Improved Human Skin Vitamin C Levels and Skin Function after Dietary Intake of Kiwifruit: A High-Vitamin-C Food

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TO THE EDITOR

Vitamin C (ascorbate) is found in all skin compartments. In vitro studies have indicated that it promotes collagen synthesis by fibroblasts, scavenges UVR-generated radicals, and stimulates keratinocyte proliferation and differentiation (Pullar et al, 2017; Sato et al, 2025; Wang et al, 2018). It is therefore assumed that boosting skin ascorbate levels will improve skin health and reverse the visible effects of aging. To this end, much attention is given to the topical application of ascorbate and to developing formulations to increase its stability in solution and penetration through the impermeable stratum corneum (reviewed in Enescu et al [2022]). However, in vivo delivery of ascorbate to the skin is tightly regulated by active transport from the plasma through the sodium-dependent vitamin C cotransporters SVCT1 and SVCT2 (Lykkesfeldt et al, 2025; Steiling et al, 2007).

Numerous nutritional intervention studies have monitored the effect of oral ascorbate intake on skin function (reviewed in Bertuccelli et al [2016] and Pullar et al [2017]) but without measurement of skin ascorbate content. Only one study has measured skin ascorbate after ascorbate supplementation (McArdle et al, 2002), and no studies have measured skin ascorbate content in association with disease states or with skin functions.

We have undertaken a comprehensive investigation to establish the relationship between plasma and skin ascorbate levels, with a focus on the major skin compartments. Skin tissue is predominantly dermis, which is mostly collagen bundles with fewer cells, with a very thin cellular epidermal layer (Figure 1a). In a cross-sectional study using skin and blood samples from healthy individuals undergoing elective surgery (Supplementary Materials and Methods and Supplementary Table S1), we found that dermal ascorbate closely reflected whole-skin levels, whereas epidermal levels were significantly higher (epidermal:dermal ascorbate mean = 2.0, range = 0.8–7.2) (Figure 1b). Approximately 20% of the ascorbate was present as dehydroascorbate (Figure 1c), probably generated during sample processing, despite our fast handling time (<15 minutes) and stringently optimized extraction process. Dehydroascorbate is typically barely detectable in cells and plasma. Whole-skin ascorbate was strongly positively correlated with dermal and epidermal ascorbate, and there was a positive correlation between dermal and epidermal ascorbate (Supplementary Figure S1a–c). Skin ascorbate levels varied considerably but were not related to age, sex, or sun exposure (Supplementary Figure S2a–c).

Because ascorbate is primarily concentrated into cells by the SVCTs (Lykkesfeldt et al, 2025), we standardized the measurements against the DNA content of each skin compartment, as a measure of cellularity. As expected, the epidermis contained ~11 times more DNA (ng/mg tissue) than the largely acellular dermis (Figure 1a and d). Using known parameters for the DNA content of human diploid cells (~6.5 pg DNA) and intracellular volume (2.6 μl/10^6 cells for fibroblasts) (Dall'Asta et al, 1994), we estimated that dermal cells, predominantly fibroblasts, contained 6.4 ± 2.6 (mean ± SD) mM ascorbate (range = 1.1–11.2 mM). In comparison, epidermal cells, predominantly keratinocytes, contained ~7-fold less (mean ± SD = 0.9 ± 0.5 mM, range = 0.3–1.9 mM) (Figure 1e). To our knowledge, this study provides a direct comparison of dermal and epidermal cellular ascorbate concentrations. Higher ascorbate concentrations in fibroblasts likely support collagen synthesis. Similar concentrations are reported for brain and adrenal cells, other known sites of ascorbate enzyme cofactor activity (Lykkesfeldt et al, 2025).

Ascorbate levels in all skin compartments were positively correlated with plasma levels, and we observed a significant positive linear relationship between epidermal and plasma ascorbate (Figure 1f–h).

To determine whether ascorbate supplementation increased skin ascorbate levels and improved critical skin functions, we undertook a pilot human dietary intervention study. Participants with low baseline plasma vitamin C status were recruited and supplemented with 2 SunGold kiwifruit for 8 weeks, delivering ~250 mg vitamin C daily (Carr et al, 2013) (Supplementary Materials and Methods, Supplementary Tables S2 and S3, and Supplementary Figure S2). The study was carried out at 2 locations owing to ethical considerations regarding skin sample collection. At site I, whole-skin biopsies were obtained for representative dermal levels. At site II, the suction blister method yielded epidermal tissue and blister fluid, and multiple skin function tests were performed (Supplementary Materials and Methods). Criteria for recruitment were similar at both sites (Supplementary Tables S2 and S3). At site I, plasma ascorbate status reached saturation

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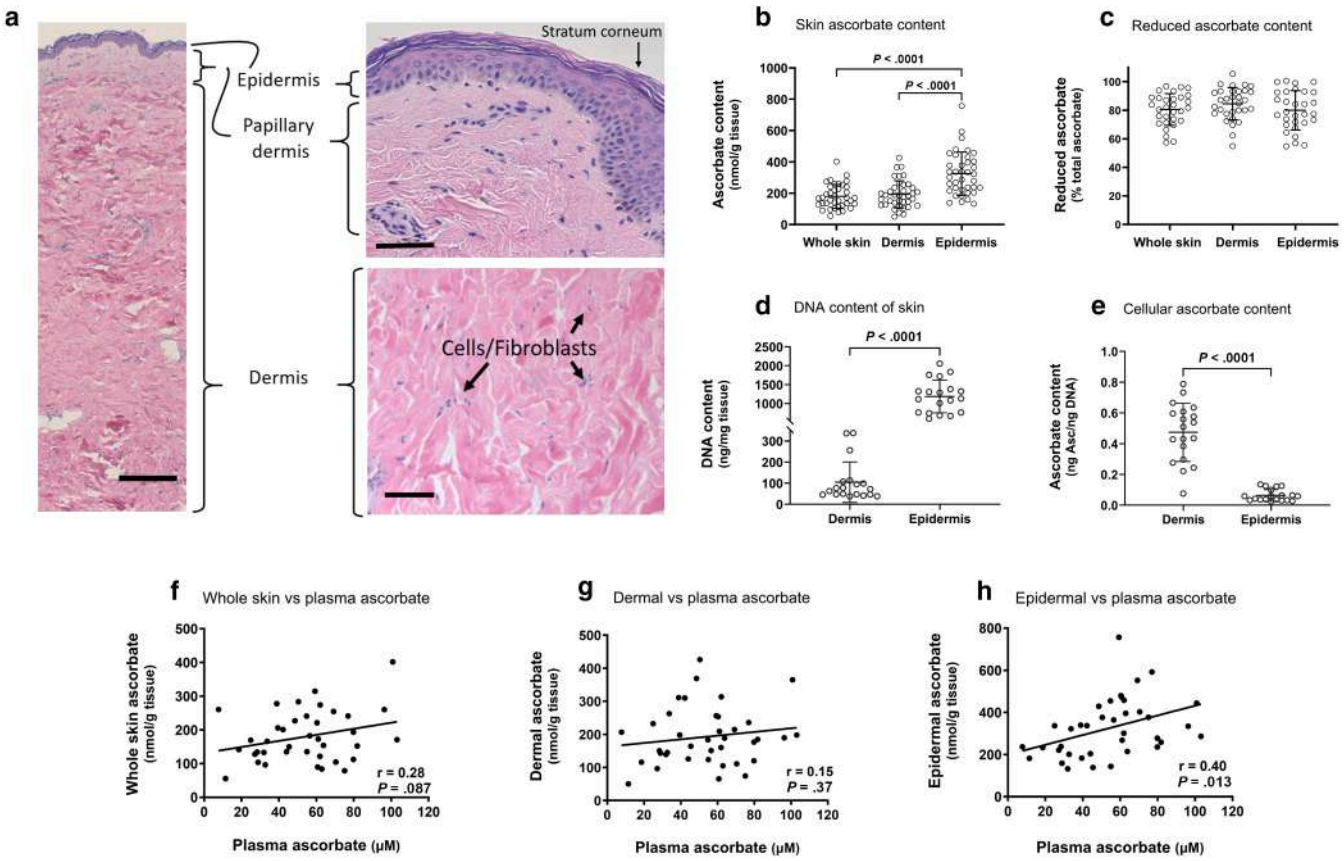

Figure 1. Skin vitamin C content by compartment and relationship to plasma ascorbate. The data are from cross-sectional analysis of skin samples obtained from 38 healthy individuals after elective surgery (Supplementary Table S1). (**a**) H&E-stained human skin samples (breast skin). Full-skin thickness is shown in the first image, highlighting the thin epidermal layer in comparison with the collagen-rich dermis. Higher magnification of epidermis and dermis as shown on the right illustrates the differences in cellularity of the skin compartments. Cell nuclei are stained blue, and the collagen bundles are stained pink. The layered strata of the epidermis are clearly visible, as is the papillary layer of the dermis. Bars shown = 200, 50, and 20 μm in left, top right, and bottom right images, respectively. **(b)** Ascorbate concentrations in whole skin (179 ± 76 nmol ascorbate/g tissue), dermis (194 ± 91 nmol ascorbate/g tissue), and epidermis (325 ± 138 nmol ascorbate/g tissue) are shown. Bars indicate the mean ± SD, with *P*-values derived from paired *t*-tests. **(c)** Ascorbate was measured with and without reduction of the samples with TCEP, allowing for measurement of total ascorbate, reduced ascorbate (minus TCEP measure), and DHA (difference between ± TCEP). Data are provided as means ± SD of n = 30–31. (**d**) DNA content of the dermis and epidermis. (**e**) Ascorbate content of dermis and epidermis, relative to DNA content (μg ascorbate/ng DNA). Bars indicate the mean ± SD with *P*-values derived from paired t-tests; n = 20. **(f–h)** Relationships between plasma ascorbate status and the ascorbate content of whole skin, dermis, and epidermis. Regression lines, *P*-values, and Pearson correlation coefficients are shown. DHA, dehydroascorbate; TCEP, tris(2-carboxyethyl)phosphine.

levels (>60 μM) after daily kiwifruit consumption, and whole-skin ascorbate increased in all participants (Figure 2a and b and Supplementary Table S4). At site II, plasma, blister fluid, and blister roof ascorbate levels increased significantly (Figure 2c–e). Participants with saturating plasma ascorbate status at baseline did not register a measurable increase (Figure 2c–e). A per-protocol analysis was therefore undertaken with participants whose plasma ascorbate concentration increased after intervention. Notably, changes in plasma, blister roof, and blister fluid ascorbate were strongly positively correlated (Figure 2f–h), indicating active uptake into the epidermis in response to variation in plasma levels.

Increased plasma ascorbate was associated with significant increases in skin density, from 0.154 ± 0.027 scanner units at baseline to 0.228 ± 0.035 after the intervention ($P < .0001$) (Figure 2i and Supplementary Table S4). Epidermal cell proliferation, measured by Ki-67 staining, also significantly increased from 21.06 ± 5.44 to 27.42 ± 4.96 intensity units ($P = .0091$) (Figure 2j and Supplementary Table S4). There was a small (∼7%), weak decrease in skin elasticity, no change in protection against UVA irradiation, and no change in the levels of procollagen type I peptides in interstitial blister fluid (Supplementary Table S4).

In summary, we have demonstrated that increasing dietary vitamin C intake boosts human skin ascorbate levels, with active uptake into the epidermis, which is particularly responsive to plasma ascorbate availability. Increased plasma ascorbate correlated with increased skin thickness and epidermal cell proliferation. These functional changes may reflect optimization of ascorbate-mediated support for collagen synthesis in tissue fibroblasts and for skin epidermal cell proliferation and differentiation through the promotion of TET-mediated gene transcription regulation, as recently demonstrated in vitro (Sato et al, 2025). We suggest that increasing dietary ascorbate intake will result in effective uptake into all skin compartments and will benefit skin function.

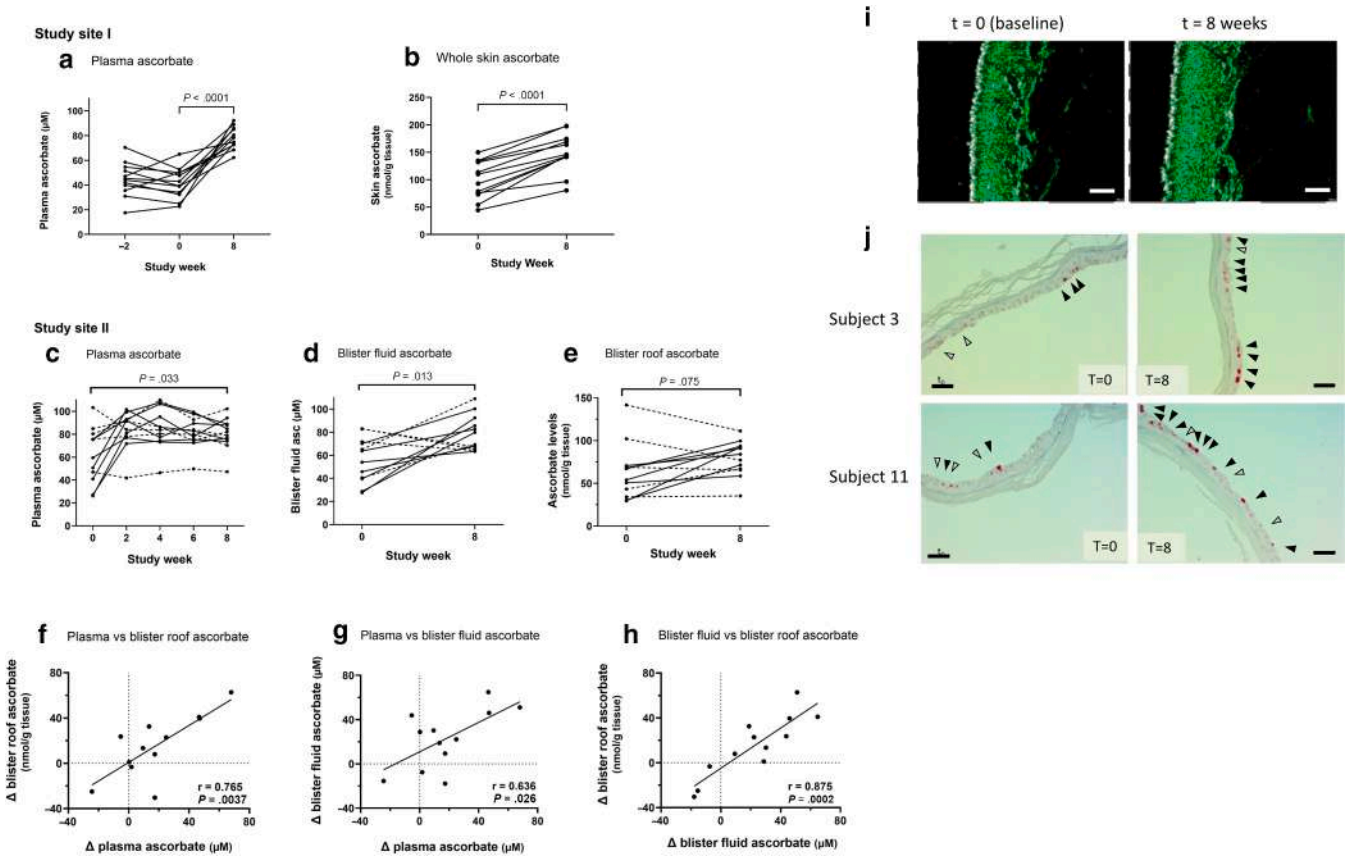

Figure 2. Changes in plasma and skin ascorbate concentrations and skin function after dietary supplementation with kiwifruit. (**a, b**) Study site I. (**a**) Plasma ascorbate. (**b**) Whole-skin ascorbate concentrations increased from 99 ± 10 nmol/g tissue to 150 ± 10 nmol/g tissue (mean ± SD, $P < .0001$, n = 12). Significant differences from paired *t*-tests are indicated. (**c**–**h**) Study site II, with data from participants excluded from the per-protocol analysis in Supplementary Table S4 shown as dashed lines. (**c**) Plasma levels from fortnight samples obtained throughout the 8-week intervention period. (**d**) Blister fluid ascorbate. (**e**) Blister roof ascorbate at beginning and end of the intervention period. (**f**–**h**) Relationship between change in plasma ascorbate, blister roof ascorbate, and blister fluid ascorbate in study participants. All data are week 8 minus week 0. Pearson's correlation data and linear regression lines are shown. (**i, j**) Skin images obtained in situ from functional analyses at study site II. (**i**) Exemplar high-resolution ultrasound image from DUB SkinScanner75 of change in skin density before and after 8 weeks SunGold kiwifruit intake. Skin density increased from 0.154 ± 0.027 scanner units at baseline to 0.228 ± 0.035 after the intervention ($P < .0001$) (Supplementary Table S4). Bar = 200 μm. (**j**) Photo documentation of staining for the cell proliferation marker Ki-67 in 2 representative participants—participant 3 (upper row) and participant 11 (lower row)—showing increased staining after kiwifruit supplementation. The blister roof slices were incubated with a mouse mAb against Ki-67, with individual cells staining red (arrowheads) in the lower layers of the epithelium. Cells with intense staining are indicated with a solid arrowhead, and lesser intensity staining is indicated with open arrowheads. Calculated staining was 21.06 ± 5.44 at baseline (T = 0), compared with 27.42 ± 4.9 units at 8 weeks (T = 8) ($P = .0091$) (Supplementary Table S4). Bar = 100 μm.

ETHICS STATEMENT

For the cross-sectional study, the collection of surplus skin tissue from patients undergoing elective surgical procedures was approved by the University of Otago Human Ethics Committee (H17/067) with the assistance of the Canterbury Cancer Society Tissue Bank under the New Zealand Health and Disability Ethics Committees approval (16/STH/92). Patient written informed consent for this study was given for the use of the samples for this project only, and no tissues were collected for tissue banking. Ethics approval for the dietary intervention study was from the Health and Disability Ethics Committee of New Zealand (Health and Disability Ethics Committee approval number 19/STH/38) (study site I) and the International Medical & Dental Ethics Commission (study site II). The trial was registered with the Australian and New Zealand Clinical Trial Registry (identification ACTRN12619000302156). Participants at study site I were recruited from local universities and tertiary institutes with notices about the study placed at key locations and disseminated electronically. At study site II, participants were recruited randomly from the SGS database. Written informed consent, demographic data, and medical history were collected at screening.

DATA AVAILABILITY STATEMENT

All original data for this study are reported in the graphs and tables. No specific datasets were generated or analyzed during this study. Further information can be obtained from the authors in response to reasonable requests (MV; margreet.vissers@otago.ac.nz).

KEYWORDS

Collagen; Dermis; Epidermis; Fibroblasts; Plasma ascorbate

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CONFLICT OF INTEREST

The authors state no conflict of interest.

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AUTHOR CONTRIBUTIONS

Conceptualization: MCMV, JMP, JS, DS, AvS; Formal Analysis: JMP, EV, DS, AvS, MCMV; Funding Acquisition: MCMV; Investigation: ES, SMB, H.R.B, JS, DS, AvS, JMP; Methodology: JMP, EV, SMB, HRM, DS, AvS, MCMV; Project administration: EV, DS, AvS; Visualization: JMP;

Writing - Original Draft Preparation: JMP; Writing - Review and Editing: SMB, DS, AvS, MCMV

Disclaimer

The funders were not involved in the study design, data analysis, writing of the manuscript, or decision to publish.

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SUPPLEMENTARY MATERIAL

Supplementary material is linked to the online version of the paper at www.jidonline.org, and at 10.1016/j.jid.2025.10.587.

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SUPPLEMENTARY MATERIALS AND METHODS

Cross-sectional study

The relationship between plasma ascorbate status and ascorbate content in the various skin compartments was investigated in tissues collected from healthy consented individuals undergoing elective plastic surgery (Supplementary Table S1). Matched fasting blood samples were collected at the time of surgery. Patient information included gender, age, ethnicity, and the site of the skin resection. Blood samples placed on ice were also processed immediately.

Dietary intervention studies

To determine the effect of increasing dietary vitamin C intake on skin ascorbate levels and functional parameters, we undertook a before-and-after dietary-intervention study at 2 sites, each with 12 healthy participants. Participants were supplemented daily with 2 SunGold kiwifruit, a high-vitamin C–containing food (Carr et al, 2012; Conner et al, 2020) for 8 weeks (Supplementary Tables S2 and S3 and Supplementary Figure S3). At site I, in Christchurch, New Zealand, we monitored ascorbate levels in plasma and in whole-skin biopsies. At site II, at SGS Institute Fresenius in Hamburg, Germany, blood samples were obtained, and suction blister technology was used to collect samples of plasma, epidermis (skin blister roof), and interstitial fluid (blister fluid) for ascorbate analysis and monitoring of skin function parameters.

Ethics approval for study site I was given by the Health and Disability Ethics Committee of New Zealand (Health and Disability Ethics Committee approval number 19/STH/38), and the trial was registered with the Australian and New Zealand Clinical Trial Registry (identification ACTRN1 2619000302156). Participants were recruited from local universities and tertiary institutes with notices about the study placed at key locations and disseminated electronically. The protocol for study site II was approved by an independent ethics committee, the International Medical and Dental Ethics Commission, prior to individuals being enrolled in the study. Participants were recruited randomly from the SGS database. Written informed consent, demographic data, and medical history were collected at screening.

Intervention study site I. From 32 screened participants, we recruited 12 individuals who met the eligibility criteria (Supplementary Tables S2 and S3 and Supplementary Figure S3), including a fasting plasma ascorbate $\leq$45 μM. Participant demographic data collected by questionnaire at screening included age, gender, ethnicity, smoking behavior, medication usage, and fruit and vegetable consumption. After a 2-week lead-in period to allow time to control their dietary vitamin C intake, participants were provided with 2 SunGold kiwifruit per day for an 8-week period.

Fasting whole blood was collected at baseline and before and after the intervention period (weeks −2, 0, and 8). Whole-skin biopsies were taken from the upper thigh from non–sun-exposed areas by 3-mm punch biopsy at weeks 0 and 8.

Intervention study site II. Participants were recruited randomly from the SGS database, and informed consent was obtained. We screened 28 individuals for eligibility (Supplementary Tables S2 and S3 and Supplementary Figure S3), collected demographic data and medical history, and recruited 12 individuals with the lowest fasting plasma ascorbate concentrations (Supplementary Figure S3). Plasma samples were collected at 0, 2, 4, 6, and 8 weeks, and skin samples were taken at weeks 0 and 8. Blister roof samples (epidermis) were obtained from the forearm area using the suction blister method (Kiistala, 1968) using plexiglass suction chambers with 7-mm circular openings. A vacuum of 550–850 mbar was applied for 2.5–3.0 hours, and the blister roof and blister fluid were removed under sterile conditions. Epidermis, blister fluids (interstitial fluids), and processed plasma samples were stored at −80 °C until shipment on dry ice to Christchurch for ascorbate analysis. Skin function tests were carried out at weeks 0 and 8.

Suction blister method

Epidermal samples and interstitial blister fluids were collected using the suction blister method. Plexiglass suction chambers with circular openings of 7-mm diameter were placed on the skin of the test sites, and a vacuum of about 550–850 mbar was applied. The blisters were induced within 2.5–3.0 hours. The roofs and the liquids of the generated suction blisters were removed under sterile conditions. The blister roofs were used for immunohistochemical analysis (cell proliferation marker Ki-67) and detection of vitamin C. The liquids were used for procollagen and vitamin C analysis in blister fluids (blisters of the unirradiated test sites) and for 8-isoprostane detection in the fluids of the irradiated and unirradiated test sites.

The small wounds induced by this procedure were covered with patches. All wounds healed completely, without scarring, within 10 days.

Ascorbate analyses

Plasma and blister fluid ascorbate processing. Peripheral blood was collected into heparin or K_3-EDTA tubes. Whole blood and blister fluids were centrifuged, and ascorbate was stabilized in the supernatant by adding an equal volume of 0.54 M perchloric acid containing 100 μM diethylenetriaminepentaacetic acid. The protein precipitate was removed by centrifugation, and the supernatant was stored at −80 °C until analysis (Pullar et al, 2018).

Ascorbate was measured using reverse-phase high-performance liquid chromatography with coulometric electrochemical detection as previously described (Lykkesfeldt, 2000; Pullar et al, 2018). For skin samples, the amount of ascorbate was related to the wet weight of the sample and expressed as nmol/g tissue. In a subset of samples, the ascorbate content was related to the DNA content of the sample as a measure of cellularity and expressed as ng ascorbate/ng DNA. Plasma and blister fluid ascorbate were expressed as μM.

Skin ascorbate processing. For the cross-sectional study, frozen skin samples ~2 cm^2 were thawed on ice, and whole skin and epidermal and dermal fractions were prepared. The epidermis was removed by scraping with a scalpel from a skin section frozen over a piece of dry ice. Confirmation that the collected fraction contained mainly epidermal cells was determined by microscopic analysis. The underlying dermal layer was cut into sections and weighed. The

skin fractions were finely disrupted with a ground glass pestle in 300 µl ice-cold 5 mM phosphate buffer at pH 7.4, containing 100 µM diethylenetriaminepentaacetic acid. An equal volume of ice-cold 0.54 mol/l perchloric acid solution containing 100 µM diethylenetriaminepentaacetic acid was added; the sample was vortexed thoroughly; the precipitate was removed by centrifugation at 12,000 r.p.m. for 2 minutes at 4 °C, and the supernatant aliquots were stored at −80 °C. For the dietary intervention study, whole-skin biopsies and blister roof samples were extracted in the same way as the skin dermal fractions.

DNA analysis

The DNA content of skin samples was determined as an estimate of tissue cellularity. The dermis or epidermis homogenates were diluted to 1 ml with ice-cold 10 mM phosphate buffer at pH 7.4, and the cells were lysed using 3 rapid freeze-thaw cycles in dry ice/ethanol and sonication on ice for 15 seconds. DNA content was estimated by fluorescence measurement at 544−590 nm after addition of 1 mg/ml propidium iodide solution and standardized against a standard curve of calf thymus DNA from 1.25−40 µg DNA per ml (Kuiper et al, 2010).

Biophysical skin function measurements

At study site II, biophysical skin function tests were performed by trained study personnel. All measurements were performed in climate-controlled rooms at 21.5 °C (±2 °C) and 50% (±10%) relative humidity, allowing for an acclimation time of at least 30 minutes before study start. Climate conditions were continuously checked and documented by a thermo-hygrograph.

Measurement of skin elasticity

Measurement of skin elasticity was performed with the Cutometer MPA 580 according to the manufacturer's instructions. Skin elasticity was determined from the R5-value (Ur/Ue, immediate recovery/elastic deformation). An increase in the R5 value indicates augmented skin elasticity. Three single measurements were performed on the volar forearm test site. The values of the 3 single measurements were electronically saved and handwritten onto a specific report form for each participant.

Measurement of dermis density changes

Skin density changes were determined in situ by ultrasound using the DUB SkinScanner 75 equipped with a 75-MHz applicator according to the manufacturer's instructions. Three separate measurements were performed on the volar forearm test site. The measurement created a scan depicting the surface of the skin and continuing down to the selected depth. The intensity of the reflections in the scan were analyzed. The density of the tissue represents the concentration of structural proteins in the dermis.

Epidermal cell proliferation

Cell regeneration and renewal of the epidermal layer were determined by monitoring cell proliferation in the epidermal suction blister roof. Histology cross sections were prepared and stained with antibody to the proliferation marker Ki-67, a nuclear protein expressed at high levels during the growth and mitotic periods of the cell cycle but absent in resting cells.

The staining intensity of cell nuclei was counted for each test participant before and after the intervention. The nuclei were either dark or light red colored, that is, 2 staining intensities (1: light; 2: strong) were observed. For each slide, the stained cell nuclei of 5 different areas were counted, and the mean ± SD was calculated. The ratio of the number of the colored cell nuclei to the total nuclei number per slice was calculated for the respective staining intensity in percentage. According to an intensity score, the value obtained for the dark red colored cell nuclei was multiplied by 2, that is, the dark red colored cell nuclei were weighted twice. For each slice, the value obtained for the light red−colored nuclei and that obtained for the dark red colored nuclei (twice weighted) were added to calculate the intensity for each section. The relative values for each test participant at baseline and after the intervention were calculated.

Antioxidant protection/UVA irradiation

The potential for antioxidant activity was determined by measuring 8-isoprostane content in the blister fluid before and after UVA-induced stress on the skin. The measurement of 8-isoprostane, an oxidation product of arachidonic acid, is a reliable approach to assess oxidative stress in vivo (Kuhn et al, 2006). Each participant was irradiated with 10 Joules per cm^2 UVA on 1 subtest site (3 × 4 cm^2) at baseline and after the intervention period. Irradiation was conducted with the solar-simulator SOL 500, equipped with an H1 UV-filter blocking the UVB portion of the solar spectrum. The 8-isoprostane levels were monitored in the blister fluids using a commercially available ELISA Kit.

Revitalization of skin structure: collagen synthesis

In the inner skin dermis layer, vitamin C is essential for the synthesis and stabilization of collagen by fibroblasts. Revitalization of the skin by de novo collagen synthesis was detected by ELISA for procollagen type I peptides present in the blister fluids at baseline and at the end of the intervention period.

Statistical analysis

Statistical analyses were carried out using GraphPad Prism (version 9.3.1). Statistical significance was tested using paired *t*-tests for within-subject comparisons and unpaired *t*-tests for between subjects (2 tailed), with significance set at $P < .05$. Correlations were tested using Pearson's correlation and linear regression analysis (least squares fit) to determine associations.

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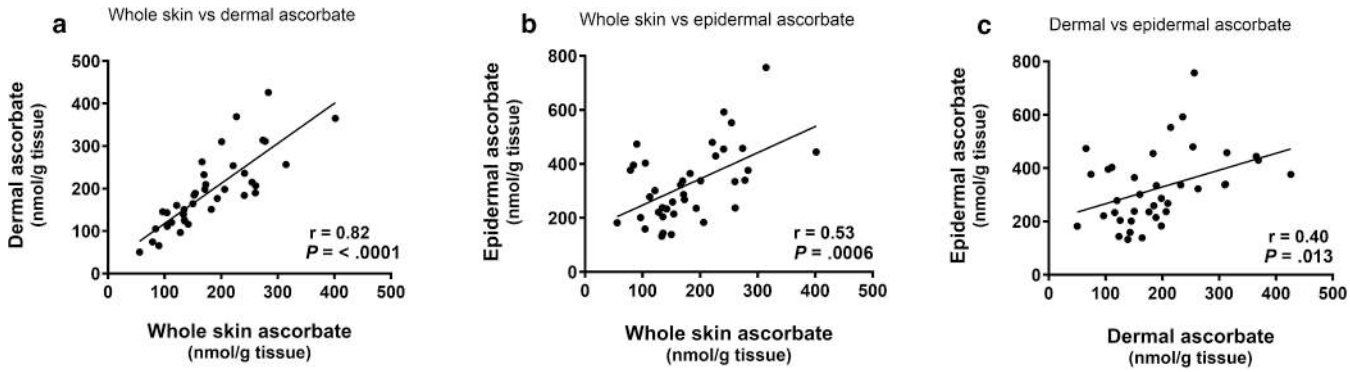
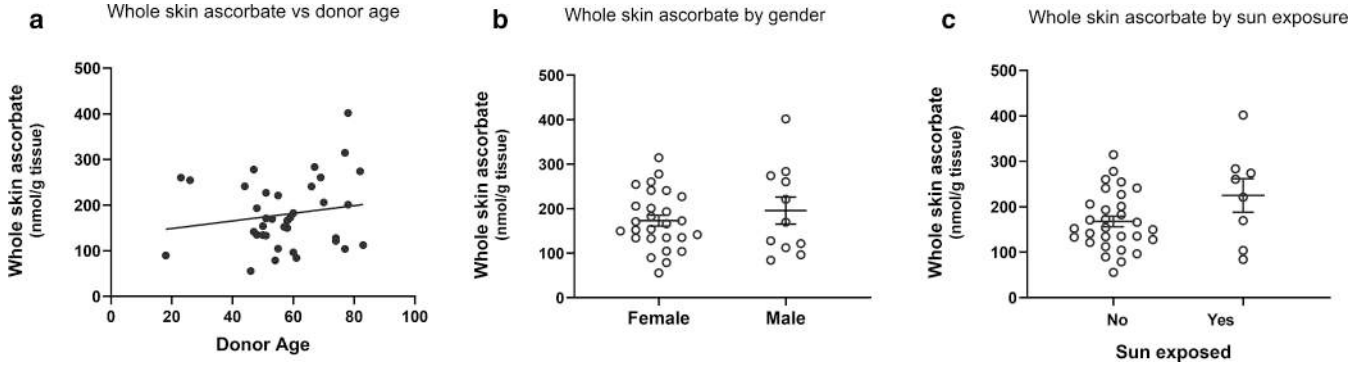
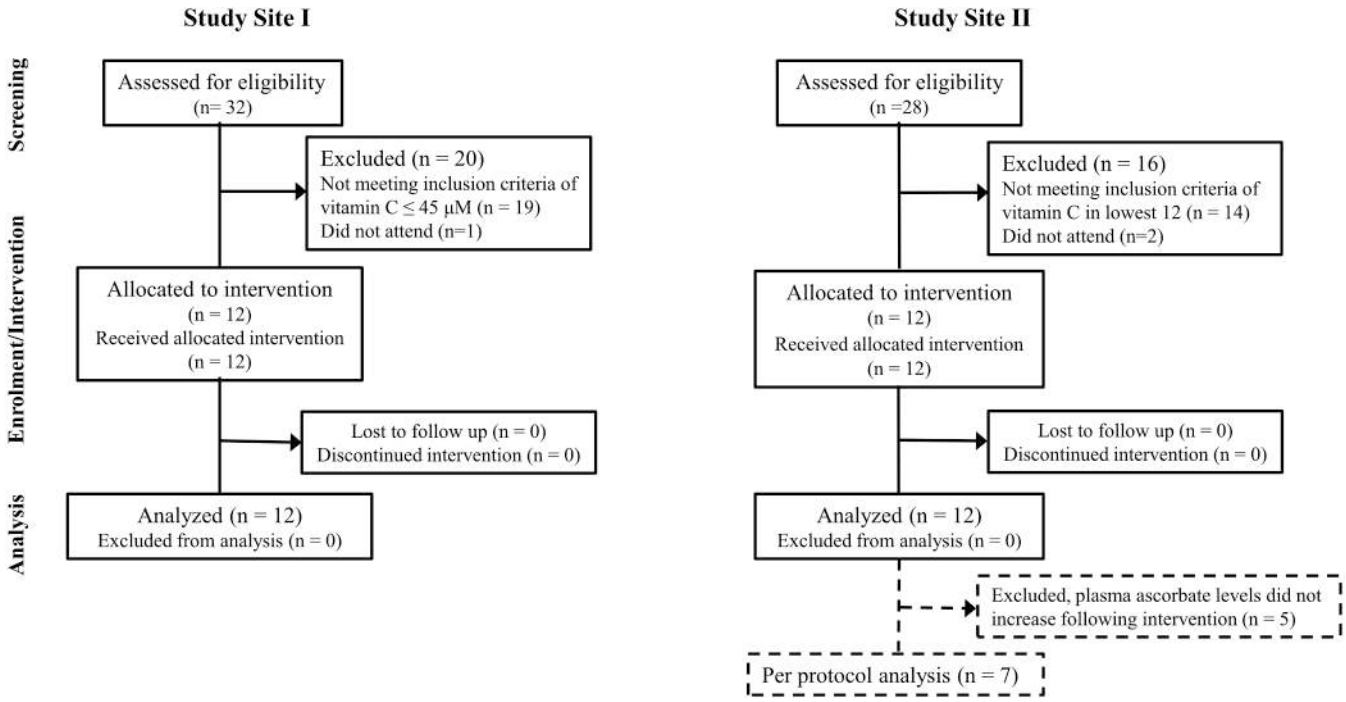


Supplementary Figure S1. Relationships between the ascorbate content of whole skin, dermis, and epidermis. (**a**) A tight correlation exists between dermal and whole-skin ascorbate, with strongly positive correlations also between (**b**) epidermal and whole-skin ascorbate and between (**c**) dermal and epidermal levels (Pearson correlation coefficient [*r*], *P*-values, and regression lines are shown).



Supplementary Figure S2. Relationship between whole-skin ascorbate and donor age, gender, and sun exposure. **(a)** Whole-skin ascorbate by donor age with the nonsignificant regression line shown (r = 0.169, *P* = .309). **(b)** Whole-skin ascorbate by gender. Bars indicate the mean ± SEM, unpaired *t*-test *P* = .4144. **(c)** Whole-skin ascorbate by sun exposure of the tissue. Bars indicate the mean ± SEM, unpaired *t*-test *P* = .0568.



Supplementary Figure S3. Consort flow diagram for dietary intervention study sites I and II, showing screening and recruitment, reasons for exclusion, and data analyzed.

Supplementary Table S1. Cross-Sectional Study Participant Characteristics (n = 38)

Parameter	n (% of sample)	Mean (SD)
Age, y		57 (15)
Sex		
Female	27 (74%)	
Male	11 (26%)	
Ethnicity		
Chinese	1 (3%)	
Irish	1 (3%)	
Māori	1 (3%)	
New Zealand European	35 (92%)	
Sun exposed[1]		
No	29 (76%)	
Yes	9 (24%)	
Site of skin sample[2]		
Breast	12 (32%)	
Torso	13 (34%)	
Head and neck	6 (16%)	
Arm and leg	6 (18%)	
Time from surgical resection to laboratory processing (min)		17 (15)

Ethnicity was self-reported by the participants, in line with requirements from the ethics committee.
[1]Sun exposed areas = head, neck, and lower arms below the elbow and legs below the knee.
[2]One participant was sampled from 2 sites (data combined).

Supplementary Table S2. Characteristics of Individuals Enrolled in the Pilot Intervention Study

Parameter[1]	Study Site I (n = 12)	Study Site II (n = 12)
Age, y	24 (4)	54.9 (13.9)
BMI (kg/m^2)[1]	24 (4)	6:6
Male: female	7:5	4:8
Vitamin C (μM)[1]	37.1 (10.0)	52.4 (11.1)

Abbreviation: BMI, body mass index.
[1]Data represent means (SD).

Supplementary Table S3. Inclusion and Exclusion Criteria for Pilot Intervention Studies

Study Site I		Study Site II	
Inclusion (all Required)	**Exclusion (1 Required)**	**Inclusion (all Required)**	**Exclusion (1 required)**
Aged 18−75 y	Fear of needles	Aged 20−65 y	Fear of needles
Below average plasma vitamin C levels (<45μmol/l)	Allergy/intolerance to kiwifruit	Low/below average fasting plasma vitamin C levels	Allergy/intolerance to kiwifruit and/or patches/plasters
Residing in Christchurch for duration of study	Excessive alcohol consumption (>21 standard drinks per week)	In general good health and mental condition	Excessive alcohol consumption (>21 standard drinks per week)
Taking prescription medication (within past 3 mo)	High fruit and vegetable intake (≥5 servings per day)	Willing and able to sign the informed consent	High fruit and vegetable intake (>3 servings per day)
	Diabetes mellitus	Skin type: healthy without any signs of disease	Diabetes mellitus
	Fitzpatrick scale type IV or more	Fitzpatrick scale types I−III	Topical or systemic prescription medication 2 weeks before and during study
	A personal history of abnormal scarring	Maximum: 50% light smokers (maximum: 10 cigarettes per day)	Prone to hyperpigmentation, hypertrophic scar formation, and/or keloidal scar formation
	Bleeding disorders		Bleeding disorders
	Skin conditions such as eczema		Acute and/or chronic skin diseases or damage (eg, atopic eczema, neurodermatitis, psoriasis, active acne, vitiligo, sunburn, wounds, rash, scars, warts) on the test sites
			Participating in any other study with a pharmaceutical preparation within a period of at least 4 weeks prior to this study or simultaneously
	Obese (BMI >35 kg/m^2)		Obese (BMI >35 kg/m^2)
			Tattoos on the test site

Abbreviation: BMI, body mass index.

Supplementary Table S4. Effect of 8-Week Dietary Kiwifruit Intervention on Plasma and Epidermal Ascorbate Levels and Skin Functions at Study Site II

	Study Site II: Full Cohort (n = 12)			Study Site II: Per-Protocol Subgroup (n = 7)		
Plasma and Skin Parameter/Function	**T0 (baseline)**	**T8 (wk)**	**[1]*P*-Value**	**T0 (baseline)**	**T8 (wk)**	**[1]*P*-Value**
Plasma ascorbate (μM)	62 ± 24	80 ± 14	**.033**	50 ± 21	83 ± 8	**.008**
Blister fluid ascorbate (μM)	56 ± 19	79 ± 15	**.013**	47 ± 16	82 ± 13	**.004**
Blister roof ascorbate levels (nmol/g tissue)	63 ± 31	79 ± 32	**.075**	53 ± 17	84 ± 14	**.004**
Skin density (DUB SkinScanner75 units)	0.154 ± 0.027	0.228 ± 0.035	**<.0001**	0.163 ± 0.032	0.231 ± 0.041	**.0023**
Epidermal cell regeneration (calculated intensity of Ki-67 staining)	21.06 ± 5.44	27.42 ± 4.96	**.0091**	23.00 ± 3.83	29.5 ± 4.10	**.0416**
Skin elasticity (Cutometer, R5 value, no units)	0.717 ± 0.185	0.668 ± 0.199	**.041**	0.767 ± 0.19	0.717 ± 0.233	.072
Δ8-isoprostane in blister fluid, after skin irradiation (relative to unirradiated skin)	1.53 ± 1.38	1.19 ± .041	.484	1.17 ± 0.815	1.18 ± 0.19	.9651
Procollagen peptide in blister fluid (ng/ml)	2650 ± 1158	2464 ± 879	.7529	2020 ± 1031	2453 ± 1010	.1807

Means ± SD are shown. Significantly different results are highlighted in boldface.
[1]Significant differences were determined with paired *t*-tests, with significance set at $P < .05$.