# *MMP1* Promoter Variation, Collagen Homeostasis, and Mechanism-Based Interventions in Skin Aging (Literature Review)

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## ABSTRACT

**Background:**
Dermal firmness and structural resilience depend on balanced collagen turnover. In aging skin, this balance is progressively disrupted by increased matrix degradation and insufficient replacement of newly synthesized collagen. Because MMP1 is a key collagenolytic enzyme in human skin, functional variation in the *MMP1* promoter represents a plausible determinant of interindividual differences in cutaneous aging susceptibility (Fisher et al., 1999; Quan et al., 2002; Quan et al., 2004).

**Methods:**
A narrative review was undertaken to integrate evidence on *MMP1*-associated collagen turnover in skin aging. Particular emphasis was placed on experimental work addressing rs1799750, studies linking MMP-1 to dermal collagen remodeling and cutaneous aging, and investigations of selected bioactives as modulators of matrix metalloproteinase activity, procollagen synthesis, tissue inhibitor or metalloproteinase regulation, oxidative stress, and ultraviolet-induced injury (Fujimoto et al., 2002; Tower et al., 2002; Tower et al., 2003; Nusgens et al., 2001; Hwang et al., 2014).

**Results:**
The literature supports a central role for MMP-1 in dermal collagen remodeling and indicates that rs1799750 is a functional promoter polymorphism. The 2G allele is associated with higher promoter activity and greater inducible *MMP1* expression than the 1G allele. This offers a plausible biological explanation for genotype-dependent differences in collagen degradation. Among candidate interventions, the strongest mechanistic support was identified for lutein, vitamin C, vitamin E, and selected phytosterols (Fujimoto et al., 2002; Tower et al., 2002; Tower et al., 2003; Philips et al., 2007a; Nusgens et al., 2001; Ricciarelli et al., 1999; Hwang et al., 2014; Beitner, 2003).

**Discussion**:
Current evidence supports the view that *MMP1* promoter variation functions as a susceptibility factor rather than a deterministic cause of skin aging. Its phenotypic effect is likely magnified by ultraviolet exposure, oxidative stress, and chronic inflammatory

signaling. A mechanism-based preventive strategy in higher-expression genotypes should therefore aim to reduce inducible MMP-1 activity while preserving the replacement arm of collagen homeostasis through support of procollagen synthesis and matrix-protective signaling (Fisher et al., 1999; Quan et al., 2004; Fisher et al., 2009).

**Subjects** Genetics, Beauty **Keywords:** Genetics, Polymorphism, Beauty, Collagen

# INTRODUCTION

Skin aging reflects cumulative alterations in the extracellular matrix, especially within the collagen-rich dermis. Structural integrity depends on coordinated turnover: damaged collagen must be removed, but this process must remain proportionate to the synthesis and maturation of replacement collagen. Photoaged skin is characterized by a persistent shift in this balance toward matrix loss, driven by matrix metalloproteinase induction and impaired transforming growth factor-β/Smad-dependent procollagen synthesis (Fisher et al., 1999; Quan et al., 2002; Quan et al., 2004).

Within this framework, MMP-1 is of particular importance because it initiates cleavage of native fibrillar collagens, including the principal dermal collagens that determine firmness and tensile strength. Excessive or prolonged MMP-1 activity is therefore expected to accelerate collagen fragmentation, reduce dermal mechanical competence, and promote visible features of skin aging. Interindividual differences in *MMP1* regulation may arise in part from common promoter polymorphisms, among which rs1799750 has been most extensively studied in functional terms.

The present review examines the biological role of *MMP1* in skin aging, the functional significance of rs1799750, the likely physiological consequences of enhanced *MMP1* expression for dermal collagen homeostasis, and the evidence supporting selected nutritional and topical compounds as mechanism-based modulators of this pathway.

## *MMP1* and Collagen Homeostasis in Human Skin

Physiological collagen degradation is not intrinsically harmful. Controlled removal of aged or damaged extracellular matrix is necessary for tissue renewal, wound repair, and structural adaptation. Pathology emerges when degradation persistently exceeds replacement. In human skin, ultraviolet irradiation promotes this imbalance by increasing transcription of matrix-degrading enzymes while simultaneously suppressing pathways required for new collagen synthesis (Fisher et al., 1999; Quan et al., 2002; Quan et al., 2004).

This imbalance may also become self-reinforcing. In aged human skin, fragmented collagen fibrils impair fibroblast function, increase oxidative stress, and further elevate MMP-1 expression, thereby amplifying extracellular-matrix deterioration (Fisher et al., 2009). Consistent with this view, age-related increases in collagenase expression in fibroblasts can be attenuated by alpha-tocopherol, indicating that oxidative and protein kinase C-dependent signaling contribute to dysregulated collagenolysis during aging (Ricciarelli et al., 1999).

Accordingly, the biologically relevant question is not whether collagen breakdown occurs, but whether MMP-1 activity remains proportionate to the skin's capacity for matrix replacement. A genotype that increases inducible MMP1 transcription would be expected to become most relevant under conditions that also drive oxidative stress, AP-1 activation, and inflammatory signaling.

## Functional Significance of *MMP1* rs1799750

The polymorphism rs1799750 is a functional guanine insertion/deletion in the *MMP1* promoter at position -1607 and is conventionally described as the 1G/2G variant. Functional studies indicate that the 2G allele creates an Ets-binding site adjacent to an AP-1-responsive region, thereby increasing promoter activity and enhancing transcriptional responsiveness to upstream signaling compared with the 1G allele (Fujimoto et al., 2002; Tower et al., 2002; Tower et al., 2003).

This molecular architecture provides a coherent explanation for genotype-dependent differences in MMP-1 expression. In promoter assays and stimulated cell systems, the 2G allele shows greater transcriptional activity than the 1G allele, particularly in signaling contexts that engage ERK/AP-1 pathways (Fujimoto et al., 2002; Tower et al., 2002; Tower et al., 2003). From a physiological perspective, these findings support a genotype-dependent effect on inducible collagenolysis, with carriage of the 2G allele expected to confer greater transcriptional responsiveness than the 1G/1G genotype (Fujimoto et al., 2002; Tower et al., 2002; Tower et al., 2003)

Importantly, rs1799750 should not be interpreted as a deterministic lesion. Rather, it is better understood as a regulatory susceptibility variant whose phenotypic effect depends on promoter context and upstream signaling pathways (Fujimoto et al., 2002; Tower et al., 2002; Tower et al., 2003; Vierkötter et al., 2015). In the context of skin, the expected consequence is not constitutive collagen destruction in isolation, but an increased propensity for MMP-1 induction under ultraviolet and oxidative stress, with AP-1-associated inflammatory signaling further contributing to this response (Fisher et al., 1999; Fisher et al., 2009; Hwang et al., 2014)

**Table 1**: **Genetic and Functional Polymorphism Studies Relevant to MMP1-Associated Collagen Remodeling and Tissue Aging**

| Study (Author, Year) | Study Design | Population (Size, Characteristics) | SNP(s) Investigated | Primary Outcome / Key Findings |
|---|---|---|---|---|
| **Fujimoto et al., 2002** | Functional promoter study with genotype–phenotype association | Amnion mesenchymal cells, WISH amnion cells, and fetal samples from pregnancies with preterm premature rupture of membranes | *MMP1* -1607 1G/2G (rs1799750) | Demonstrated that the 2G allele creates an Ets-binding site, shows higher promoter activity than the 1G allele, and is associated with stronger inducible MMP1 expression. |
| **Tower et al., 2002** | Cell-based functional promoter study | Human melanoma cells | *MMP1* -1607 1G/2G (rs1799750) | Showed that ERK1/2 signaling differentially regulates 1G and 2G promoter constructs, supporting higher transcriptional responsiveness of the 2G allele. |
| **Tower et al., 2003** | Cell-based functional promotor study | MCF-7/ADR breast cancer cells | *MMP1* -1607 1G/2G (rs1799750) | Confirmed that the 2G promoter variant contributes to high MMP1 transcription in a stimulus-responsive cellular context. |

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| **Vierkötter et al., 2015** | Cohort/cross-sectional genetic association study | 697 elderly women from the SALIA cohort | *MMP1* rs1799750 | Reported that wrinkle severity and airflow obstruction clustered in carriers of the MMP1 2G promoter variants, suggesting shared extracellular-matrix aging susceptibility. |
| **Ferrari et al., 2012** | Case-control genetic association study | 233 women: 137 pelvic organ prolapse cases and 96 matched controls | *MMP1* rs1799750 | Found that the MMP1 promoter polymorphism was associated with susceptibility to pelvic organ prolapse, supporting broader connective-tissue relevance of altered MMP-mediated remodeling. |
| **Diniz-Fernandes et al., 2018** | Case-control histologic-genetic study | 22 patients with posterior tibial tendinopathy and 5 controls | *MMP1* rs1799750; | Linked MMP1 polymorphisms with increased remodeling of collagen III and V in degenerative tendon tissue, suggesting reduced extracellular-matrix stability in permissive genotypes. |

## Consequences of Heightened *MMP1* Activity for Skin Aging

Numerous studies point towards biological plausibility of rs1799750 and visible skin aging. In a cohort of elderly women, promoter variants in *MMP1* were interpreted as markers of a shared susceptibility to skin aging, suggesting that extracellular-matrix aging phenotypes may cluster across tissues in individuals with permissive promoter backgrounds (Vierkötter et al., 2015).

Additional support comes from non-cutaneous connective tissues. Associations between matrix metalloproteinase gene polymorphisms and pelvic organ prolapse indicate that altered collagen-remodeling balance may contribute to reduced tissue support in collagen-dependent structures (Ferrari et al., 2012). Likewise, *MMP1* variants have been linked to remodeling collagen III and V in posterior tibial tendinopathy, further supporting the concept that genetically altered metalloproteinase activity can shape extracellular-matrix vulnerability (Diniz-Fernandes et al., 2018). Although such studies do not establish a skin-specific phenotype by themselves, they reinforce the broader biological relevance of *MMP1*-dependent tissue remodeling.

For skin aging, the mechanistic implication is clear: a higher-expression *MMP1* promoter genotype is most likely to matter when dermal collagen is already under stress. Chronic ultraviolet exposure, oxidative injury, and low-grade inflammation can increase MMP transcription and impair collagen replacement, thereby allowing a promoter variant with greater inducibility to shift the balance further toward net matrix loss (Fisher et al., 1999; Quan et al., 2004; Fisher et al., 2009). In this setting, accelerated collagen fragmentation may contribute to loss of firmness, reduced structural resilience, and progressive wrinkle formation.

## Interventions for *MMP1*-Mediated Matrix Loss

A rational intervention strategy for higher-expression *MMP1* genotypes should not aim to abolish collagen turnover, which is necessary for tissue renewal. Instead, it should seek to restore equilibrium between matrix degradation and matrix replacement. This favors compounds that reduce inducible MMP-1 signaling, dampen oxidative stress and AP-1 activation, or enhance procollagen synthesis and tissue inhibitor of metalloproteinase expression.

### Lutein

Among the candidate compounds, lutein is supported by a relatively coherent body of mechanistic evidence. Xanthophyll esters are detectable in human skin, demonstrating that lutein reaches the target tissue and is therefore biologically plausible as a cutaneous modulator (Wingerath et al., 1998). In dermal fibroblasts, lutein suppresses MMP-1 expression at the transcriptional level and favorably influences extracellular-matrix remodeling under ultraviolet-stressed conditions (Philips et al., 2007a). In chronically UVB-irradiated hairless mice, dietary lutein and zeaxanthin partially reduced photoaging and photocarcinogenesis (Astner et al., 2007). In human subjects, combined oral and topical lutein/zeaxanthin improved several skin physiology parameters in a placebo-controlled setting (Palombo et al., 2007).

Taken together, these findings position lutein as one of the most mechanistically relevant compounds for a phenotype characterized by excessive MMP-1 induction. Its value appears to lie in both tissue availability and direct modulation of pathways involved in ultraviolet-associated matrix degradation.

### Vitamin C

Vitamin C is particularly important for the replacement arm of collagen homeostasis. In human dermis, topical ascorbate increased mRNA levels for collagens I and III, the enzymes required for procollagen processing, and tissue inhibitor of metalloproteinase-1, indicating a coordinated enhancement of matrix-supportive processes rather than nonspecific antioxidant activity alone (Nusgens et al., 2001). This is especially relevant in settings where collagen degradation is increased, because support of replacement synthesis may help restore balance even if MMP-1 is not completely suppressed.

At the same time, ascorbate biology is context dependent. Reciprocal effects on matrix metalloproteinase expression and transforming growth factor-β pathways have been described in transformed cell systems, suggesting that dose, redox environment, and cell state influence biological response (Philips et al., 2007b). In the context of skin aging, however, the strongest evidence supports vitamin C as a promoter of procollagen synthesis and dermal matrix maintenance rather than as a direct inhibitor of *MMP1* transcription.

## Vitamin E

Vitamin E is relevant to collagen homeostasis through its effects on oxidative signaling and collagenase regulation. In fibroblasts, alpha-tocopherol reduced age-dependent collagenase expression through inhibition of protein kinase C, directly linking vitamin E biology to a pathway implicated in matrix degradation (Ricciarelli et al., 1999). In human studies, combined systemic supplementation with vitamins C and E reduced sunburn reactivity, indicating that these antioxidants can attenuate ultraviolet-induced skin injury in vivo (Eberlein-König et al., 1998). A broader antioxidant combination containing vitamins C and E, carotenoids, selenium, and proanthocyanidins also improved photoprotection in UV-irradiated human skin (Greul et al., 2002).

Additional mechanistic support comes from cellular aging models in which tocopherol and tocotrienol-rich fractions enhanced collagen synthesis and reduced collagen degradation-related gene expression in stress-induced premature senescence of human diploid fibroblasts (Makpol et al., 2013). The overall evidence supports its role as an upstream modulator of the oxidative signaling environment that favors excessive MMP activity.

## Alpha-lipoic Acid

Alpha-lipoic acid has been investigated primarily as a topical antioxidant in photoaged skin. In a randomized, placebo-controlled, double-blind trial, a cream containing 5% alpha-lipoic acid improved clinical parameters of facial photoaging after 12 weeks of treatment (Beitner, 2003). Its supportive role remains biologically plausible because ultraviolet-induced matrix degradation is closely linked to oxidative stress, and collagen fragmentation further amplifies intracellular oxidant levels and MMP-1 expression in dermal fibroblasts (Fisher et al., 1999; Fisher et al., 2009).

Alpha-lipoic acid's use is therefore best justified as a supportive adjunct within a broader matrix-protective strategy aimed at attenuating upstream oxidative stress and limiting secondary amplification of collagenolytic signaling, rather than as a targeted modulator of rs1799750-specific transcriptional regulation (Beitner, 2003; Fisher et al., 2009).

## Phytosterols

Phytosterols are among the most interesting candidates because several studies directly connect them to *MMP* and procollagen regulation in ultraviolet-stressed skin models. A glycosidic spinasterol promoted procollagen production and inhibited MMP-1 expression in UVB-irradiated human dermal fibroblasts, indicating a dual effect on both degradation and replacement pathways (Lee et al., 2011). Fucosterol suppressed matrix metalloproteinase expression and increased type I procollagen production in UVB-induced HaCaT keratinocytes (Kim et al., 2013). In human dermal fibroblasts, fucosterol additionally reduced MMP-1, IL-6, c-Jun, and c-Fos while increasing TGF-β1 and type I procollagen, thereby linking its activity to both AP-1 suppression and restoration of collagen-synthetic signaling (Hwang et al., 2014).

This combination of effects is highly relevant to a higher-expression *MMP1* genotype. Rather than acting solely as a general antioxidant, phytosterols appear capable of modulating the precise signaling nodes that connect ultraviolet stress to matrix degradation.

### Clinical Interpretation

The cumulative evidence supports a mechanism-based rationale for emphasizing lutein, vitamin C, vitamin E, alpha-lipoic acid, and selected phytosterols in individuals with a biologically plausible tendency toward increased *MMP1* inducibility. (Philips et al., 2007a; Nusgens et al., 2001; Ricciarelli et al., 1999; Lee et al., 2011; Hwang et al., 2014).

It is also important to distinguish pathway-based plausibility from direct pharmacogenetic proof. No genotype-stratified randomized trials were identified showing that carriers of the higher-expression rs1799750 genotype derive greater benefit from these compounds than non-carriers. The available evidence therefore supports biologically informed personalization, but not definitive claims of genotype-specific treatment efficacy.

Finally, because ultraviolet exposure is a major upstream driver of dermal MMP induction and impaired collagen replacement, any mechanism-based intervention strategy should be viewed as complementary to rigorous photoprotection rather than as a substitute for it (Fisher et al., 1999; Quan et al., 2004). In practical terms, the most rational approach for individuals with greater expected *MMP1* responsiveness is likely to combine environmental exposure control with agents that reduce inducible MMP signaling and reinforce the synthesis arm of collagen homeostasis.

## CONCLUSION

*MMP1* plays a central role in dermal collagen remodeling, and rs1799750 represents a functional promoter polymorphism with clear biological relevance to extracellular-matrix regulation. The higher-expression 2G allele increases transcriptional responsiveness through Ets/AP-1-dependent mechanisms and is therefore a plausible contributor to accelerated collagen degradation under ultraviolet, oxidative, and inflammatory stress. In skin aging, this genetic background is best understood as a susceptibility factor that may amplify matrix loss when environmental stressors are present. Current evidence supports a mechanistically coherent preventive strategy aimed at restoring collagen homeostasis rather than indiscriminately suppressing matrix turnover.

From a translational perspective, this framework also provides a rationale for gene-informed supplementation: when genetic variation predicts greater inducibility of MMP1, nutritional and topical interventions may be prioritized according to their capacity to attenuate ultraviolet- and oxidative stress-responsive signaling, limit secondary amplification of collagenolysis, and support procollagen synthesis and TIMP regulation (Quan et al., 2004; Philips et al., 2007a; Nusgens et al., 2001; Ricciarelli et al., 1999; Hwang et al., 2014). In this context, supplementation can be regarded as a biologically targeted approach intended to counterbalance a genetically favored shift

toward matrix degradation. Lutein, vitamin C, vitamin E, and selected phytosterols show the strongest pathway-level evidence for opposing this shift by suppressing MMP-related signaling and/or reinforcing the replacement arm of collagen homeostasis, whereas alpha-lipoic acid appears most appropriately positioned as an adjunctive antioxidant within a broader matrix-protective strategy (Philips et al., 2007a; Nusgens et al., 2001; Makpol et al., 2013; Lee et al., 2011; Hwang et al., 2014; Beitner, 2003). The functional character of rs1799750 supports the view that supplementation is likely to be most meaningful when aligned with a biologically relevant genetic background rather than applied empirically alone (Fujimoto et al., 2002; Tower et al., 2002; Tower et al., 2003; Vierkötter et al., 2015).

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