Normal structures for epidermal and dermo-epidermal adhesion

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The essential function of the skin is a barrier against the outside. Adhesion molecules are required for integrity of skin. Autoimmune blistering skin diseases are characterized by the production of autoantibodies directed against adhesive structures of the skin. These organ specific autoimmune diseases included pemphigus in which autoantibodies target proteins of the desmosomal complex, and subepidermal autoimmune diseases characterized by autoantibodies directed against structural proteins of the dermoepidermal junction.

The desmosome is the main structure allowing the cohesiveness of the keratinocytes. Among the desmosomal cadherins (desmogleins and desmocollins) that mediate epidermal cell–cell adhesion, it has been demonstrated that desmoglein 1 and desmoglein 3 are the autoantigens of pemphigus foliaceus and pemphigus vulgaris. Paraneoplastic pemphigus is associated with autoantibodies directed against the desmosomal plaque protein, desmplakin. The anchoring complex of dermoepidermal junction consists of hemidesmosomes, anchoring filaments and anchoring fibrils. Of the constituents of hemidesmosomes, the plaque protein, BP230 (BPAG1), and transmembrane protein, BP180 (BPAG2, type XVII collagen), are the autoantigens of bullous pemphigoid. Subepidermal autoimmune blistering diseases target proteins beneath hemidesmosomes, for example laminin 5 (cicatricial pemphigoid), ladinin (LAD-1; linear IgA disease) and type VII collagen(anchoring fibril; epidermolysis bullosa acquisita). Recent insight of normal structure of epidermal and dermoepidermal adhesion will be give us better understanding of the pathogenesis and development of diagnostic tools and treatment targets of autoimmune blistering diseases.
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