Phenotypic and Genotypic Analyses of Genetic Skin Disease through the Online Mendelian Inheritance in Man (OMIM) Database


In the publication by Feramisco et al., Figure 3 is incorrect; please see below for Figure 3 in its entirety. In addition, some deletions to the text were not implemented at the time of proofs. These are detailed below. The authors regret the errors.

Figure 3. Skin findings associated with the genetic skin diseases. (a) Ranking of cutaneous features by dermatological phenotype. The CGenDerm phenotypic subclass is shown on the X-axis, and the absolute number of disease units exhibiting each subclass is shown on the Y-axis. (b) Prevalence of cutaneous features is shown in clusters of classes and subclasses. (c) Distribution of cutaneous features among discrete skin disease units. Most genetic skin diseases have a single dominant cutaneous feature; however, there are many diseases with multiple distinct cutaneous features. The number of cutaneous features is shown on the X-axis.
1. In the abstract, the sentence reading “The most common elemental skin features included cornifying, erosive and hair/nail phenotypes while the most common systemic features included those associated with developmental, musculoskeletal, and neurological systems’ should be corrected to: “The most common elemental skin features included hair/nail phenotypes while the most common systemic features included those associated with developmental, musculoskeletal, and neurological systems.”

2. In paragraph 2 of Results, the third sentence (“Individually, ichthyosis/scaling (group 1D) and other abnormal features of cornification (group 1B) represent the most common cutaneous features in genetic skin disease.”) should have been deleted.

3. In paragraph 2 of Results, the fourth sentence should read: “Hair/nail abnormalities represent the largest group of cutaneous features.”

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**The Overexpression of Keratin 17 as a Putative Psoriasis Autoantigen Is Coupled to Hyperproliferation of Keratinocytes due to Interleukins 22 and 23 in vitro**


**Correction to:** *Journal of Investigative Dermatology* (2009) **129** (Suppl 2); S99 (abstract 589)

In the publication by Böckelmann, there is an error in the authorship and affiliation. The correct authors and affiliation for this abstract are: R. Böckelmann and B. Bonnekoh; Clinic for Dermatology, Otto-von-Guericke-University, Magdeburg, Germany. The authors regret the error.

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**Injury Is a Major Inducer of Epidermal Innate Immune Responses during Wound Healing**


**Correction to:** *Journal of Investigative Dermatology* (2009). E-pub ahead of print 3 September 2009. doi:10.1038/jid.2009.284

In the publication by Markus Roupé et al., the bottom row of Figure 1 (lactoferrin staining) was inadvertently omitted. The figure is reproduced here in its entirety. The authors regret the error.

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![Figure 1](image.jpg)

**Figure 1.** AMP expression increases over time in skin wounds *in vivo*. Samples of normal skin and of wounds 2, 3, and 4 days old were immunostained for SLPI, hBD-2, elafin, and lactoferrin (LF). Normal skin was obtained by punch biopsy. New biopsies of the wound samples were taken on days 2, 3, and 4 around the edges of the initial biopsy. Color was developed with Vulcan Fast Red Chromogen, and Harris hematoxylin was used for counterstaining. Bars = 100 μm (black).