CDB SEMINAR

Speaker: Masayuki Amagai

< Department of Dermatology, Keio University School of Medicine >

Title: "Cadherins as targets in skin autoimmune and infectious diseases"

Date: Thursday, February 26
Time: 216:00 -17:00
Place: 7th floor Conference Room of Building A,CDB

Summary:

Desmogleins (Dsgs) are desmosomal cadherins which play an important role in holding keratinocytes together to maintain the tissue integrity of epithelia. Dsgs are now known to be targeted in skin autoimmune disease, pemphigus, and skin infectious disease, staphylococcal scalded skin syndrome and bullous impetigo. In pemphigus IgG autoantibodies are directed against Dsg1 and Dsg3 and cause blisters and erosions in the skin and mucous membranes. Patients with pemphigus show complex clinical pictures, but their clinical and microscopic localization of blisters is now explained by a simple logic, desmoglein compensation theory. As an extension of this theory, the molecular mechanism of staphylococcal exfoliative toxin (ET) which causes staphylococcal scalded skin syndrome and bullous impetigo has been unveiled 30 years after the discovery of the toxin. ET is a serine protease which specifically binds and cleaves Dsg1. To explore molecular and cellular mechanisms of harmful IgG autoantibody production, an active disease model for pemphigus is generated by a unique approach using autoantigen knockout mice. Adoptive transfer of Dsg3-/- lymphocytes to mice expressing Dsg3 induces stable anti-Dsg3 IgG production with development of the pemphigus phenotype. Stories that come in sight through studies on desmogleins in skin diseases will be discussed.

Host: Shin-Ichi Nishikawa <Stem Cell Biology, CDB>
E-mail: nishikawa@cdb.riken.go.jp Tel: 078-306-1893
RIKEN Center for developmental Biology http://www.cdb.riken.go.jp/