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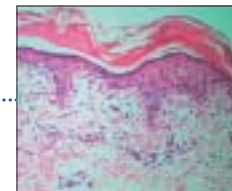
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## Vitiligo, psoriasis and imiquimod: Fitting all into the same pathway

Sir,

I read with interest the letter describing imiquimod-induced vitiligo-like depigmentation.<sup>[1]</sup> There are other similar case reports<sup>[2,3]</sup> and 68 reports of pigmentary changes related to imiquimod use listed by FDA.<sup>[4]</sup> The most widely accepted theory is that imiquimod activates the cell-mediated arm of the immune system via the stimulation of multiple pro-inflammatory cytokines which up-regulates a Type-1 cell response (Th1) that unmasks an innate predilection for the development of vitiligo.<sup>[5]</sup>

The mode of action of imiquimod involves activation of Toll-like receptors (TLR) 7 and 8, which initiates a signaling cascade leading to the production of pro-inflammatory cytokines like IFN- $\alpha$  and TNF- $\alpha$ . However, TLR 7 and 8 also initiates ubiquitin-mediated proteolysis and apoptosis through the myeloid differentiation factor 88-dependent pathway as evident from the pathway map<sup>[6]</sup> hsa04620 available from KEGG.<sup>[7]</sup> In a previous letter to *IJDVL*, I presented the genomic evidence for ubiquitin-mediated melanocyte-specific apoptotic process in vitiligo.<sup>[8]</sup> Hence, the ubiquitin-mediated proteolysis may be important in the pathogenesis of vitiligo-like lesions following treatment with imiquimod.

A Th1 type response with increased TNF- $\alpha$  is vital in the pathogenesis of psoriasis.<sup>[9]</sup> Hence, it is hardly surprising that there are several reports of imiquimod-induced psoriasis.<sup>[10-13]</sup> Psoriasis is a proliferative disorder while vitiligo is an apoptotic disorder. It is interesting to note that these disparate conditions converge at a fundamental pathway level.

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