cytes, or natural killer cells (Roark et al., 2008).

Taken together, our results indicate that Th17 and IL-17 pathway are active in BD patients, and has an important role particularly in acute attacks of the disease. Neutrophil activity is increased in BD, and the affected organs show a significant neutrophile infiltration. Our data suggest that this result might be caused by increased IL-17A response in patients with BD. Treatment modalities attempting to evaluate novel approaches to eliminate the overactivity of IL-17A and/or IL-23-Th17 pathway may clarify the biological importance of IL-17A and Th17 cells in patients with BD.

Our study, however, had also some limitations. It is wise to keep in mind that the increased IL-17A serum levels is not clinical sign-specific and even not disease-specific; it indicates inflammation during the acute phases of the disease with involvement of neutrophils. It might have been of more scientific value to investigate longitudinal evaluation of IL-17A and other cytokine (IL-6, IL-8, tumor necrosis factor-α, and so on) serum levels in a group of patients with active and inactive periods of the disease.

CONFLICT OF INTEREST
The authors state no conflict of interest.

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Human β Defensin-2: Too Good to Be Dismissed in Atopic Dermatitis

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TO THE EDITOR
I read with interest the study by Harder et al. (2010), as they confirmed the previous finding that the human β defensin-2 (hBD-2) level in atopic dermatitis (AD) lesions is lower than that in psoriatic lesions, but higher than healthy skin (Ong et al., 2002).

Based on the observation that psoriasis patients rarely suffer from skin infections (Henseler and Christophers, 1995), Harder et al. (1997) made their seminal discovery of hBD-2 in psoriatic lesions. This antimicrobial peptide (AMP) has since been proven to be the strongest and most psoriasis-specific protein (de Jongh et al., 2005; Gambichler et al., 2008). In addition, it has been shown to have immunomodulatory effects on innate and adaptive immunity (Yang et al., 1999). Although hBD-2 has weak direct antimicrobial activity against Staphylococcus aureus (Harder et al., 1997), the amount of hBD-2 present in psoriatic lesions significantly exceeds the amount required for both antimicrobial effects (2.5–25 μM) and immunomodulation (2.5–250 nM) (Jansen et al., 2009). On the other hand, the amount of hBD-2 present in AD lesions, although higher than that in healthy skin, has not been proven to exceed these ranges. Furthermore, correlation between hBD-2 level and clinical infection in AD has not been studied. However, the findings of Harder et al. 2010.
TO THE EDITOR

Bogh et al. (2010) recently found that the increase in 25-(OH)-hydroxycholecalciferol (25-(OH)-D) after UV-B exposure in 28 non-sun-worshippers was positively correlated with baseline total cholesterol levels. In their discussion of this interesting observation, the authors cite from a biochemistry textbook:

The synthesis of vitamin D starts in the bowel epithelial with the oxidation of cholesterol from food or bile to pro-vitamin D$_3$ (7-dehydrocholesterol), which is then transported to the skin, mainly the epidermis, wherein it is isomerized to pre-vitamin D$_3$ (cholecalciferol) by UVB radiation (Champe et al., 2005).

A similar statement (regarding the formation of 7-dehydrocholesterol in the intestine and its transport to the skin) is on page 732 in the most recent (10th) edition of a German Pharmacology textbook (Flockerzi, 2009). Although we have no access to the cited textbook of Champe et al. (2005), in the textbook by Rang et al. (1995), one can find on page 449:

Cholecalciferol (D$_3$) generated in the skin from 7-dehydrocholesterol by the action of ultraviolet radiation, the 7-dehydrocholesterol having been formed from cholesterol in the wall of the intestine.

A biochemistry textbook (Campbell and Farrell, 2009) shows in Figure 8.30