-γ and TNF-α (Fig 1, *C* and *D*, and see Fig E4, *D* and *E*). This pattern of enhanced DR PPE immunogenicity was maintained in a similar experiment, in which PPE and CPH were kept homologous (raw/raw or DR/DR) during the subcutaneous prime and intragastric exposures (see Fig E4, *F*). In a further experiment we fed mice previously primed with DR or raw PPE with raw peanut kernels (see Fig E3, *B*) and observed significantly increased anti–raw and anti–DR PPE IgE only in DR-primed mice (Fig 1, *E*, and see E4, *G*). Finally, we excluded

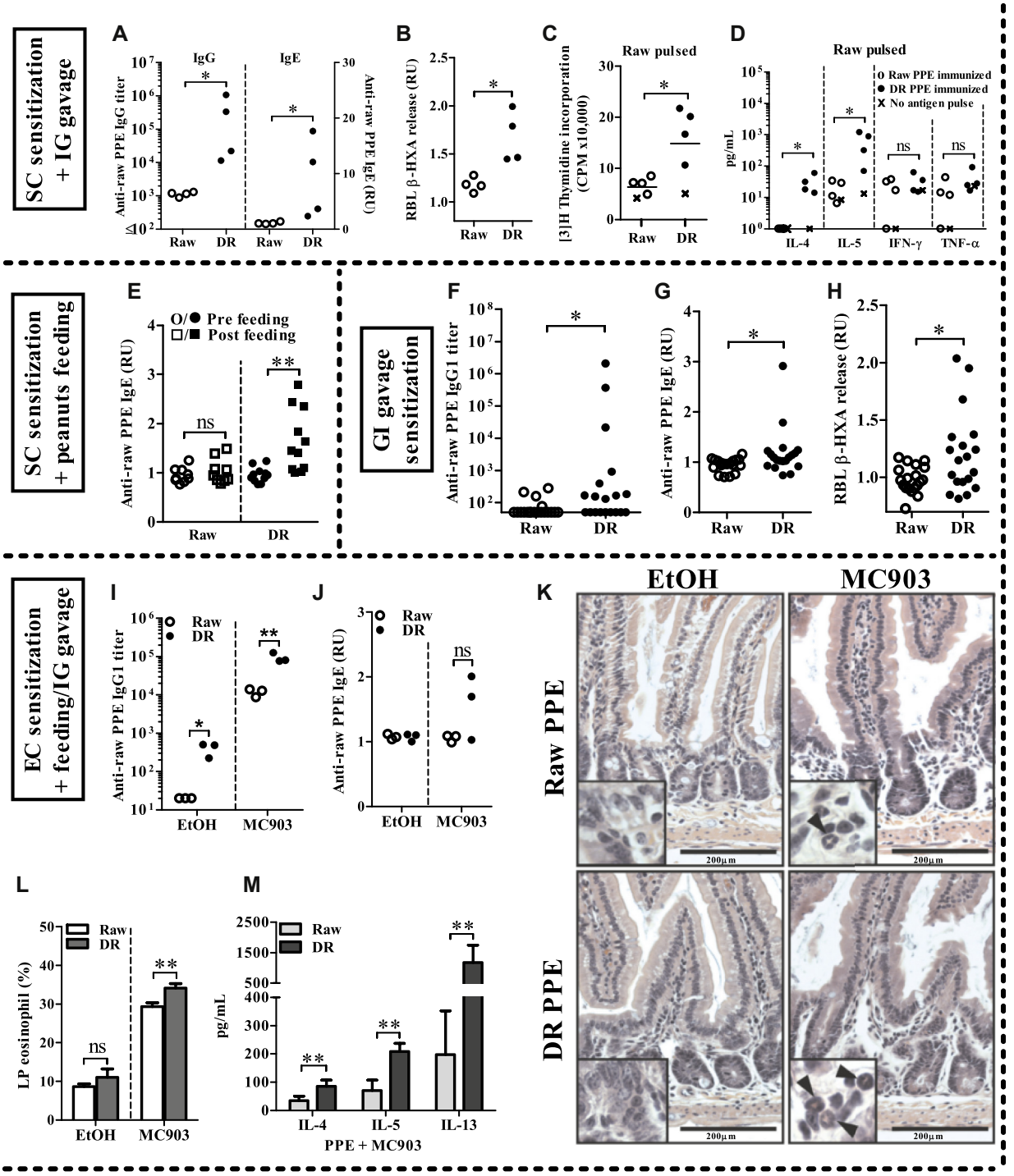

FIG 1. Immunogenicity of raw and DR peanut extracts in BALB/c mice. **A** and **B,** Subcutaneously *(SC)* primed, post–intragastric *(IG)* gavage anti–raw PPE IgG and IgE (Fig 1, *A*) and IgE RBL degranulation (Fig 1, *B*). **C** and **D,** Tritiated thymidine incorporation and cytokines in raw PPE–pulsed mesenteric lymph node cell cultures from the experiment in Fig 1, *A*. **E,** Anti–raw PPE IgE responses to SC sensitization and raw peanut feeding. **F-H,** Anti–raw PPE antibody and RBL degranulation to direct gastrointestinal *(GI)* sensitization. **I** and **J,** Anti–raw PPE antibody responses to epicutaneous *(EC)* sensitization. **K,** Eosinophils *(arrowheads)* in hematoxylin and eosin staining of jejunum lamina propria. **L** and **M,** Flow cytometric analysis of disaggregated lamina propria (*LP*; Fig 1, *L*) and PPE-pulsed mesenteric lymph node cell cytokines (Fig 1, *M*). *EtOH*, Ethanol. $*P \leq .05$ and $**P \leq .01$.

subcutaneous priming and directly administered multiple adjuvant-free intragastric CPH gavages (see Fig E3, *C*, and the Methods section in this article's Online Repository). DR but not raw CPH elicited anti-peanut IgG_1 and functional IgE responses (Fig 1, *F-H*, and see Fig E5 in this article's Online Repository at www.jacionline.org).