serve as a useful learning and assessment tool to enhance dermatology resident education.

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Family Risk Discussions After Feedback on Genetic Risk of Melanoma

First-degree relatives (FDRs) of patients with melanoma may be among the first to seek melanoma genetic risk information.1 Testing may subsequently prompt useful discussions regarding melanoma risk, early detection, and prevention with multiple family members. To explore this potential, this study examined the intended discussions of FDRs of patients with melanoma with diverse family members after receiving hypothetical melanoma genetic risk information.

Methods | The study was approved by the institutional review board of the Memorial Sloan Kettering Cancer Center. The study used a 3-by-2 experimental pre-post design, where feedback type and risk level were varied and participants were randomized to 1 of the 6 scenario conditions.2 For feedback type, “mutation feedback” was modeled on inherited mutations in CDKN2A (gene encoding p16INK4A) linked to hereditary melanoma.3 “Gene environment feedback” was modeled on the melanocortin receptor gene (MC1R), which interacts with sun exposure to heighten population melanoma risk.4 “Nongenetic feedback” was based on a nongenetic melanoma risk assessment that includes factors such as mole number.5 Risk level was varied by whether the findings were positive (test identified higher risk) or negative. All scenarios explicitly reminded participants of their increased risk due to family history regardless of test findings.

Assessment of family discussions at baseline (before scenario exposure), asked how much (Not at all to A lot) participants had spoken about melanoma risk with their (a) mother, (b) father, (c) sister(s), (d) brother(s), (e) children, and (f) grandchildren (if they currently had this relative). Assessment at follow-up (after scenario exposure), asked how much participants intended to speak about melanoma risk with each family member.

The McNemar test of change in proportions was used to test pre-post changes in discussion rates. A generalized estimating equation (GEE) model was used to account for the clustered data of multiple family members per participant and to examine the extent to which the experimental manipulations influenced intention to discuss melanoma risk at follow-up.

Results | The sample (N = 139) was mostly female (n = 97 [70%]) and non-Hispanic white (n = 135 [97%]). The median age was 48 years; most patients had only 1 FDR with melanoma (n = 128 [92%]) and no personal melanoma history (n = 110 [79%]). Approximately half (n = 76 [55%]) had a sun-sensitive phenotype (skin type I/II, indicating skin prone to burning). Baseline discussion rates did not differ by experimental conditions (P > .05 for all).

At baseline, frequency of melanoma risk discussions across all family member types was higher, on average, among women than men ($$t_{35.47} = 2.34, P < .05$$), but did not differ based on whether they had 1 or more FDRs with melanoma, whether they had a personal history of melanoma, or whether they had a sun-sensitive phenotype (skin type I/II) or not (P > .05 for all). The GEE model-estimated intentions were higher if the participant received positive (n = 128 [92%]) rather than negative (n = 100 [72%]) feedback ($$\chi^2 = 11.98, P = .001$$). There were no significant differences by feedback type, nor a significant interaction (risk level by feedback type). As reported in the Figure, discussion with all family members increased signifi-
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**OBSERVATION**

**Coexistence of Staphylococcal Scalded Skin Syndrome and Acute Graft-vs-Host Disease**

We report a case of adult staphylococcal scalded skin syndrome (SSSS) superimposed on acute graft-vs-host disease (GVHD) and discuss the unique histologic features of both conditions seen on skin biopsy.

**Report of a Case** | A man in his 60s with a history of myelodysplastic syndrome underwent a matched unrelated donor hematopoietic stem cell transplant (HSCT) after conditioning with fludarabine and total-body irradiation. Prophylaxis against GVHD included cyclosporine A, 175 mg, and mycophenolate mofetil, 1000 mg, both given orally twice daily. On day 35 after HSCT, the patient developed nontender erythematous macules and papules on the thighs, which subsequently spread to the trunk and extremities. The patient later developed diarrhea and mildly elevated serum total bilirubin levels (1.5 mg/dL). Skin and colon biopsies confirmed acute GVHD, and the patient was treated with intravenous methylprednisolone, 200 mg/d, with resolution of symptoms.

On day 144 after HSCT, the patient developed diarrhea and mildly tender diffusely scattered erythematous macules and papules on the trunk, neck, face, and extremities, along with acute kidney injury (creatinine, 2.94 mg/dL). Owing to a recent fever, the patient began treatment with intravenous li-