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Review on Use of Niacinamide and Kojic Acid - A Skin-Lightening Agent Ingredients

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ABSTRACT

Nicotinamide, also known as niacinamide, is the amide form of vitamin B_3. It is a precursor of essential coenzymes for numerous reactions in the body including adenosine triphosphate (ATP) production. The concentration for most formulations are 5 percent or less which is effective in treating hyperpigmentation and damage related to sun exposure. Kojic acid can act as an anti-oxidant, antiseptic, and preservative. Kojic acid can be found in a number of different types of cosmetic products, including powders, serums, creams, cleansers, and soaps. The Cosmeceutical Ingredient Review (CIR) indicates that this ingredient can be safely used at a concentration not higher than 1% due to its cytotoxicity. It is shown to be helpful in the treatment of hyperpigmentation disorders, such as freckles, age spots, post-inflammatory hyperpigmentation, and melasma.

NIACINAMIDE

INTRODUCTION [1-3]

Niacinamide, also called nicotinamide, is a form of vitamin B_3. It's found in many foods including meat, fish, milk, eggs, green vegetables, and cereals. Niacinamide is required for the function of fats and sugars in the body and to maintain healthy cells.

Niacin is a component of important coenzymes involved in hydrogen transfer. Here, the two codehydrogenases nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP) are of central importance. NADP is produced from NAD by phosphory-lation using an NAD- kinase and ATP. There are three ways in which biosynthesis of NAD from niacin occurs:

- Starting with nicotinic acid;
- From niacinamide;
- Biosynthesis from l-tryptophan via quinoline acid.

NAD and NADP are the coenzymes of countless dehydrogenases. NAD-dependent dehydrogenases are found mainly in the mitochondria. Their main function is to supply hydrogen to the respiratory chain for oxidation and energy production. NADP-dependent dehydrogenases are mainly localized in the cytosol. The most important function of the NADP system is to provide hydrogen for reductive biosyntheses such as in fatty acid synthesis, cholesterol synthesis and hydroxylations. The lipid-reducing agent of niacinamide is explained by its direct influence on adipocytes and spleen cells via a G-protein-coupled receptor. This results in the inhibition of lipolysis. At pharmacological concentrations, niacinamide can lead to modification of the various lipoprotein fractions. This, in turn, leads to an increase in HDL and simultaneous reduction of LDL and triglycerides.

CHEMISTRY [4]

Structure =

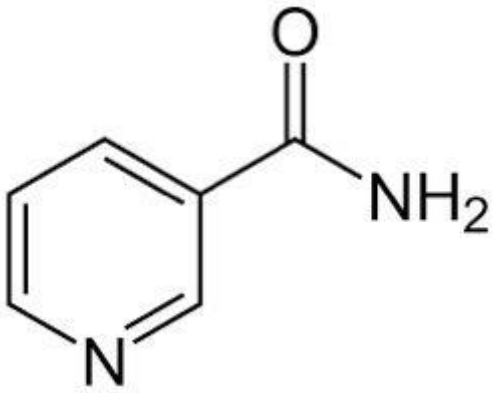

Figure 1.1: Structure of Niacinamide

Molecular Formula = $C_6H_6N_2O$

Synonyms = Nicotinamide, Nicobion, Nicotinsaureamid Jenapharm

IUPAC Name = pyridine-3-carboxamide

Molecular Weight = 122.12 g/mol

Physical description = White powder, solid

Form = White powder, needles from benzene

Taste = Bitter taste.

Odor = Odorless

Boiling Point = 302 to 320 °F at 760 mmHg

Melting Pont = 264 to 268 °F

Solubility = Very soluble in water (1 g is soluble in 1 mL water); soluble in butanol, chloroform

logP = -0.37

pH = 10% w/v solution in water is neutral to litmus

Refractive Index = 1.466 at 25 °C/D

Dissociation constants (pKa) = 3.35 (at 20 °C)

MECHANISM OF ACTION [5]

The anti-aging benefits of niacinamide are due in part to its ability to increase intracellular NAD and NADP, whose reduced forms (NADH and NADPH) function as antioxidants. Topical niacinamide increases collagen production, inhibits deposition of excessive glycosaminoglycans, and prevents protein glycation (Fig. 1.2). Glycation results in the cross-linking of collagen and elastin molecules, making them stiff and rigid, changing the visco elastic properties of the skin.

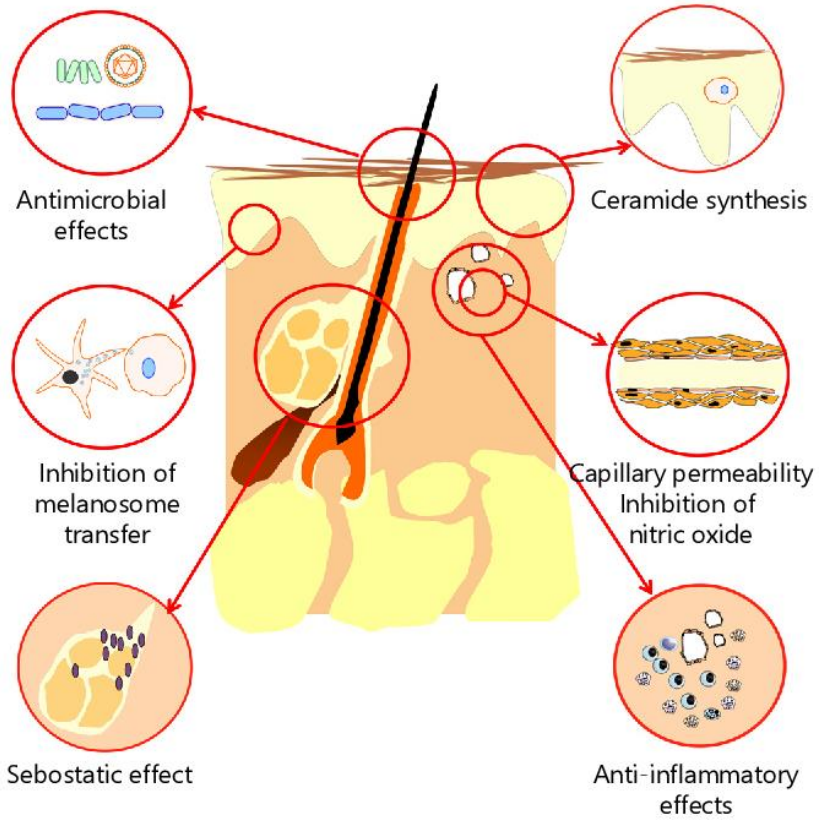

Figure 1.2: Mechanism and topical use of niacinamide

PHYSIOLOGICAL ROLE [23-25]

The substituted pyridine derivative niacin-amide is an essential constituent of the oxi-do reduction coenzymes nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP) (Fig. 1.3). During glycolysis and the TCA cycle, 10 molecules of NAD+ (per molecule of glucose) are reduced to 10 NADH by the transfer of a hydride ion to the 4-position of the niacinamide ring. The hydride ion of NADH serves

effectively as an energy storage unit, giving up a pair of high-energy electrons to the mitochondrial electron transport chain when needed. In this process of oxidative phosphorylation, electron pairs are transferred from NADH to a final acceptor (oxy-gen) via a series of electron carriers. This transfer of electrons is thermodynamically favorable, i.e., ΔG is negative, and is coupled to the pumping of protons out of the mitochondrial matrix. The flow of protons backs into the matrix, in turn, catalyzes the production of ATP by F0F1ATP-synthase. The total energy yield (ΔG') for this process is high (–52.7kcal). Whereas NADH is involved in catabolism, NADPH tends to serve as an electron (hydride ion) donor in anabolic processes, that is, biosyntheses. For example, NADPH is the reducing co-factor used by fatty acid synthetase in lipid biosynthesis and by desmolases and hydroxylases in steroid biosynthesis.

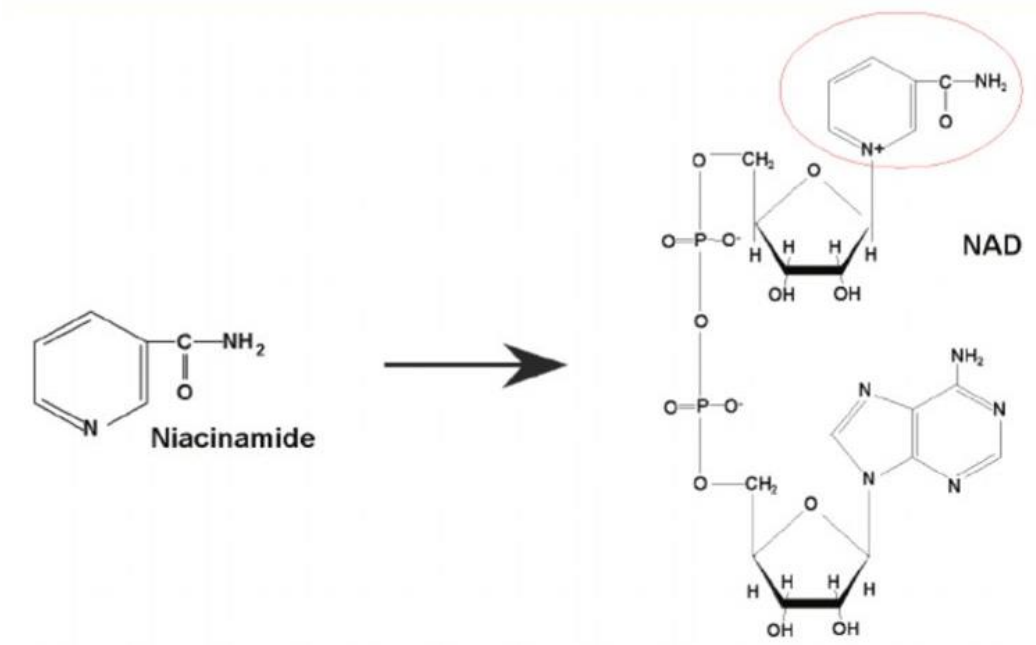

Figure 1.3: Structure of Niacinamide and inclusion within NAD molecule

USES

♦ Niacinamide and wound healing [6-7]

Myczkowski has shown, by split skin removal, that niacinamide induces an improvement in wound healing. Three groups were compared, one of which was treated with an ointment dressing. In the second group, a dry viscose dressing was used. The third group was given infusions with Complamin^{R} in addition to the viscose dressing. This is a nicotinic acid salt with a xanthine

corpus (xanthinol nicotinate). Using the infusion treatment, it was possible to reduce the time to wound healing from 15–17 days to 7–10 days.

Collins et al. investigated the postoperative healing of wounds caused by reconstructive plastic surgery and established better wound healing in the case of parenteral administration of niacinamide. In the case of mono-therapy with saline solution, the skin flaps were 45.67 % (± 31.14) vital. Niacinamide increased the vitality rate to 85.30 % (±9.24). The results are statistically highly significant (P <0.01). The figure 1.4 below shows the directions for using the serum and the ingredients which prepared it.

Figure 1.4: Directions for use and ingredients

♦ Niacinamide and skin aging [8]

Skin aging involves morphological and functional changes. The external appearance changes through atrophy and the development of wrinkles as a result of the decrease in epidermal cell layers and, as dermal components, from a reduction in protein and collagen synthesis. Reduced protein synthesis is reflected in keratin, filaggrin and involucrin. Keratin deficiency affects the epidermal cell structure and its water-binding capacity. Filaggrin is an antecedent of natural moisturizing factor (NMF). Involcurin seen as significant for the cell envelope and structure of the stratum corneum. In sum, the effects of reduced protein synthesis are poorer structure and reduced skin elasticity, as well as a decrease in the efficiency of the epidermal

barrier function with a reduction in horny layer moisture. The functional limitations of aging skin include reduced 'turnover' of the epidermis (slower epidermal cell cycle), which results from a deficiency of NADP in aging cells.

In studies on cell cultures, Oblong et al. found that in aging cells, niacinamide enabled NADP content to be increased to a level comparable with that of young cells. It was also possible to prove that niacinamide, as a precursor of NAD/ NADP, had a stimulatory effort on collagen synthesis, epidermal biopolymers (proteins) keratin, Filaggrin and involucrin. In general, niacinamide enabled improved dermal and epidermal cell growth. Thus, niacinamide is a therapeutic option in the case of age-related skin changes.

♦ Effect of niacinamide on the surface structure of the skin [9-10]

Using a multiple- angle reflectance spectrophotometer and in vivo tests of the back of the hand, Matts and Solechnik established a beneficial effect for the topical application of niacinamide in smoothing the surface structure of the skin. Using long-term application of an emulsion containing 2.5 % niacinamide, it was possible to correct the damage to the surface of the skin as a result of ageing. The results were statistically significant compared with the influence of the vehicle ($P<0.05$). Matts and Solechnik confirmed the results of two clinical studies carried out by Bissett et. al. In the context of a vehicle- controlled study, it was possible to show a smoothing of the skin surface structure and a reduction in wrinkle depth on the facial skin of women following 12 weeks topical treatment with niacinamide, results which differed statistically significantly from the influence of the vehicle. The studies were carried out with 5 and 2 % niacinamide. The results demonstrated that the effect of niacinamide was dose- dependent.

♦ Niacinamide and light damage to the skin [11-12]

UV radiation leads to the formation of reactive oxygen species (ROS) which are responsible for the creation of free radicals that lead to direct damage to DNA, lipids and proteins, i.e. mainly damage to membranes, and which are held responsible for photocarcinogenesis. All oxygen compounds that have reactivity greater than that of molecular oxygen are termed ROS. These include singlet oxygen (O_2), hydrogen superoxide (H_2O_2), the superoxide anion (O_2^-) and the hydroxyl radical (OH).

Shen et al. treated cell cultures with H2O2 and irradiated cells with UVC; both treatments led to cell death. Morphologically, there were signs of apoptosis, which is a typical finding in

photocarcinogenesis. By adding various concentrations of niacin, the morphological changes to the cells could be substantially reduced. Gensler, in an in vivo study in mice, inhibited photocarcinogenesis by the nutritive administration of niacinamide. There was a clear dose/ effect ratio between the quality of niacinamide administered and per cent light-provoked carcinoma development. Gensler noted that it was also possible to inhibit both photo-immunosuppression and photocarcinogenesis by the topical application of niacinamide. There are no fixed ideas on the mechanism by which niacinamide exerts its beneficial effects.

♦ Niacinamide in cases of hyperpigmentation [13-14]

In two studies, Hakozaki has shown a reduction in pigment disorders as a result of niacinamide. In the first study, facial pigment disorders in 18 Japanese women were treated on one side with 5% niacinamide and on the other side with vehicle only. The pigment disorders were evaluated qualitatively and quantitatively using high-resolution digital images and subjective judgments. In evaluation procedures, it was found that after 8 weeks of treatment, there was a significant lightening of hyperpigmentation as a result of niacinamide compared with the effect of the vehicle ($P < 0.05$). In a second study on 120 Japanese women, in the context of a three-part study using the same evaluation parameters as in the first study, comparisons were made among an SPF 15 sun protection cream with and without 2 % niacinamide and the relevant vehicle (Fig 1.5). As a result of niacinamide treatment, there was a lightening of the skin after 4 and 6 weeks, which was significantly better than with either the sun cream without niacinamide or the vehicle. In their study on the topical application of niacinamide on ageing skin, in addition to a stabilizing effect on the epidermal barrier function, Bissett et al. were able to establish a lightening of pigment disorders.[10] One possible explanation for this effect is given by Boissy et al. who established that melanosome transfer from melanocytes to keratinocytes was inhibited as a result of niacinamide treatment. These results justify the use of niacinamide both as a prophylactic and for the treatment of pigment disorders.

Figure 1.5: Niacinamide 10% + Zinc 1%

ANTIOXIDANT AND ANTI-INFLAMMATORY EFFECTS OF NICOTINAMIDE

♦ Antioxidant Properties of Nicotinamide [15-19]

Ingestion of nicotinamide prevents lipid peroxidation and normalizes the reduced antioxidants and antioxidant enzymes in experimental animal models, such as other antioxidants, e.g., α- tocopherol.

Kamat et al. showed that nicotinamide scavenged singlet oxygen at the rate constant of 1.8×10^8 M^{-1} s^{-1} and inhibited lipid peroxidation of rat liver microsomes induced by the photosensitized reaction of methylene blue irradiated with visible light in the presence of oxygen. They also showed that nicotinamide inhibited lipid peroxidation induced by NADPH/ ADP- Fe^{3+} in rat liver microsomes. Nicotinamide inhibited lipid peroxidation and protein oxidation (carbonylation) induced by the ascorbate- Fe^{2+} system in the rat brain mitochondria, whereas such action was not observed for nicotinic acid.

♦ Anti-Inflammatory Effects of Nicotinamide [20-22]

Nicotinamide suppressed interleukin (IL)- 8 production at the mRNA and protein levels through modulation of the nuclear factor (NF)- κB and mitogen-activated protein kinase (MAPK) pathways in HaCaT cells and primary keratinocytes stimulated by Propionibacterium acnes, the etiological agent causing inflammatory acne vulgaris. Nicotin-

amide down regulated the expression of IL-6, IL-10, monocyte chemoattractant protein- 1 and tumor necrosis factor (TNF)-α in UV-irradiated keratinocytes.

Nicotinamide attenuated the synthesis of inflammatory mediators, such as prostaglandin (PG) E2, IL-6, and IL-8 in human epidermal keratinocytes and in full-thickness three-dimensional skin organotypic models that were stimulated by UV radiation. In a clinical trial, pretreatment with 5% nicotinamide reduced erythema that was induced by UV radiation. Analysis of IL-1α and its receptor antagonist (IL-1αRA) ratios showed that nicotinamide significantly reduced the UV-induced inflammatory response, compared to the control sites.

NICOTINAMIDE COENZYMES IN SKIN ARE DEPLETED WITH AGE; NIACINAMIDE CAN HELP NORMALIZE THIS IMBALANCE [26]

NADH and NADPH can, thus, be viewed as fundamental energy currency units within cells, driving the metabolism of cells involved in both catabolic and anabolic processes. There is an increasing pool of evidence for a decline in systemic and intracellular concentrations of these two coenzymes with age in human and animal models and recent new data appear to confirm this. Oblong et al. established human dermal fibroblast cell lines from a 7-year-old and a 72-year-old and used these to measure endogenous NADPH/NADP+ ratios and total NADPH + NADP+ levels. It was found that fibroblasts from the aged donor contained decreased NADP redox ratios and total NADPH + NADP+ levels relative to those from the young donor (51% and 28% respectively). It does appear, therefore, that there is a reduction in nicotinamide coenzymes associated with senescence. Importantly, Oblong et al. also found that supplementation of human dermal fibroblast cultures derived from elderly donors with 14C-niacinamide and 14C-nicotinic acid (niacinamide precursor) increased intracellular concentrations of NADPH (Fig. 1.6). It appears that a localized supply of niacinamide, therefore, can be utilized by aged cutaneous cells to restore intracellular nicotinamide coenzyme homeostasis. It is worth noting, however, that despite the efficacy noted above, nicotinic acid (niacin) produces a well-documented cutaneous vasodilatation (»flushing«) when applied topically. Niacin, therefore, presents a challenge to cosmetic applications; there are no such issues with the use of niacinamide.

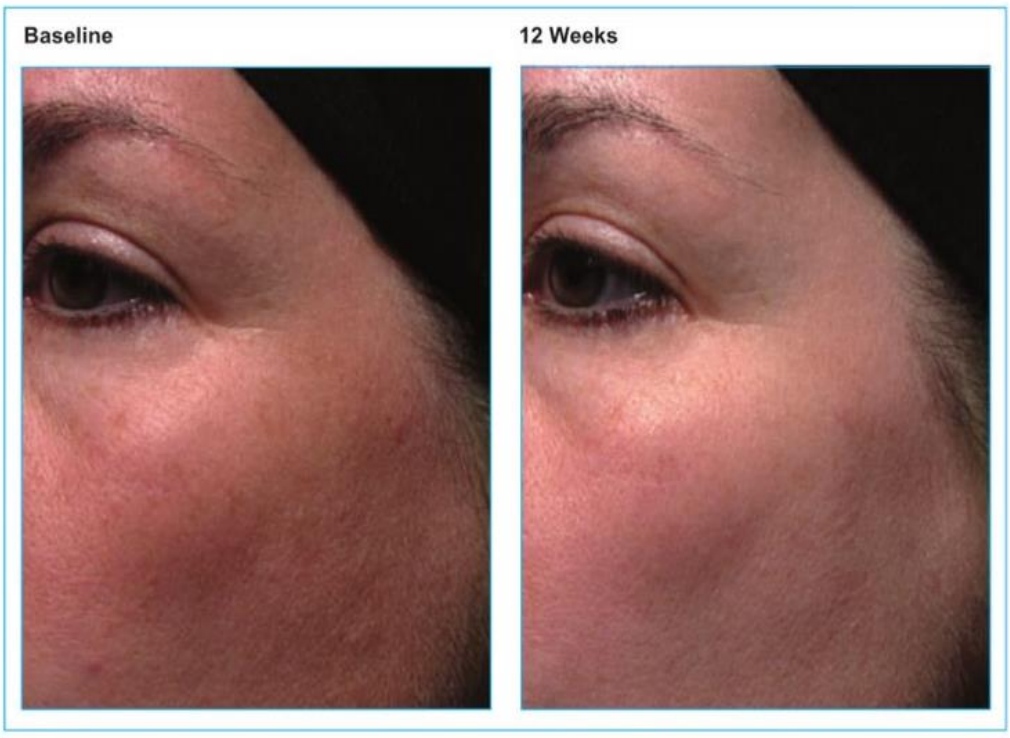

Figure 1.6: Same subject at baseline and after 12 weeks of topical treatment with 5 % niacinamide

SIDE EFFECTS [1]

When applied to the skin: Niacinamide is possibly safe. Niacinamide cream might cause mild burning, itching, or redness.

HOW AND WHEN TO USE? [27]

Niacinamide can be used in the morning and night (Fig 1.7).

Because it plays well with other skincare ingredients (even potentially tricky actives such as exfoliating acids and vitamin C) it will sit happily alongside anything used on the skin.

Acids such as glycolic, salicylic, and azelaic enhance the benefits of niacinamide on skin concerns such as uneven tone, dark spots, post-acne marks, and large pores. Results can be evident in just a few days.

Using niacinamide during the day can also help to protect the skin from sun damage; there's interesting evidence that shows niacinamide and SPF could be a dream team. Niacinamide can interrupt the terrible inflammation UV light causes in the skin, so using a niacinamide booster under the sunscreen is a great way to protect and repair.

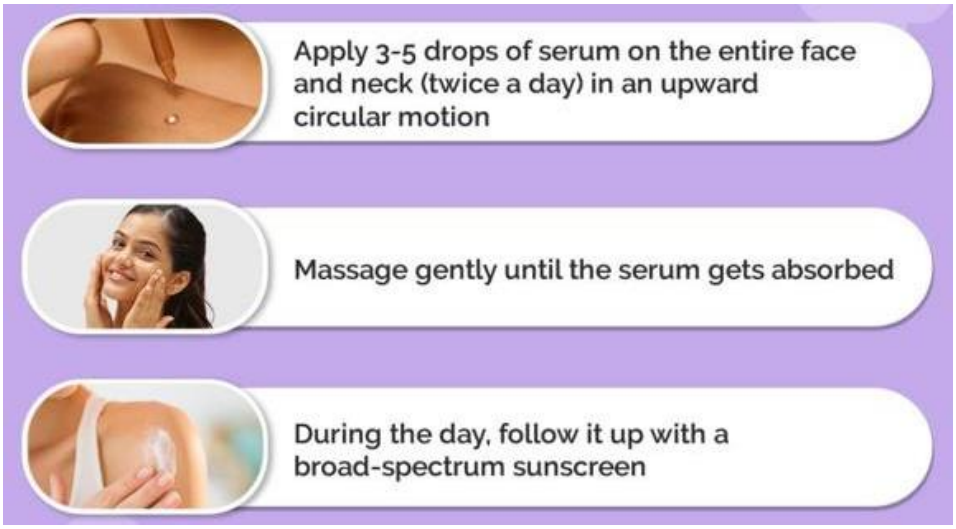

Figure 1.7: How to use

Jongkind, J.F., Verkerk, A., and Poot, M., Glucose flux through the hexose monophosphate shunt and NADP(H) levels during in vitro ageing of human skin fibroblasts, Gerontology 33 (5) (1987) 281-28.

2. KOJIC ACID

INTRODUCTION [28-32]

Kojic acid has mild antioxidant, antimicrobial, and exfoliating properties. Kojic acid is a chemical derived from mushrooms, but it can also be created during the sake brewing process from fermented rice. Because of its ability to really penetrate the layers of your skin and stop the production of melanin, kojic acid is usually sought out as a dark spot and age- spot-fading treatment and is often considered a less aggressive answer to hydroquinone.

KA inhibits tyrosinase and has been commonly researched in the cosmetic industry. KA and its derivatives have radioprotective, skin-lightening, anti-inflammatory, antioxidant, and anti-proliferative properties. Due to its tyrosinase inhibitory activity, KA can protect the skin from ultraviolet (UV) rays, reduce hyperpigmentation, and prevent melanin formation. It is produced by several types of fungi, and it is also a by-product of the fermentation process of certain foods, such as soy sauce and sake.

KA is incorporated in many kinds of cosmetic products. The CIR approved KA as safe at a concentration of 1% in cosmeceutical products. The existing dermatological safety data also

support the safety of KA at a concentration of 2% in cosmeceuticals, indicating that a limit of 2% might be applicable.

CHEMISTRY [33]

Structure =

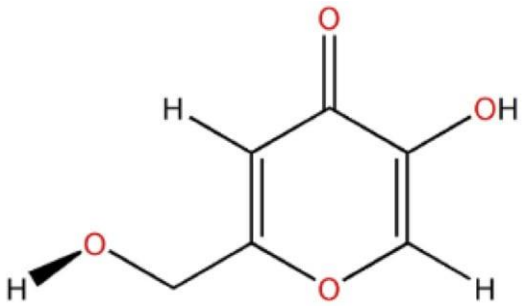

Figure 2.1: Kojic acid structure

Molecular Formula = $C_6H_6O_4$

IUPAC Name = 5-hydroxy-2-(hydroxymethyl) pyran-4-one

Molecular Weight = 142.11 g/mol

Physical description = Solid

Form = Prisms, needles from acetone

Melting Pont = 153.5 °C

Solubility = Soluble in water, acetone: slightly soluble in ether: insoluble in benzene

logP = -0.64

Dissociation constants (pKa) = 7.67 (at 25 °C)

MECHANISM OF ACTION [34-38]

KA is a kind of secondary metabolite, whose biosynthesis pathway continues to be uncertain to date. However, it is stated that it chelates divalent ions and acts as a tyrosinase inhibitor and a free radical scavenger. It works by chelating the copper (Cu^+) at the active site of the tyrosinase enzyme.[31] The tyrosinase enzyme, also known as polyphenol oxidase, limits the rate of melanin synthesis, and it is responsible for converting L-tyrosine to L-3–4

dihydroxyphenylalanine. It belongs to the type 3 copper-containing protein family, with two copper ions (CuA and CuB) in the active site. CuA and CUB catalyze the conversion of monophenols (e.g., tyrosine) into o-diphenols (monophenolase activity) followed by the oxidation of the o-diphenols to the resultant o-quinone derivatives (diphenolase activity) (Fig. 2.2).

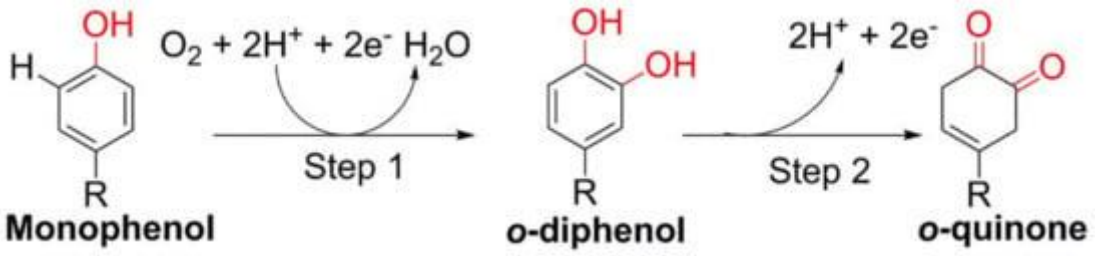

Figure 2.2: The reaction catalyzed by tyrosinase.

Low-pigmenting agents can be generally classified according to which step of melanin production is disrupted. This depends on whether the agents can act before, during, or after melanin production. KA acts during the actual synthesis of melanin, exhibiting a sufficient inhibitory effect on monophenolase activity and a varied inhibitory effect on the diphenolase activity of mushroom tyrosinase.

KOJIC ACID DERIVATIVES [39-43]

KA causes skin irritation, has inadequate inhibitory activity, and is not stable during storage, thus reducing its use in cosmetic products. To overcome these disadvantages, many derivatives of KA have been produced. These derivatives were produced to improve stability and solubility.

By modifying the alcoholic hydroxyl group of KA, it can be converted into an ester, glycoside, amino acid derivatives, hydroxyphenyl ether, or tripeptide derivatives. [29]

The KA derivatized through an ethylene linkage of the phosphonate with aldehyde using intermediates derived from KA is about eight times more effective in tyrosinase inhibitory activity than KA.

Recently, methods for the synthesis of a variety of KA derivatives, such as KA di-palmitate, KA ester, and KA laureate, have been reported. KA peptides have also been investigated as potent tyrosinase inhibitors.

COSMETIC APPLICATIONS OF KOJIC ACID [41, 44-45]

KA is a popular ingredient and is used by various industries globally. In the cosmetic industry, it is used as a topical treatment for skin conditions such as spots, melasma, and patches of light brown color resulting from post-inflammatory hyperpigmentation.

KA has skin-lightening properties and can act as a UV protector, whereby it prevents the development of hyperpigmentation in human skin by inhibiting the formation of melanin through the prevention of tyrosinase formation.

KA also enhances the shelf life of cosmetic products through its preservative properties. It is normally combined with alpha-hydroxy acid in the formulation of skin-lightening products to manage age spots and lightened freckles. Due to its manganese and zinc complexes, it can be used as a radioprotective agent against γ-ray. Figure (Fig. 2.3) below summarizes the above-discussed applications of KA.

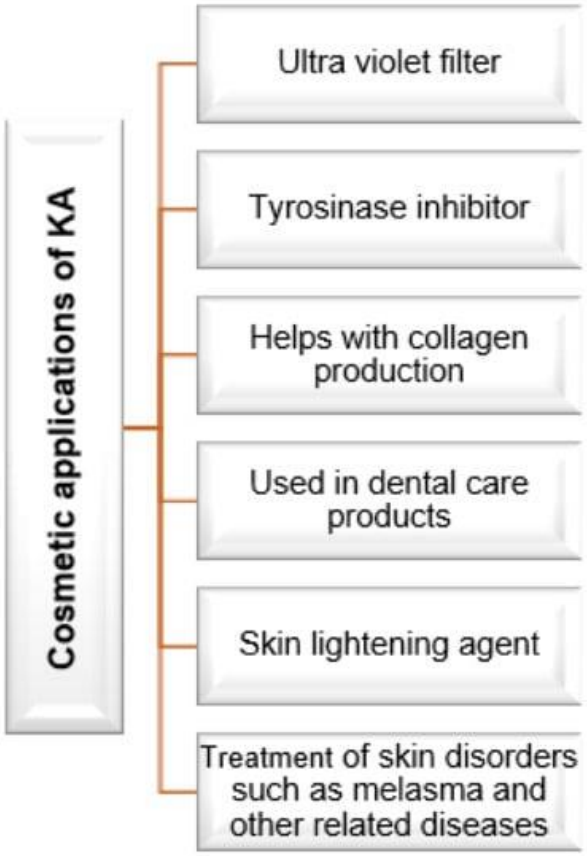

Figure 2.3: Cosmetic applications of Kojic acid

BIOLOGICAL ACTIVITIES OF KOJIC ACID

The available literature indicates that this ingredient has various biological activities, and they are listed below.

♦ Antibacterial and Antimicrobial Activity [29, 41, 46-48]

KA has antifungal and antibacterial properties. Preceding antimicrobial activity assays showed that KA was more active against Gram-negative bacteria than against Gram-positive bacteria. However, some of its derivatives have shown conflicting effects distinct from KA's antibacterial activity.

When used in cosmetic products, KA can prevent the growth of microorganisms and can be used as a preservative.

The antimicrobial activity of the ethyl acetate (EtOAc) extract of Colletotrichum gloeosporioides and its major compound KA were evaluated, and the results showed considerable antimicrobial activity against all tested strains. When tested against various microorganisms, KA was most active against Micrococcus luteus and least active against Pseudomonas aeruginosa.

Due to its antifungal properties, KA is incorporated into some antifungal products to improve their effectiveness. Furthermore, it could be useful in treating various fungal infections of the skin as well as yeast infections, ringworm, athlete's foot, and candidiasis.

KA and its derivatives have potent activity against bacteria such as Staphylococcus aureus. The KA derivatives were also validated for antifungal activities against Fusarium oxysporum, Rhizoctonia solani, and Pythium graminicola, which cause fungal infections such as fusarium wilt, sheath blight, and seedling blight. Besides its antibiotic properties, KA also shows some insecticidal activity against Spodoptera frugiperda and Heliothis zea insects.

♦ Antioxidant Activity [48-50]

KA has anti-oxidant properties and is used as a substitute for hydroquinone (HQ) for skin lightening by the cosmeceutical industry.

Studies by Zhang et al., (2017) showed that KA improved oxidative stress response in fungi, thus showing the anti-oxidant ability of this metabolite. Other preceding bioactivity studies on KA revealed that it has anti-oxidant properties.

The correlation between anti-melanogenic activity with oxidative effects of KA and KA esters was investigated by Lajis et al., 2012. The results of the study showed that both KA and its esters had mild free radical scavenging activities at concentrations ranging from 1.95 to 1000 μg/mL.

♦ Anti-Inflammatory Activity [51-52]

KA may exert slight anti-inflammatory effects that may favorably improve by subsequent derivation of chosen KA derivatives. To develop a safe anti-inflammatory compound, a derivative of KA and p-coumaric acid were synthesized, as they are known to have anti-inflammatory properties. The anti-inflammatory action of KA was enhanced by the addition of cinnamate moiety in p-coumaric acid as a hydrophobic part.

♦ Tyrosinase Inhibition Activity [29, 53-54]

KA is regarded as one of the best skin-lightening agents in the beauty industry. It exerts a slow and effective reversible inhibition of tyrosinase, thus preventing melanin formation, and also plays an important role in cellular melanin formation. It can be used as a monotherapy or combined with other agents. In Japan, this ingredient is known as a quasi-drug. Due to its ability to inhibit tyrosinase activity, KA has been used in several studies as a standard. [43] KA inhibits melanosis by interfering with the uptake of oxygen required for enzymatic browning. Figure below (Fig. 2.4) shows the directions for using the serum.

Figure 2.4: Directions to use

BENEFITS [55]

The benefits of using products containing kojic acid may include the following:

- **Anti-aging effect**: Products containing kojic acid may lighten the skin, which can improve the appearance of age spots and sun damage. The reduction of dark spots can have an anti-aging effect.

- **Treat melasma**: Kojic acid may also help decrease melasma, which is a darkening of the skin due to pregnancy.

- **Decrease the appearance of scars**: Kojic acid may also reduce the discoloration of scars. Although the acid does not improve the thickness of scar tissue, it may reduce dark pigmentation associated with certain types of scars. Lightening the scar may make it less noticeable.

- **Antifungal benefits**: Kojic acid is also thought to have some antifungal benefits. It may be helpful in preventing and treating certain fungal infections, such as athlete's foot and yeast infections.

- **Antibacterial effects**: Kojic acid may also provide antibacterial benefits. It may help decrease the chances of developing common types of bacterial skin infections.

It is thought that its fundamental role as a precursor of reduced nicotinamide coenzymes such as NADH and NADPH is pivotal to its observed effects. It is thought that its fundamental role as a precursor of reduced nicotinamide coenzymes such as NADH and NADPH is pivotal to its observed effects.

USES [55]

Kojic acid is mainly used in health and beauty products.

Kojic acid is sometimes used in health and beauty products to lighten the skin. It may be used to treat skin conditions, such as sun damage, scars, and age spots.

The science behind how kojic acid works as a lightning agent involves its effect on melanin production.

Melanin is a naturally occurring pigment in the body that gives the eyes, hair, and skins their color. An amino acid called tyrosine is needed to support the production of melanin.

Kojic acid works by blocking tyrosine from forming, which then prevents melanin production. Decreased melanin production may have a lightening effect on the skin.

Kojic acid is most commonly used in cosmetic products, such as creams, lotions, and serums. It is also used in some soap. Many products with kojic acid are intended for use on the hands or face.

Products containing kojic acid can also be used on other parts of the body, such as the legs and arms. The concentration of kojic acid in cosmetics is often between 1 and 4 percent.

Certain products containing kojic acid, such as serums, are meant to be applied to the skin and left on and absorbed. Some products, such as soaps, are applied and washed off.

CONCLUSION:

This review carried out an overview of niacinamide and kojic acid. It shows positive characteristics in dermatology. It treats many skin disorder. Niacinamide is used in cosmetic as anti- ageing, in hyperpigmentation, anti-oxidant. Niacinamide increases intra- cellular NAD and NADP from NADH & NADPH and act as anti- oxidant.

Kojic acid is a secondary metabolite and derived from mushrooms. K.A is used in removing dark spots, fading age- spots, skin- lightning, anti- oxidant. It is also used in cosmetic products for the enhancement of the shelf life.

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