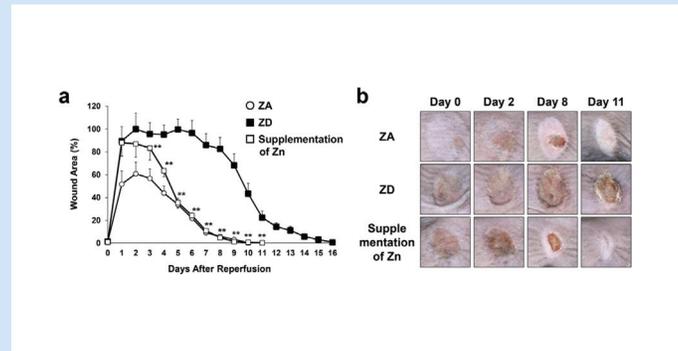


## Zinc deficiency exacerbates pressure ulcers by increasing oxidative stress and ATP in the skin

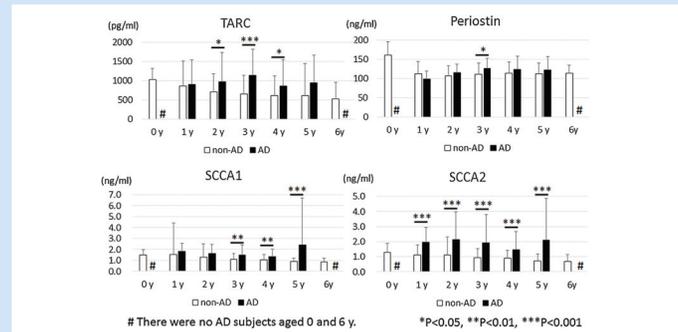
Zinc deficiency is believed to be a predisposing factor for the development and intractable healing of pressure ulcers (PUs); however, the mechanisms of this association have not been elucidated. Nakamura H et al elucidated the mechanisms of the formation of severe and prolonged PUs under the zinc deficiency condition. Dietary zinc deficiency caused the formation of severe and prolonged PUs in mice. Zinc deficiency increased the vascular disorder, oxidative stress, and apoptosis induced by cutaneous ischemia-reperfusion (I/R) injury. I/R injury-induced oxidative stress signals were significantly higher in zinc-deficient mice than in zinc-adequate mice. Additionally, zinc deficiency reduced the number of LCs and increased the amount of ATP in cutaneous I/R-injured skin. Zinc deficiency might increase cutaneous I/R injury-induced vascular damages, oxidative stress, and apoptosis, as well as ATP amount in I/R area due to the loss of LCs. These mechanisms might partly account for zinc deficiency-induced formation of severe and prolonged PUs.



**Fig. 6.** Oral supplementation of Zn improved Zn deficiency-associated PUs. (a) Percent wound area at each time points in ZA, ZD and Zn-supplemented mice relative to the wound area in ZD mice at 2 days after reperfusion ( $n = 10-12$  wounds for each time point and groups). All values represent the mean  $\pm$  SEM. \*\*\* $P < 0.01$ , \* $P < 0.05$ . (b) Representative images of the wound after cutaneous I/R in ZA, ZD and Zn-supplemented mice at 0, 2, 8 and 11 days after reperfusion.

## Serum squamous cell carcinoma antigen (SCCA)-2 correlates with clinical severity of pediatric atopic dermatitis in Ishigaki cohort

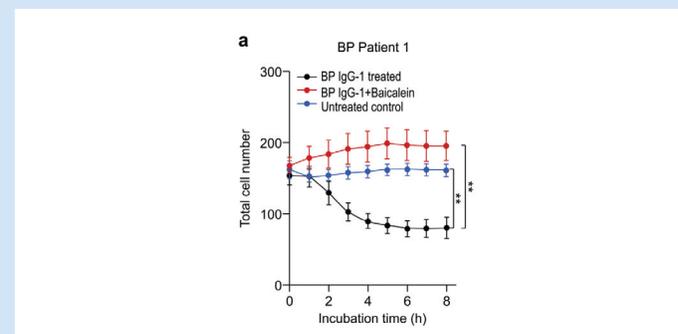
Recent case control studies have indicated the usefulness of squamous cell carcinoma antigens (SCCAs) in pediatric and adult AD. Relatively low prevalence and mild disease severity of pediatric AD are observed in our Ishigaki cohort presumably due to the moisturising subtropical climate, which could conversely mean possible higher allergic potential of non-AD subjects towards AD. Takeuchi S et al investigated the feasibility of using SCCAs together with TARC and periostin as biomarkers for pediatric AD even in the Ishigaki cohort. Serum SCCA1, SCCA2, periostin and TARC levels were all significantly increased in AD compared with those in non-AD, but only serum SCCA2 showed a significant increase in AD when assessed in each age group or in subgroup analysis. Among the biomarkers tested, serum SCCA2 also showed the best correlations with clinical AD severity and TARC and showed the best diagnosability for AD in ROC analysis. SCCA2 is a potent biomarker for pediatric AD in the Ishigaki cohort.



**Fig. 2.** Serum TARC, periostin, SCCA1 and SCCA2 as biomarkers for AD in each age group. Only serum SCCA2 was significantly increased in pediatric patients with AD at the different ages.

## Baicalein protects normal human epidermal keratinocytes against bullous pemphigoid immunoglobulin G-induced alteration

Bullous pemphigoid (BP) is a common autoimmune bullous dermatosis in the elderly, with immunoglobulin G (IgG) autoantibodies playing a major role in its pathogenesis. Kampo medicine has been used to ameliorate BP. Da Xet al determine whether baicalein, a major compound in Ou-gon, a crude drug used in Kampo medicine, protects normal human epidermal keratinocytes (NHEKs) against BP-IgG-induced damage. We found that BP-IgG induced cell detachment, interleukin (IL)-6 and IL-8 production, and intracellular ROS accumulation in NHEKs. In contrast, these BP-IgG-induced reactions were significantly prevented by baicalein in NHEKs. This study provides the evidences that traditional Kampo medicine containing baicalein might be efficacious against BP.



**Fig. 2.** (a) Effects of baicalein on BP IgG-induced cell detachment. NHEKs were treated with  $2 \text{ mg mL}^{-1}$  BP IgG in the presence or absence of  $100 \text{ }\mu\text{g mL}^{-1}$  baicalein (treatment time: 8 h). Normal IgG-treated (treatment time: 8 h) and untreated NHEKs were used as the controls. Data are presented as mean  $\pm$  standard error of three independent experiments; \*\* $p < 0.01$  and \*\*\* $p < 0.001$ .