

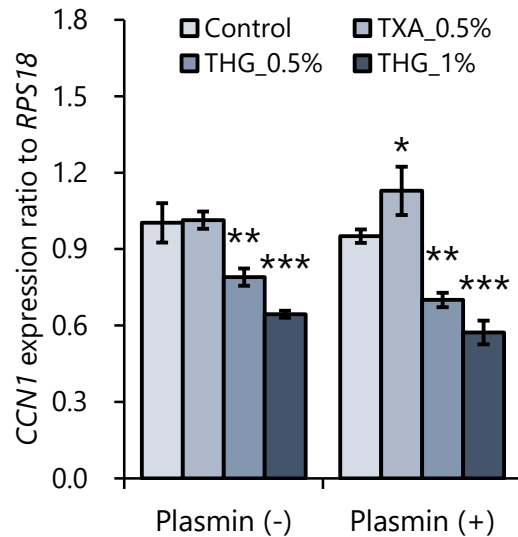
Trehangelins ameliorate inflammation-induced skin senescence by suppressing the epidermal YAP-CCN1 axis

Mami Yokota^{1*}, Yoshiyuki Kamiya¹, Tamie Suzuki¹, Shinsuke Ishikawa², Akira Takeda², Shinya Kondo¹, Takeshi Tohgasaki¹, Takuji Nakashima³, Yoko Takahashi⁴, Satoshi Ōmura⁴ & Tetsuhito Sakurai¹

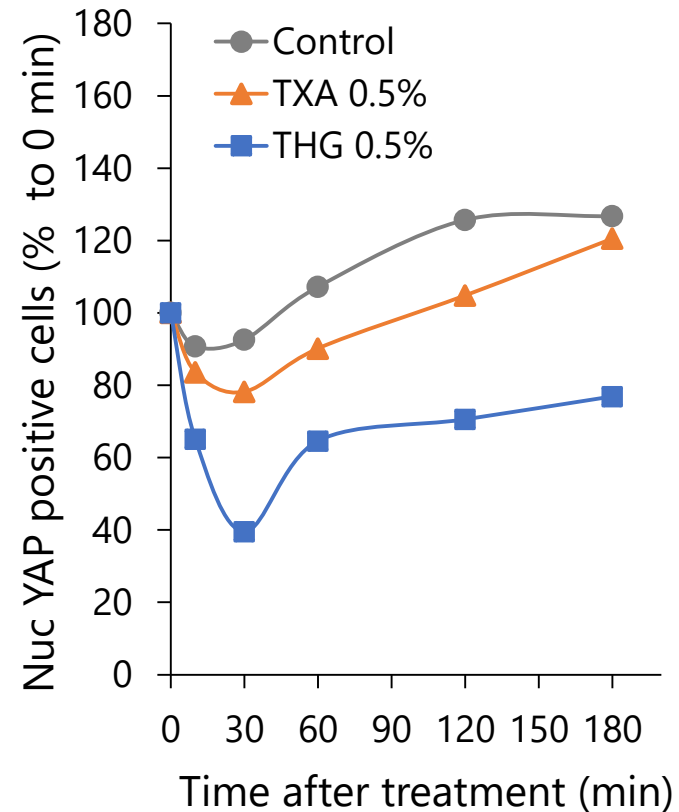
¹FANCL Research Institute, FANCL Corporation, 12-13 Kamishinano, Totsuka-ku, Yokohama, Kanagawa, Japan. ²Department of Plastic and Aesthetic Surgery, Kitasato University School of Medicine, 1-15-1 Kitasato, Minami-ku, Sagamihara, Kanagawa, Japan. ³Research Organization for Nano & Life Innovation, Waseda University, Wasedatsurumaki-cho, Shinjuku-ku, Tokyo, Japan. ⁴Ōmura Satoshi Memorial Institute, Kitasato University, 5-9-1 Shirokane, Minato-ku, Tokyo, Japan.

Supplemental data 1

S1. a



S1. b

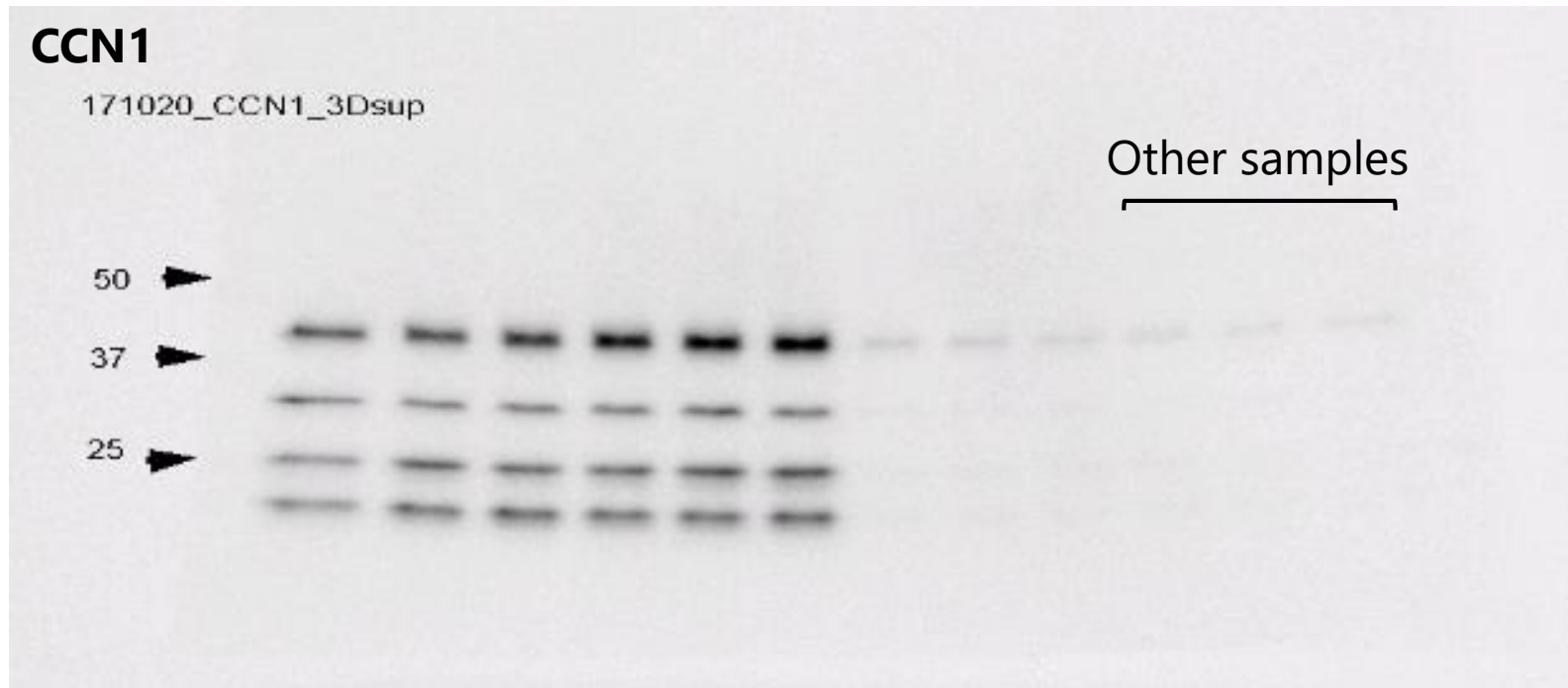


Supplemental Data

S1. Comparison of the mechanism of action between Tranexamic acid and THG. RNA samples were collected after 3 h incubation with plasmin and/or THG, Tranexamic Acid (TXA), a competitive inhibitor of plasmin. CCN1 expression was determined by qPCR. Values reported are means $\pm$ SD of n=3 replicates, Dunnett's test, *p<0.05, **p<0.01, ***p<0.001 (a). Immunocytochemistry of YAP and analysis of nuclear YAP-positive cells 0-3 h after treatment with TXA or THG. Values reported are means of n=10,000 cells (b). These analysis revealed that THG, but not TXA, significantly decreased the mRNA expression level of CCN1 after 3 h treatment.

Supplemental data 2

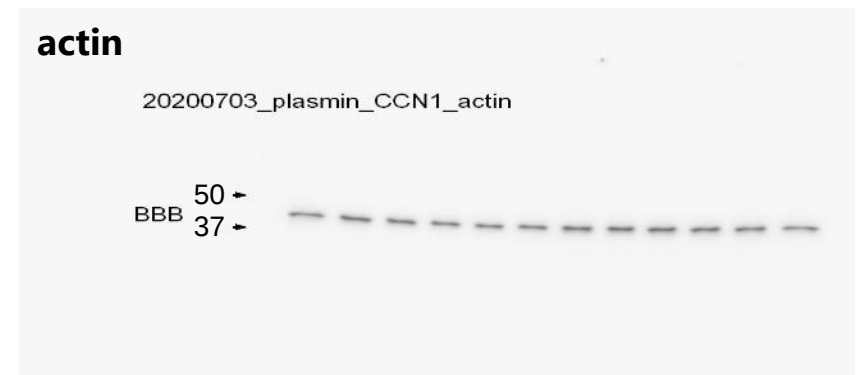
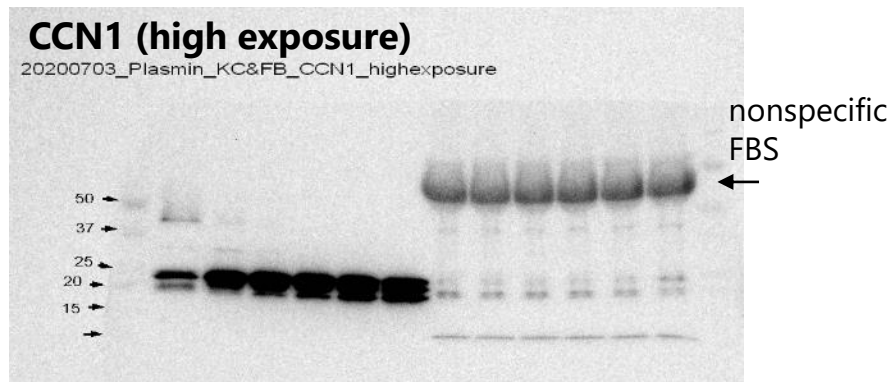
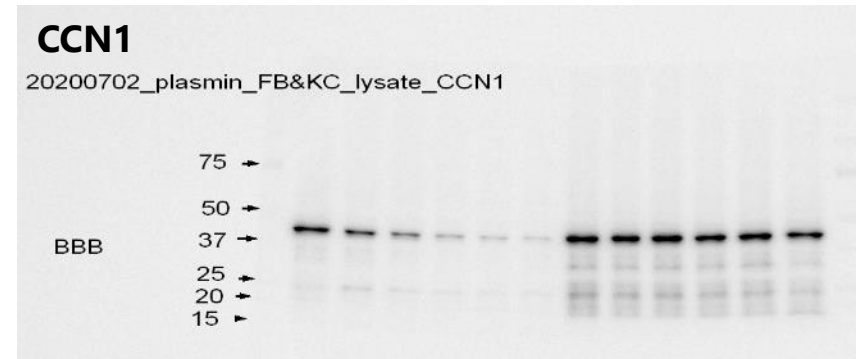
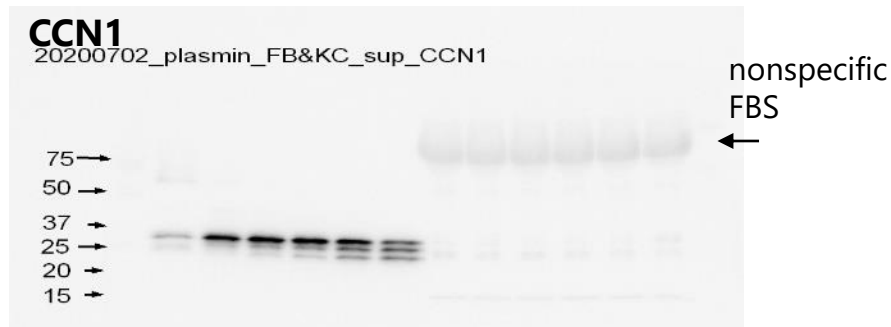
S2. a



S2. Full-length blots shown in the main text. CCN1 blot from Fig. 2b (a), 2f(b), 3a(c).

Supplemental data 2

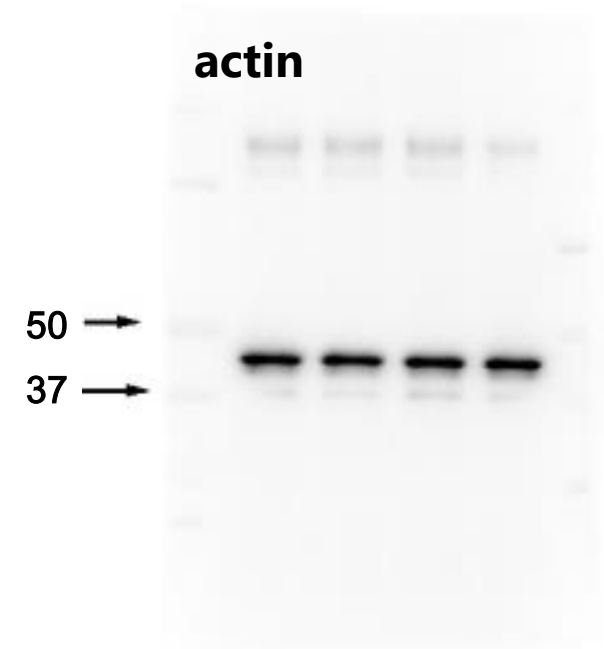
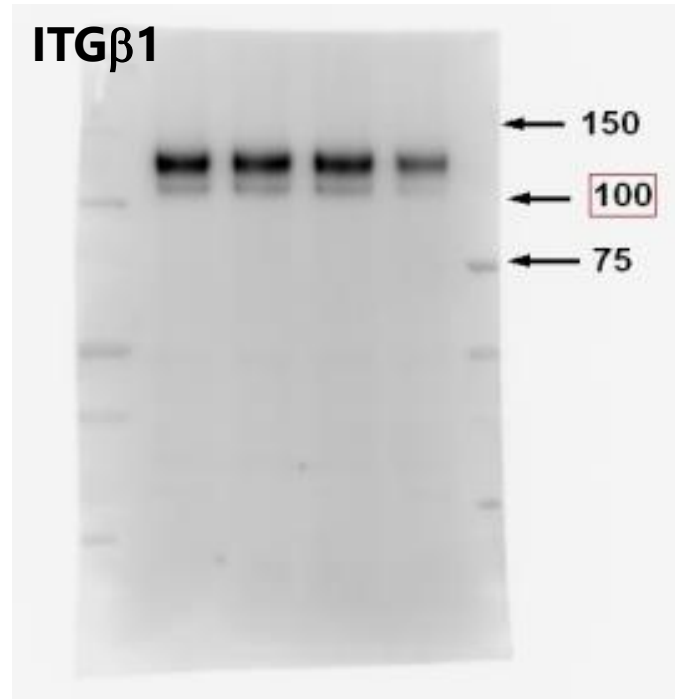
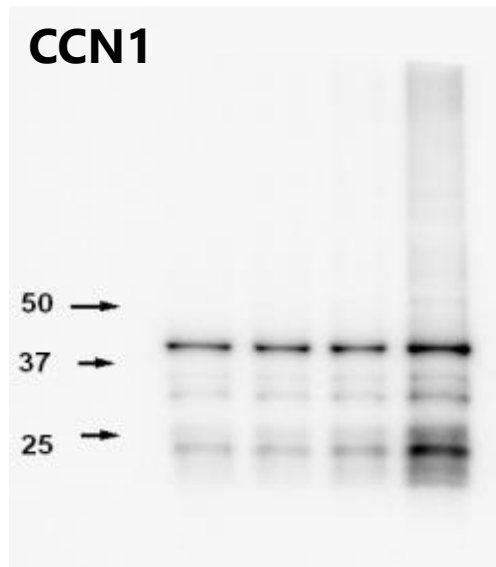
S2. b



S2. Full-length blots shown in the main text. CCN1 blot from Fig. 2b (a), 2f(b), 3a(c).

Supplemental data 2

S2. c



S2. Full-length blots shown in the main text. CCN1 blot from Fig. 2b (a), 2f(b), 3a(c).