什么是衰老（Aging）

López-Otín, C. (*2013*). The hallmarks of aging. ***Cell***, 153(6)

18岁之后就要抗衰？！谁"盯"上了年轻人？

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随着医美行业的火爆，不少年轻人愿意为美买单。《中国医美行业2023年度洞悉报告》显示，受访的医美潜在消费人群平均年龄为28岁，其中30岁以下的占75%；25岁以下人群中，29%计划在2023年增加医美开支或尝试更多项目。在此背景下，不少商家将目光放在年轻人身上。然而，为抢占市场扩展客流，一些医美机构在宣传时大肆贩卖容貌焦虑、年龄焦虑，**鼓吹"抗衰越早越好""18岁之后就要抗衰"**，引得求美心切的年轻人盲目做医美，忽视了其真实需求。

"盯"上年轻人的医美机构都采用了哪些宣传手段？以容貌焦虑、年龄焦虑吸引消费者是否合理？机构大力宣称的"抗衰越早越好"等广告词是否存在问题？让我们一起来看一下。

"20多岁的年轻人过度抗衰是不可取的，人的衰老是由端粒决定的，在年轻的时候本身不存在衰老的问题。现在一些年轻人抗衰主要是在医美机构的忽悠下、对衰老抵触进而产生焦虑情绪，过度抗衰会对身体产生不利影响，也是一种资源的浪费。"山东省枣庄市某三甲医院内科主任医师张女士说。

皮肤老化因素

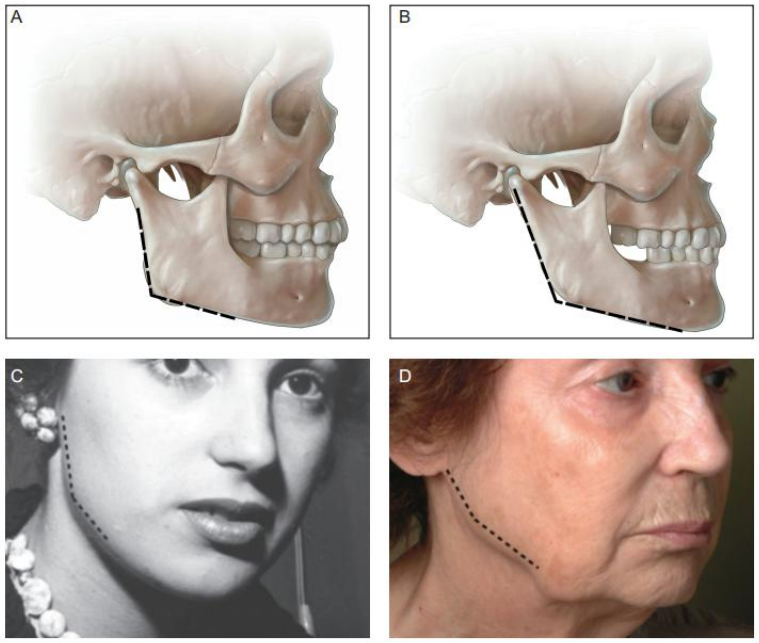


Figure 1. Progressive bony remodeling occurs with age in areas that resorb in a specific and predictable manner. The mandibular angle in females increases, as does the anterior projection of the chin. Images show this process in a young female (A) vs older (B) adult. Panel C shows the clinical presentation during young adulthood in this female patient at 22 years of age, whereas panel D reflects changes secondary to bony remodeling of the mandible with aging in the same patient at 70 years of age. Dashed lines represent areas of the chin and jawline visibly impacted by bony remodeling of the mandible. Patient images in panels A and B provided by Arthur Swift, MD. Patient images in panels C and D provided by B. Kent Remington, MD, owner of these original images, who has granted permission to publish them.

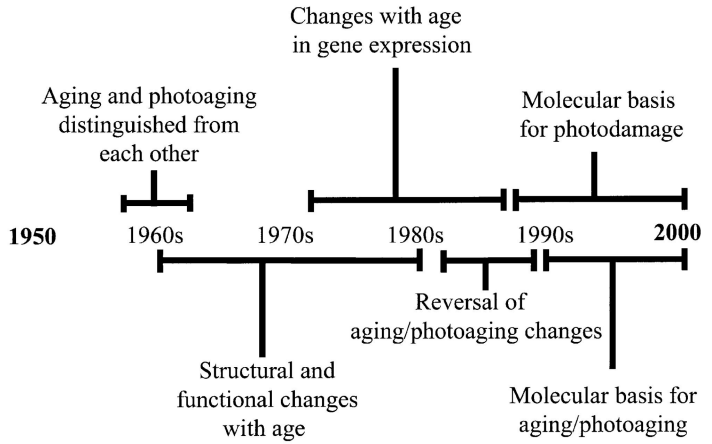
皮肤老化

- **年龄增长老化（chronological aging）**
- **过早老化（premature aging）**

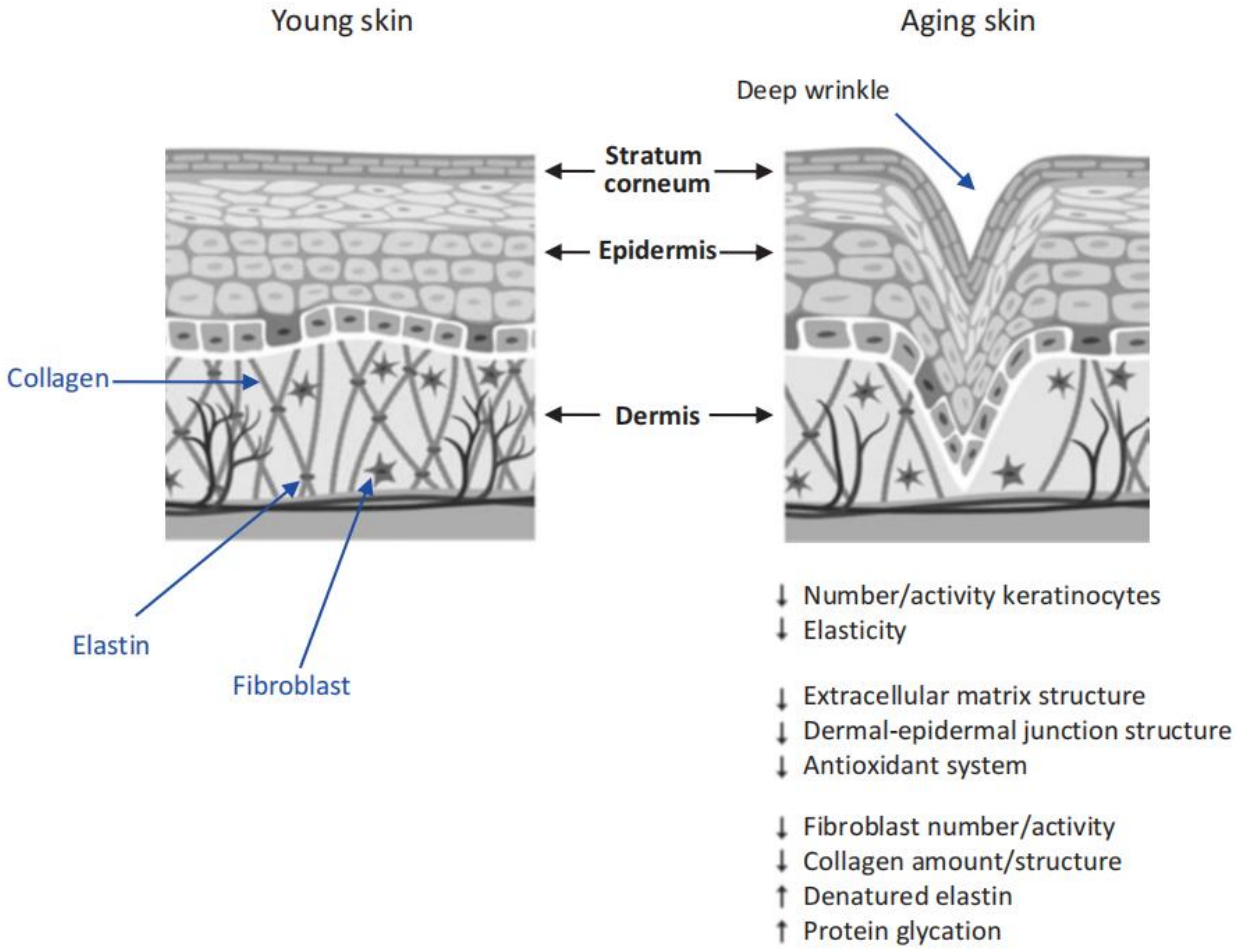
皮肤老化率受到内在和外在因素以及生物功能的几乎各个方面的影响。

- **内在的皮肤老化因素:** 种族和皮肤组织中的激素变化。
- **外在因素**: 营养不良、吸烟、尼古丁、**空气污染和UVR暴露**

Figure 4. Perioral dynamic discord is illustrated across the ages. With aging, the skin envelope deteriorates faster than the muscle strength, leading to the orbicularis oris overwhelming the perioral skin. Images provided by Steven Liew, MD.



皮肤老化过程



老化的皮肤具有较薄和色素沉着的特点，松弛、粗糙、起皱、颜色黄、毛细血管扩张、干燥、脆弱、容易瘀伤和皮肤恶性肿瘤。内在皮肤老化是由遗传或代谢因素引起的正常生理过程。随着表皮变薄，真皮-表皮交界处（DEJ）面积减少，基底细胞增殖能力减弱，基底细胞层的组织学变化最为明显

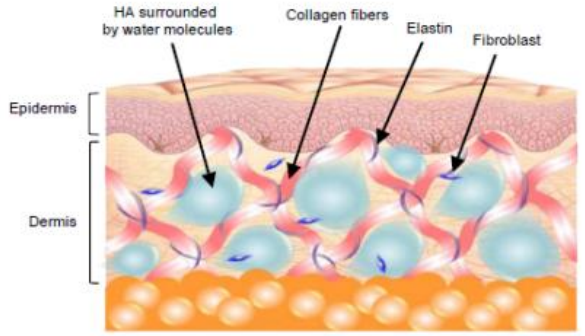
The extrafibrillar matrix consists of a complex mixture of

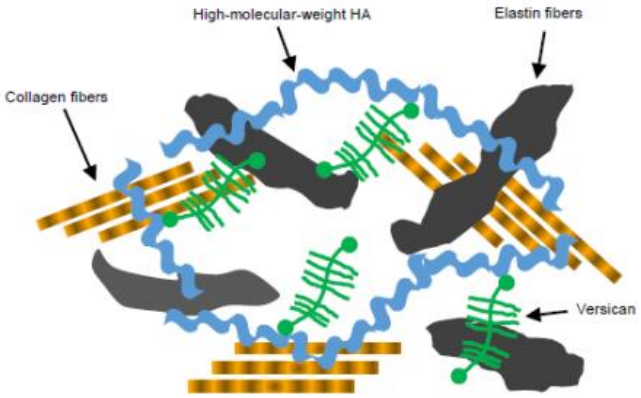
- proteoglycans
- glycoproteins
- glycosaminoglycans
- water
- hyaluronic acid

- 蛋白聚糖
- 糖蛋白类
- 糖胺聚糖
- 水
- 透明质酸

Photoprotected skin

Inner forearm

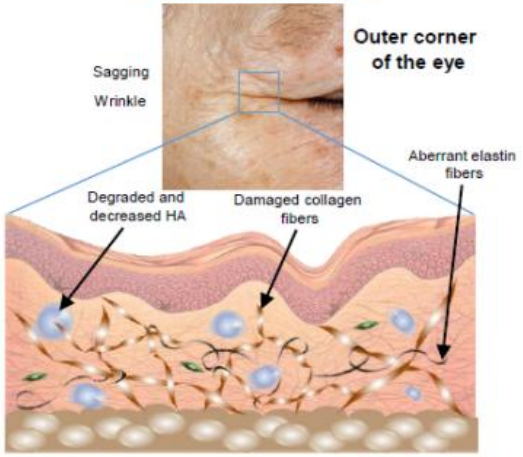


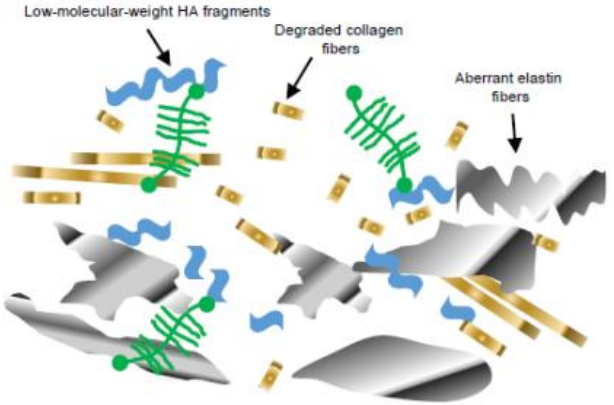


Well maintained ECM structures in the papillary dermis

Photoaged skin

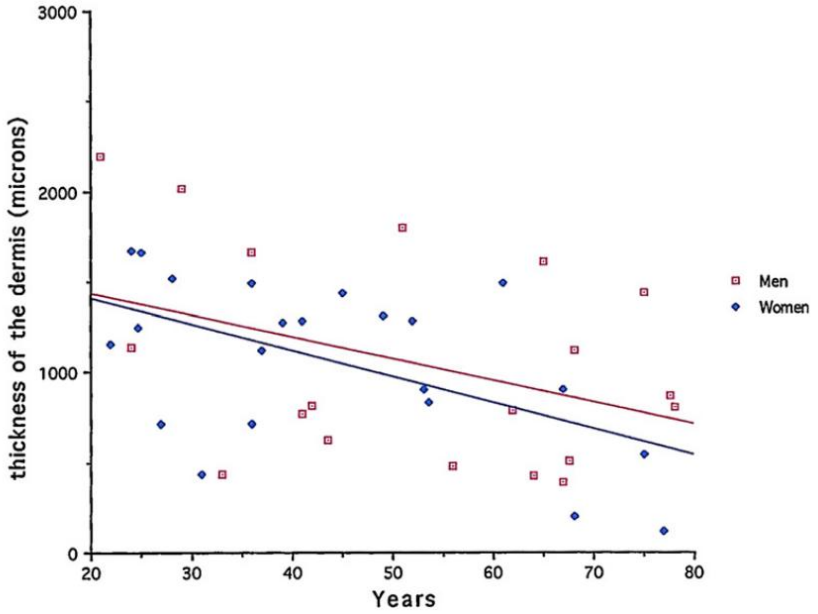
Outer corner of the eye

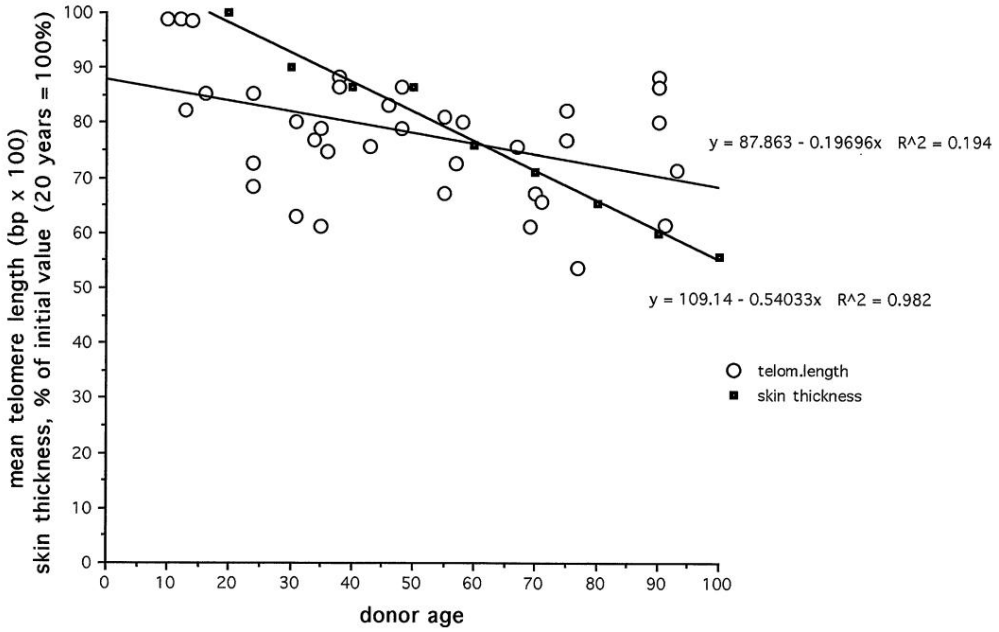


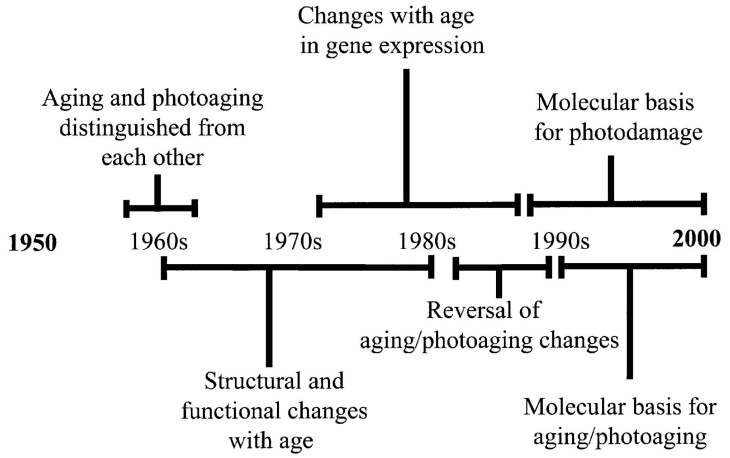


Disruption of the integrity of dermal ECM secondary to HYBID-mediated HA degradation

皮肤老化过程


thickness of the dermis (microns)
3000
2000
1000
0
20
30
40
50
60
70
80
Years
Men
Women

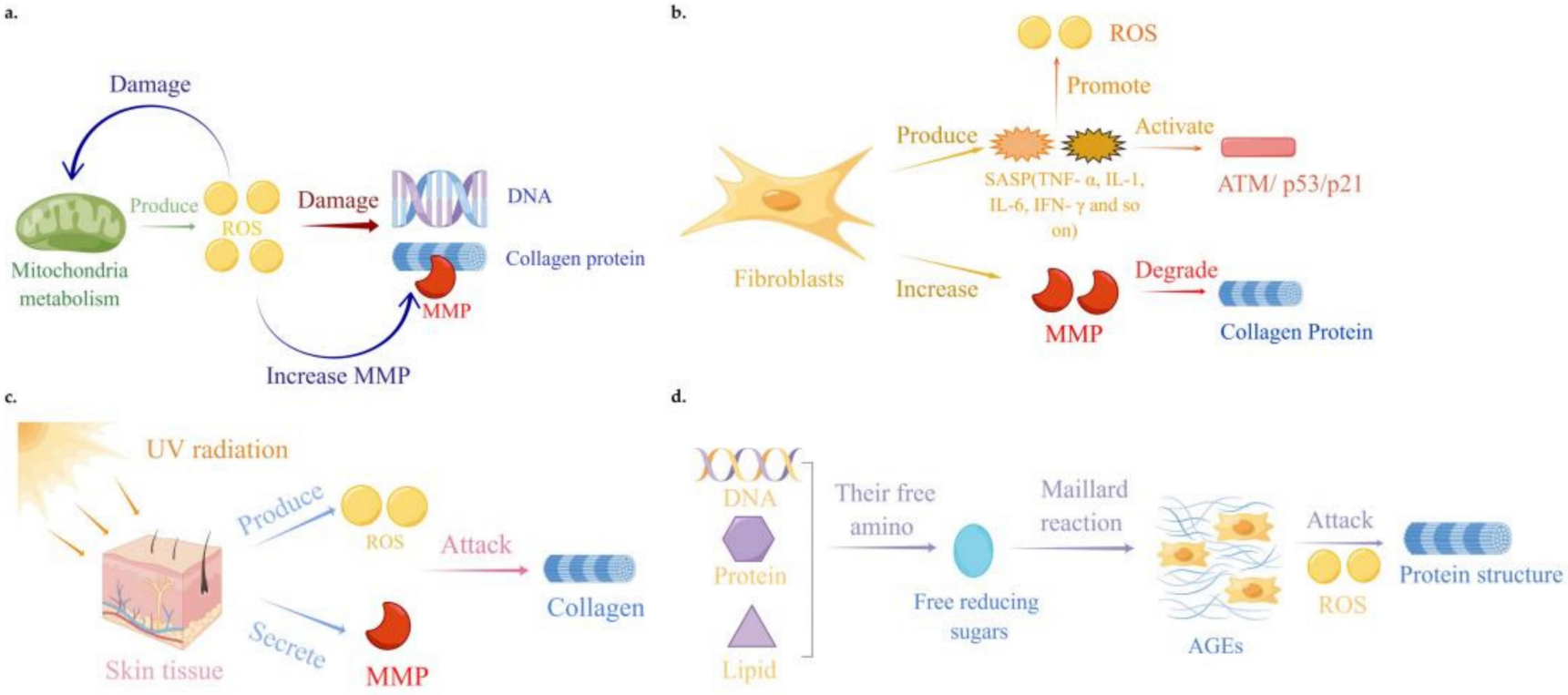

mean telomere length (bp x 100)
skin thickness, % of initial value (20 years = 100%)
y = 87.863 - 0.19696x R^2 = 0.194
y = 109.14 - 0.54033x R^2 = 0.982
telom.length
skin thickness
donor age


Changes with age in gene expression
Aging and photoaging distinguished from each other
Molecular basis for photodamage
1950
1960s
1970s
1980s
1990s
2000
Reversal of aging/photoaging changes
Structural and functional changes with age
Molecular basis for aging/photoaging

皮肤老化过程的机制

- 自由基和氧化应激理论
- 炎症老化理论
- 皮肤皮肤老化理论
- 非酶糖基化学理论

Matrix MetalloProteinases (MMPs)：基质金属蛋白酶



遗传因素，DNA修复和稳定性、细胞衰老和端粒、线粒体、凋亡、雌激素缺乏、昼夜节律、神经内分泌、疾病、身体活动、压力，和其他环境因素（如饮食、污染和吸烟）也很重要的皮肤老化领域，并已经在动物和实验研究中研究。

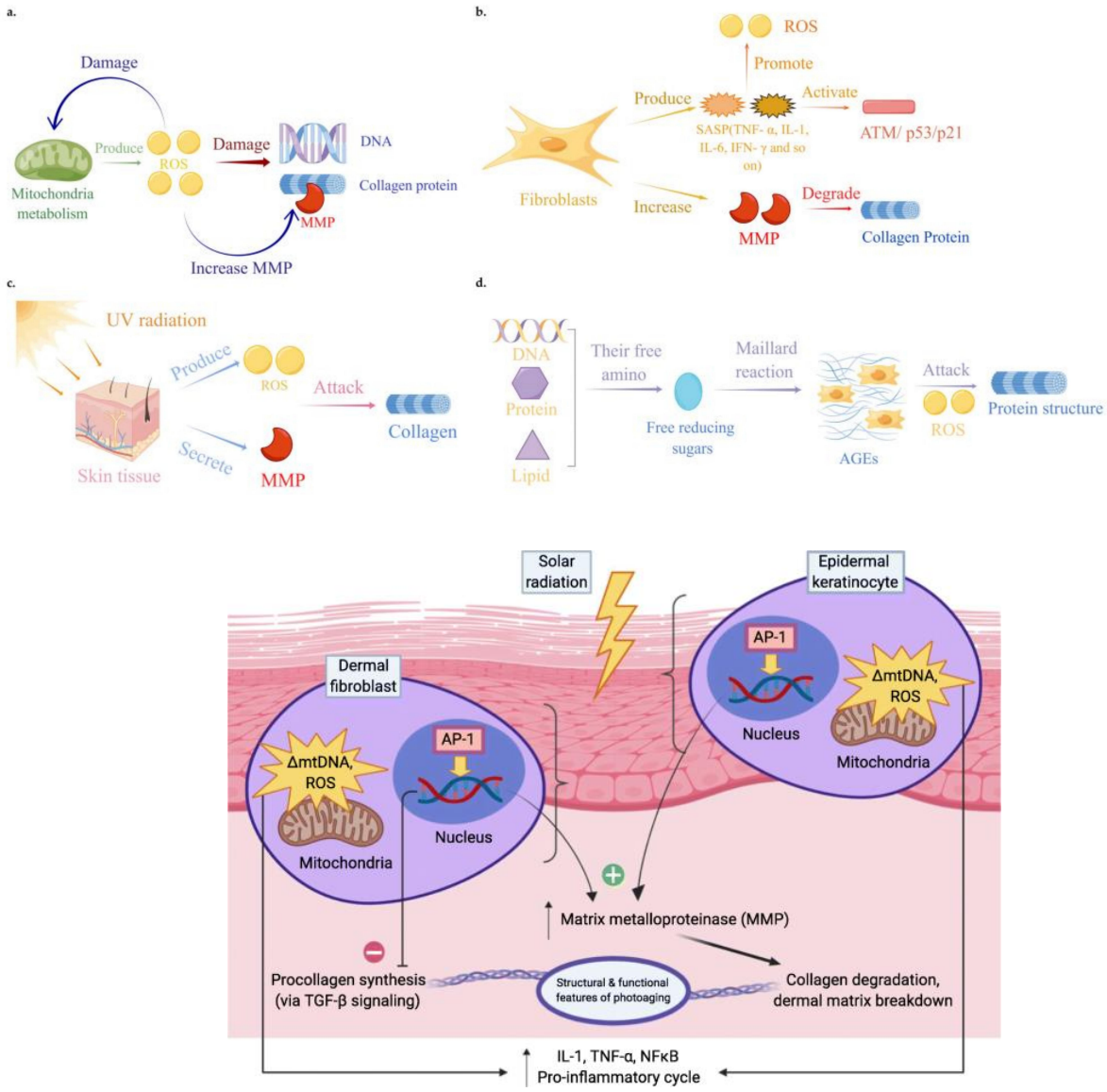
人参：可以通过调节信号通路发挥抗皮肤老化的作用，研究表明稀有人参皂苷能增加紫外线照射的人真皮成纤维细胞内I型胶原的产生并减少MMP-1 的分泌。

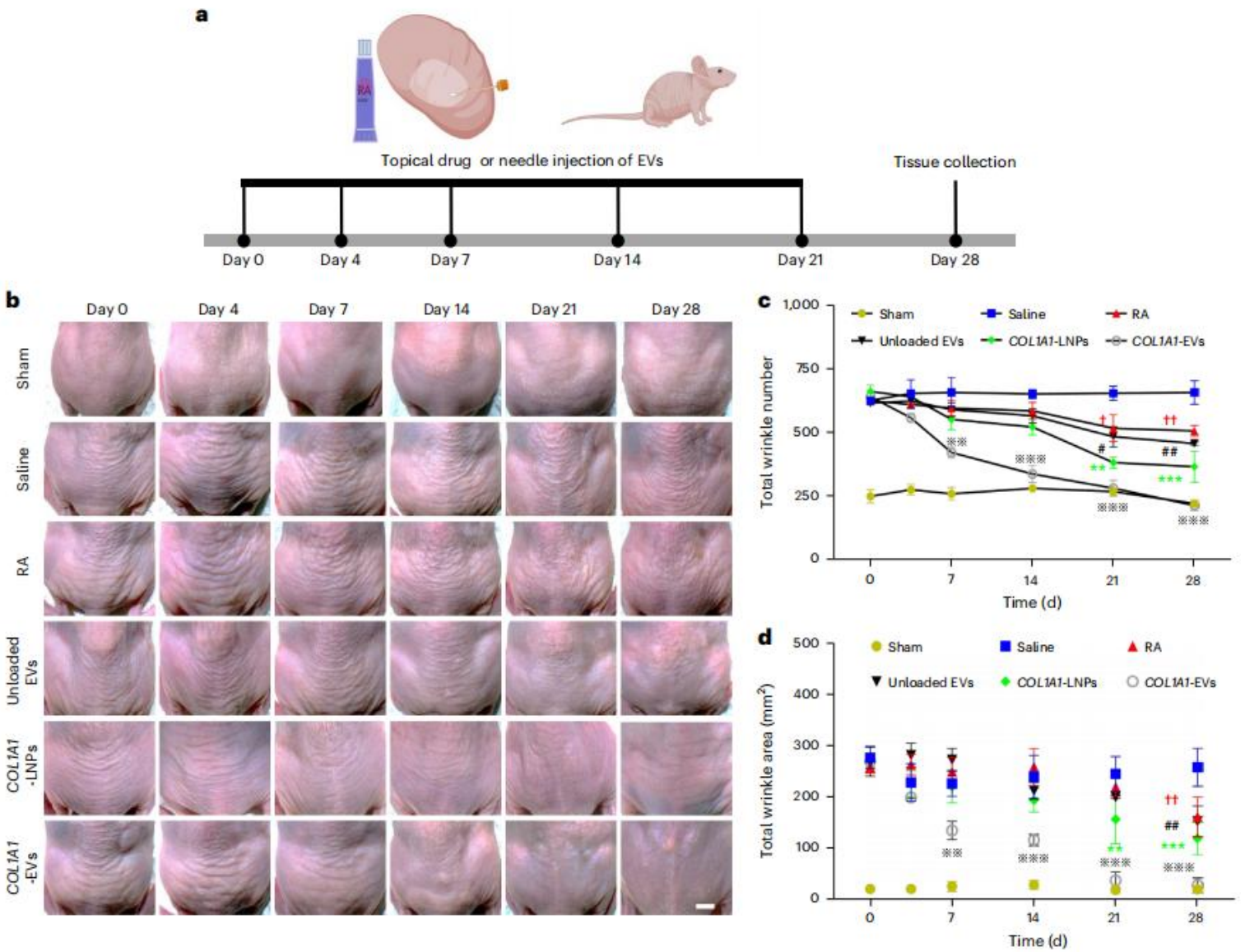
紫芝：紫芝中的多糖，可以起到保湿作用，同时还可以提高皮肤中羟脯氨酸和SOD含量。而且《神农本草经》记载：“肉灵芝，无毒、补中、益精气、增智慧，治胸中结，久服轻身不老。”据研究表明，灵芝多糖、灵芝三萜、灵芝多糖肽对延缓衰老均有一定效果。

麦角硫因：通过抑制AP-1通路和激活Nrf2介导的抗氧化基因对人皮肤成纤维细胞的皮肤老化发挥保护作用。

虾青素：可降低皮肤老化过程中真皮细胞的炎症反应，避免后续对皮肤损伤反应的发生，可降低皮肤皮肤老化。虾青素的保护作用与多种抗炎机制有关，包括PI3k/Akt、Nrf2、NF-κB。Erk1/2、JNK、p38 MAPK和jak-2/stat-3。人体实验发现，虾青素给药组受试者的皮肤水分流失减少，皮肤粗糙度改善，并防止了由紫外线引起的皮肤退化。

透明质酸钠：皮肤老化最明显的临床特征是皱纹的出现，其组织特征是真皮中异常弹性蛋白的积累和胶原纤维的严重丢失。真皮中弹性蛋白和胶原纤维的完整性是通过与透明质酸和蛋白多糖网络结构的相互作用来维持的；因此，透明质酸的降解可能是原纤维成分分解之前的初始过程。人体临床试验已经证实口服透明质酸钠，可增加皮肤老化患者的皮肤水分含量，减少褶皱深度，增加皮肤弹性和强度。





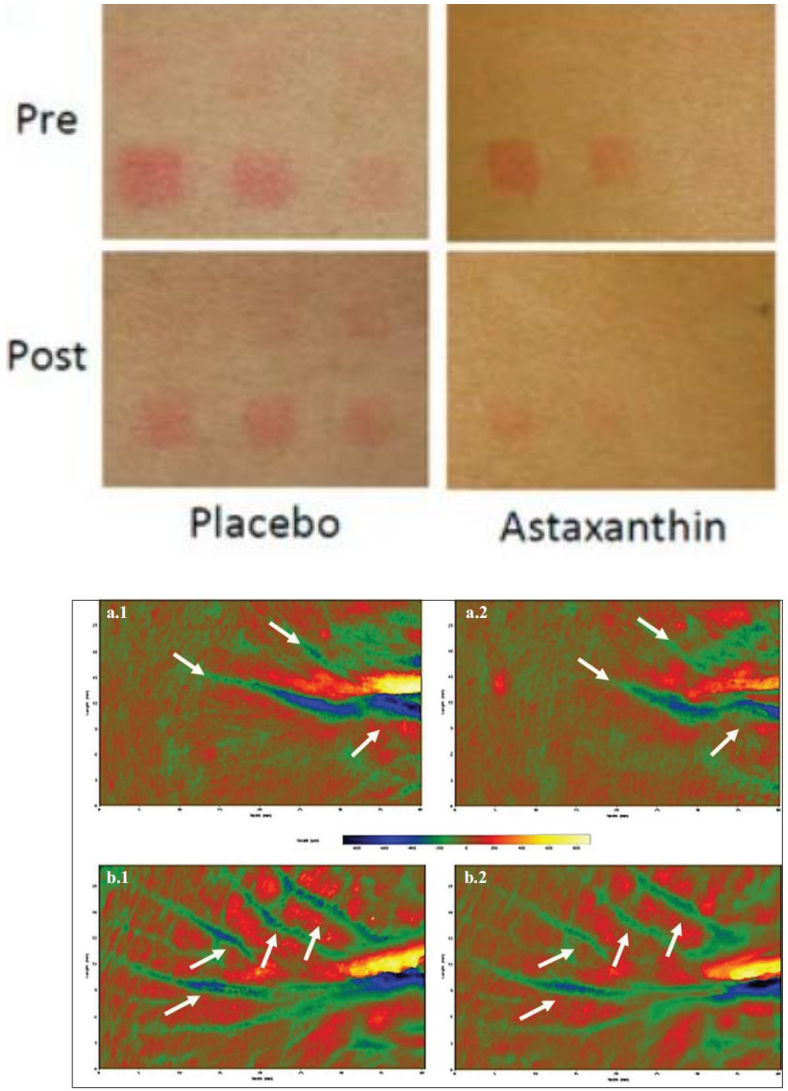


Figure 2. Primos 3D images of crow's feet areas of examples-active treated subjects. a.1 and b.1 refer to samples collected at baseline, whilst a.2 and b.2 refer to samples of the same skin surface area collected after 28 days of treatment. Yellow denotes the most elevated zones and blue denotes least ones.

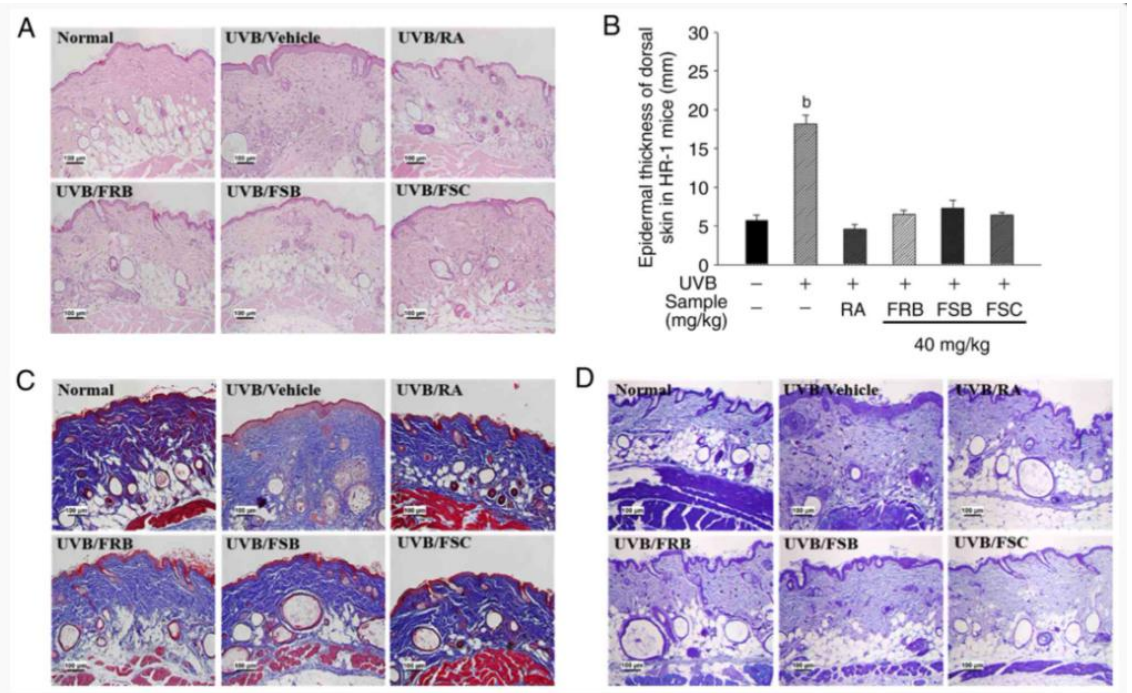
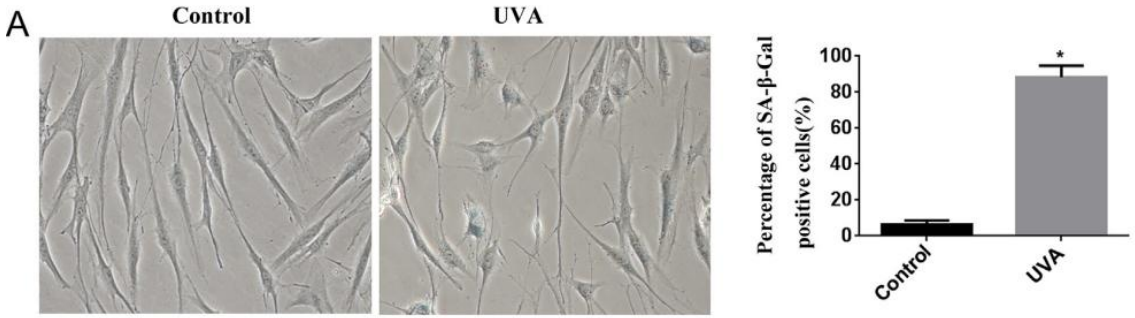


Figure 4 - Inhibition of UVB-induced histological alteration by FRB, FSB and FSC in hairless mice. Histological alterations of epidermal thickness, collagen degradation and mast cell infiltration were analyzed using (A) haemotoxylin and eosin staining, (B) epidermal thickness of dorsal skin, (C) Masson's trichrome staining and (D) toluidine blue staining, respectively. All values are presented as the mean ± standard deviation. A Kruskal-Wallis nonparametric test and Bonferroni's post-hoc tests were used to control for multiple comparisons. bP<0.01 vs. normal control mice. FRB, fermented rice bran; FSB, fermented soybean cake; FSC, fermented sesame seed cake; UVB, ultraviolet B; RA, retinoic acid.



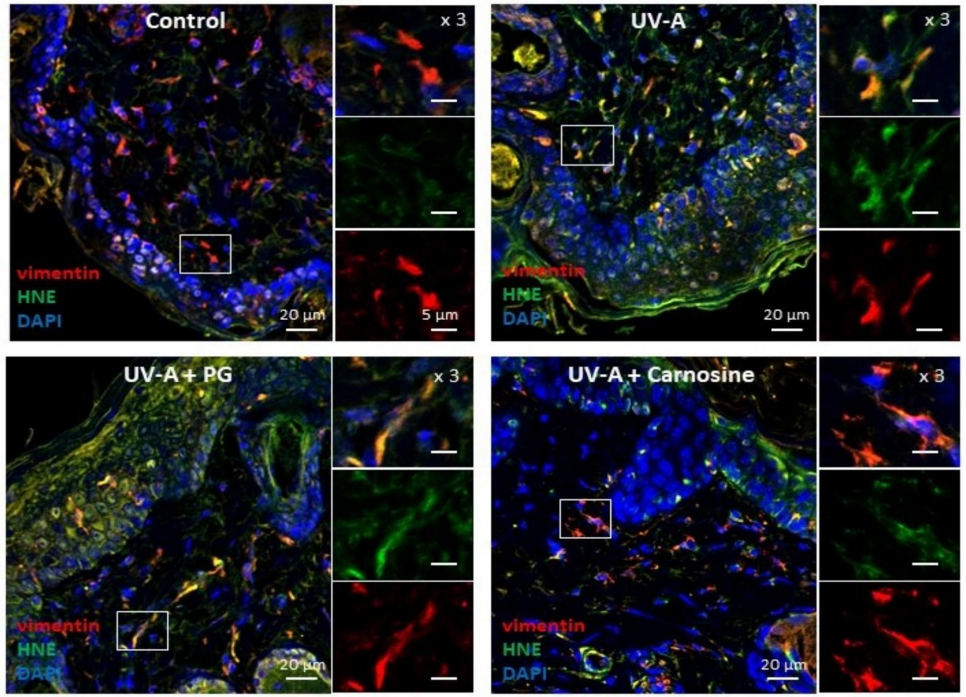


Figure 1. Expression of HNE adducts in fibroblasts in the skins of UV-A-exposed hairless mice. Immunofluorescence pictures (and higher magnification ×3 of each staining on the right of each picture) showing the presence of vimentin in fibroblasts (red), HNE adducts (green), and the merge (yellow). Upper left panel, control (untreated); Upper right panel, UV-A irradiated (20 J/cm^2/d up to 600 J/cm^2); lower left panel, propylene glycol (PG)-treated UV-A irradiated; lower right panel, carnosine (1% in PG)-treated/UV-A irradiated, as indicated in [32]. Scale bar, 20 µm, magnification 5 µm. Nuclei were stained with DAPI (blue). These data are representative of five separate experiments.