SA-beta-gal activity and relieving of G1/G0 cell cycle arrest. UVB-induced increased protein expression of cyclin-dependent kinase (CDK) inhibitors p21(WAF) (1) and p16(INK) (4) was also repressed by salidrosdie treatment in a dose-dependent manner. Meanwhile, the increment of malondialdehyde (MDA) level in UVB-irradiated HDFs was inhibited upon salidroside treatment. Additionally, salidroside significantly attenuated UVB-induced synthesis of MMP-1 as well as the production of IL-6 and TNF-alpha in HDFs. CONCLUSION: Our data provided the evidences for the protective role of salidroside against UVB-induced premature senescence in HDFs probably via its anti-oxidative property and inhibition on production of MMP-1 and pro-inflammatory cytokines, which indicated its potential utilization as an active ingredient in the preparation of photoprotective formulation.

Menghini, L. et al. (2010). "Antiproliferative, protective and antioxidant effects of artichoke, dandelion, turmeric and rosemary extracts and their formulation." *Int J Immunopathol Pharmacol* 23(2):601–610.

Artichoke, dandelion, turmeric extracts and rosemary essential oil are commonly used as ingredients in many herbal preparations to treat hepatic and gallbladder disorders. In the present work we compare the activity of each single extract with a commercial mixture for antiproliferative, antiradical and protective effects against induced oxidant stress effect. In ABTS and DPPH tests, turmeric extract is the most active, followed by artichoke and dandelion. All samples exhibited antiproliferative activity in a dose-dependent manner against HepG2 cells. In the same cell lines, the protective effect of pre-treatment with the extracts were detected by evaluating the prostaglandin E2 release, a marker of oxidative stress induced by hydrogen peroxide. The treatments with the extracts were efficient in reducing the release of PGE2 induced by oxidative stimulus. The positive results of the cell viability test, together with the protective and antiradical activity confirm the rationale for the use of these ingredients in commercial formulations as a health aid tool in modern phytotherapy.

Moon, S. Y. et al. (2014). "Tryptanthrin protects hepatocytes against oxidative stress via activation of the extracellular signal-regulated kinase/NF-E2-related factor 2 pathway." *Biol Pharm Bull* 37(10):1633–1640.

Tryptanthrin [6,12-dihydro-6,12-dioxoindolo-(2,1-b)-quinazoline], originally isolated from Isatidis radix, has been characterized as having anti-microbial and anti-tumor activities. It is well-known that excess oxidative stress is one of the major factors causing cell damage in the liver. This study investigated the cytoprotective effects and molecular mechanism of tryptanthrin against tert-butyl hydroperoxide (tBHP)-induced oxidative stress in human hepatocyte-derived HepG2 cells. Tryptanthrin pretreatment blocked the reactive oxygen species production, mitochondrial dysfunction, and cell death induced by tBHP. Moreover, tryptanthrin reversed tBHP-induced GSH reduction. This study also confirmed the activation of nuclear factor erythroid 2-related factor 2 (Nrf2) by tryptanthrin as a plausible molecular mechanism for its cytoprotective effects. Specifically, tryptanthrin treatment induced nuclear translocation and transactivation of Nrf2 as well as phosphorylation of extracellular signal-regulated kinase (ERK), a potential upstream kinase of Nrf2. Tryptanthrin also up-regulated the expression of the heme oxygenase 1 and glutamate-cysteine ligase catalytic subunits, which are representative target genes of Nrf2. Moreover, inhibitor of ERK was used to verify the important role of the ERK-Nrf2 pathway in