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**Impaired function of EC1 underlies the pathological disorganization of vascular and basement membrane molecules in lichen sclerosus**

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**Introduction:** Lichen sclerosus (LS) is an acquired inflammatory condition that mainly affects genital skin. Immune cells to pathogenecis and extracellular matrix (ECM) abnormalities play a significant role in the pathology of LS. In this study, we investigated the expression of vascular and basement membrane molecules in LS tissue and compared them with normal skin (NC).

**Materials and Methods:** We collected skin biopsies from 33 patients with LS and 23 NC subjects. Immunohistochemical analysis was performed using antibodies against vascular and basement membrane molecules, including von Willebrand factor (vWF), CD31, and laminin 5 (Lam5). Quantitative analysis was conducted using image analysis software.

**Results:** In LS, vWF and CD31 expression was significantly decreased compared to NC. However, Lam5 expression was increased in LS. Furthermore, the ratio of Lam5 to vWF was significantly higher in LS than in NC.

**Conclusion:** The impaired function of EC1 underlies the pathological disorganization of vascular and basement membrane molecules in LS, suggesting a novel therapeutic target for this condition.

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**Differential diagnosis between skin conditions and patients with chronic spontaneous urticaria based on the ability of anti-IgE autoantibodies to induce FceRI cross-linking**

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**Introduction:** Chronic spontaneous urticaria (CSU) is defined as the occurrence of systemic daily wheals for at least 6 weeks. Since the presence of IgG autoantibodies (Abs) to FcεRI cross-linking anti-IgE Ab (CSU Ab) has been repeatedly observed in patients with CSU, autoimmunity is thought to be one of the major causes of CSU. The reported prevalences of IgG autoantibodies to FcεRI and IgE in sera from patients with CSU have varied, and these Abs are also often observed in healthy control subjects. Thus, we sought to determine the prevalence and FcεRI cross-linking ability of these Abs in a large number of patients with CSU and nonatopic control (NC) subjects.

**Materials and Methods:** We designed experiments to compare the concentrations of anti-IgE and anti-FcεRI Abs and the abilities of these Abs to cause FcεRI aggregation in patients with CSU (n = 134) and NC (n = 55) using ELISA and an in vitro elicitation test, respectively.

**Results:** We demonstrated that the concentration of anti-IgE Abs was significantly different between the NC subjects and the CSU patients (P < 0.0001, cutoff value: 0.558 ng/mL), whereas the concentration of anti-FcεRI Abs was not. A significant difference in the duration of illness was noted between patients with lower and those with higher concentrations of anti-IgE Abs relative to the cutoff value. The anti-IgE Abs, but not anti-FcεRI Abs, were in patients with CSU.

**Conclusion:** The study demonstrated the involvement of anti-IgE Abs in the pathogenesis of CSU in the Japanese population.

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**Inhibitory effect of kaempferol on skin fibrosis in systemic sclerosis by the suppression of oxidative stress**

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**Introduction:** Systemic sclerosis (SSc) is a chronic connective tissue disorder characterized by fibrosis and inflammation, and oxidative cellular damage. Administration of kaempferol might be an effective approach to reducing these damages.

**Materials and Methods:** We investigated the effect of kaempferol on fibroblast activation and oxidative stress in sclerotic skin. Kaempferol was administered in vivo to bleomycin-treated mice. Furthermore, the oxidant-induced intracellular accumulation of reactive oxygen species (ROS) in SSc fibroblasts was inhibited by kaempferol treatment. In sclerotic skin, kaempferol also reduced bleomycin-induced TUNEL+ apoptotic cells in the lesional skin of bleomycin-treated mice. These results suggest that the use of kaempferol as a potential treatment for SSc.

**Conclusion:** Our study demonstrated the inhibitory effect of kaempferol on oxidative stress in bleomycin-treated mice and SSc fibroblasts.

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**Phenotypic Changes of a Monocyte Cell Line under the Culture Temperature**

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**Introduction:** Monocytes are a heterogenous population of cells with diverse phenotypes. The temperature at which monocytes are cultured can affect their phenotype and function.

**Materials and Methods:** We used the THP-1 cell line, which is a human monocyte cell line, to investigate the effects of culture temperature on monocyte phenotype.

**Results:** We observed that THP-1 cells cultured at 37°C exhibited a higher expression of the monocyte marker CD14 compared to cells cultured at 29°C. Furthermore, the expression of the M1 macrophage marker CD80 was higher in cells cultured at 29°C than at 37°C. This suggests that culture temperature can affect the phenotypic characteristics of monocytes.

**Conclusion:** Our findings highlight the importance of considering culture temperature in studies involving monocyte phenotypes.

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**T cell induction of IL-10-producing plasmablasts during contact hypersensitivity**

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**Introduction:** Contact hypersensitivity (CHS) is a type IV delayed-type hypersensitivity reaction mediated by T cells. Th17 cells are a subset of T cells that produce pro-inflammatory cytokines, including IL-17A and IL-22. IL-10 is a cytokine that promotes Treg cell differentiation and suppresses immune responses.

**Materials and Methods:** We used an ear sensitization model to study CHS and the induction of IL-10-producing T cells. IL-10Venus+ T cells were identified in the ear tissue of sensitized mice.

**Results:** We observed that the number of IL-10Venus+ T cells was significantly higher in mice treated with IL-10KO mouse plasma compared to those treated with wild-type mouse plasma. This suggests that IL-10 production is essential for the induction of IL-10Venus+ T cells during CHS.

**Conclusion:** Our findings indicate that IL-10 production is crucial for the induction of IL-10Venus+ T cells during CHS.

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**Evolution of autoreactive B and T cells in pemphigus patients with Rituximab or corticosteroids regimen treatment**

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**Introduction:** Pemphigus is an autoimmune blistering disease mediated by autoantibodies (Abs) directed against desmogleins (Dsg). The current treatment options include corticosteroids, immunosuppressive agents, and targeted therapies such as Rituximab (RTX).

**Materials and Methods:** We analyzed the phenotype and antigen specificity of B cells and T follicular helper cells (TFH) in pemphigus patients treated with RTX or corticosteroids (CS).

**Results:** We observed that RTX treatment led to a significant decrease in the frequency of IgG-switched Dsg+ memory B cells compared to CS treatment. Additionally, the proportion of autoreactive TFH cells was much lower in patients treated with RTX than in those treated with CS.

**Conclusion:** Our findings suggest that RTX treatment may be a more effective treatment for pemphigus than CS, as it leads to a significant reduction in autoreactive B and T cell populations.