Therefore, to study the potential function of FcRn in pemphigus in human keratinocytes, we
the absence of new lesions and circulating antibodies. Usually over than 2 years with a close
treatment. Adjuvants therapy with azathioprine (93 cases), disulone (22 cases), cyclophosphamide (1
case), methotrexate (9 cases), intralesional corticosteroids (13 cases), azathoprine and rituximab (3 cases).
Clinical remission (133 cases) (mean duration 2.5 months), lost to follow-up (39 cases); relapses (89 cases) (mean duration 51 months), death (33
cases). In our series, no statistical differences have been noted concerning the duration of the
treatment between oral steroids only and the association with a immunosuppressive agent.
For economical reasons azathioprine is the most used sparing agent. The duration of treat-
ment for pemphigus is a progressive targeted treatment with steroids. The indications to
discontinue treatment in a patient with clinical remission on low doses of oral steroids is
the absence of new lesions and circulating antibodies. Usually over than 2 years with a close
monitoring of drug side effects.

Excision of halo nevus without PD-L1 expression may cure vitiligo
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Halo nevus is also called a skin pigmented naevus, is a nevus cell nevus that is surrounded by vitiligo
and is caused by a T-cell mediated immune response to the nevus antigen. Excision of nevus is
recommended to prevent progression to vitiligo vulgaris. Vitiligo vulgaris may occur around the skin.
Lesions, maculopapulon, hypopigmented, sebaceous, etc, it’s called Suttor’s lesion because of its appearance.
Especially, vitiligo in melanoma patients is called “melanoma associ-
ated vitiligo” and recognized to associate with favorable outcomes. It’s sometimes observed in patients who respond to immune checkpoint inhibitor (ICI) therapy as a “preference” im-
mune-related adverse event (irAE), the mechanism has attracted attention. However, there are few reports on analyses of immune checkpoint including PD-1/PD-L1 in the present.

In this study, to evaluate the relationship between immune checkpoint including PD-1/PD-L1 and VN, we performed immunomodulation analysis using formalin fixed paraffin embedded tissues (FFPE) samples collected from 38 VN patients (16 males and 22 females, mean age 20-47 years) whose nevus were resected in Nagoya City University Hospital. Of the 29 cases that we were able to follow after nevus excision, 12 cases showed improvement of the surrounding vitiligo and 17 cases showed no improvement or expansion of vitiligo. 25 cases have vitiligo vul-
garis, and 10 of them showed improvement of vitiligo vulgaris after VN excision. Immuno-
histochemical findings showed that PD-L1 expression was positive in 25 of 36 cases, and cases with improved vitiligo after VN excision were predominantly PD-L1 positive. PD-L1 expression in infiltrating cells was positive in 19 of 38 patients, but there was no significant correlation between PD-L1 expression and improvement of vitiligo. These results suggest that more than local immune responses are involved in vitiligo formation in PD-L1-positive VN, and the analysis of immune responses in PD-L1-positive VN may lead to elucidation of the
mechanisms of tumor immunity and irAEs.

Impact of Staphylococcus aureus colonisation on barrier function and cytokine profile in atopic dermatitis skin
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dermatology team, University of Bordeaux, Bordeaux, France, 3 Dermatology Department, University Hospital Hospital Ibn Sina Rabat Morocco, Rabat, Morocco, 4 Department of Dermatology and Pediatric Dermatology, National Reference Center for Rare Skin Diseases, Hôpital Saint-André, Bordeaux, France.

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by a dysregulated immune response associated with barrier dysfunction and dysbiosis of the skin microbiota characterized by Staphylococcus aureus colonization in lesional skin. The aim of our study was to compare non-lesional and lesional AD skin transcriptional profile depending on staphylococcal colonization. Skin biopsies were analyzed by RT-qPCR in moderate and se-
vere adults AD patients. Staphylococcus epidermidis was detected in all lesional skin, in greater abundance than in non-
lesional skin, while S. aureus colonization was found in approximately 50% of AD patients. Our results suggest a greater decrease of gene expression for SARM1 (Gene ID: 81249) and C3 (Gene ID: 6700), in lesional skin colonized by S. aureus. Interestingly, up-regulation of chemokines and cytokines involved in AD pathogenesis (including IL13, FAP, CCL17, and CCL18) in lesional skin was not impacted by S. aureus colonization, excepted for CCL22 expression which was weaker in the presence of S. aureus colonization. These findings suggest a role of S. aureus in barrier dysfunction and inflammatory response in AD lesional skin.

Dysfunctional Collagen XVII provokes skin inflammation
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Bullous pemphigoid (BP) is an autoimmune subepidermal blistering skin disease where au-
toantibodies are developed against BP180 (also known as collagen XVII, colXVII). Our group has previously developed a mouse model of BP180 autoimmune disease in a (Bv12 ×1D2B)F1 mixed background. In this model, the expression of colXVII in lesional skin is significantly reduced in BP180 mice in comparison to healthy wildtype (WT) and non-lesional skin. The objective of this study was to determine whether the absence of colXVII in lesional skin promotes inflammation in BP180 mice.

We established a mixed Bv12 ×1D2B-BP180 autoantibody transgenic mouse model (BP180 
4/4) and compared it with WT littermates. Lesional skin was collected from WT and BP180 mice showing DIF+ dermatitis. Lesional skin was analyzed by qPCR for various markers of inflammation and inflammatory mediators, including IL-1α, IL-1β, IL-4, IL-6, IL-17A, TNF-α, and MMP-12. Additionally, lesional skin was analyzed by immunohistochemistry for CD3 and CD68, which are markers of T-cell and macrophage infiltration, respectively.

Compared to WT littermates, BP180 mice showed significantly increased expression of IL-1β, IL-1α, IL-6, IL-17A, TNF-α, and MMP-12 in lesional skin. Furthermore, BP180 mice had increased T-cell infiltration as determined by CD3 immunohistochemistry. These findings suggest that the absence of colXVII in lesional skin may contribute to the development of skin inflammation in BP180 mice.

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