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C. Diehl
Universitá Degli Studi Guglielmo Marconi, Rome, Italy

Use of nicotinamide in dermatology

Nicotinamide (synonym — niacinamide) is the water-soluble, amide isotype of vitamin B_3, whilst niacin (synonym — nicotinic acid) is the corresponding acid isotype. It is used in dermatology for a long time. It has numerous applications, and it seems useful to make an updated review of its multiple uses.

Nicotinamide is normally brought by our diet; meat and fish are very rich in niacinamide, which is less present in vegetables. The lack of this vitamin can cause pellagra, presenting with the triad of dementia, dermatitis and diarrhoea. Nicotinamide is the catalyst for multiple molecular reactions throughout the body, and is converted into several coenzymes, including nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP), which are of central importance for metabolism. NAD and NADP are the coenzymes of countless dehydrogenases involved in hydrogen transfer.

Their main function is to supply hydrogen to the respiratory chain of mitochondria for oxidation and energy production. Nicotinamide increases ATP production which increases DNA repair, protecting the skin from photodamage. In solar-simulated, UV-irradiated, niacinamide-treated human HaCaT keratinocytes, an increased amount and rate of DNA excision repair was demonstrated in cells treated with nicotinamide vs. control.

This review aims to describe the major dermatological disorders where nicotinamide was studied, mainly photoprotection and skin cancer prevention, deficiencies in skin barrier function — especially atopic dermatitis — pigmentation disorders, inflammatory diseases or acne. In all these indications, use of oral or topical nicotinamide is well-documented and offer interesting therapeutic perspectives.

In some other cases like wound healing, bullous or pruritic disorders or cosmetic indications, the use of nicotinamide seems also promising, but less documented. This is also the case regarding the antibacterial properties of nicotinamide. This review also contemplates the side effects of nicotinamide, which appear to be low and lack of severity, permitting to conclude that nicotinamide is worth of being an important weapon in the dermatologists' armamentarium.

Key words

Nicotinamide, niacinamide, side effects, photoprotection, skin cancer, skin barrier, acne, pigmentation.

Introduction

Nicotinamide is used in dermatology for a long time. It has numerous applications, and it seems useful to make an updated review of its multiple uses.

Nicotinamide (synonym: niacinamide) is the water-soluble, amide isotype of vitamin B_3, whilst niacin (synonym: nicotinic acid) is the corresponding acid isotype [1]. Nicotinamide is normally brought by our diet; meat and fish are very rich in niacinamide, which is less present in vegetables [2]. The lack of this vitamin can cause pellagra, presenting with the triad of dementia, dermatitis and diarrhoea. Nicotinamide is the catalyst for multiple molecular reactions throughout the body, and is converted into several coenzymes, including nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP), which are of central importance for metabolism. NAD and NADP are the coenzymes of countless dehydrogenases involved in hydrogen transfer. Their main function is to supply hydrogen to the respiratory chain of mitochondria for oxidation and energy production.

1. Generalities about use of niacinamide in dermatology

Niacinamide is a small molecule (Figure) whose molecular mass is 122.12 g/mol. According to the 500 Dalton rule for the skin penetration of chemical

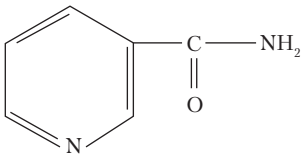

Figure. **Chemical structure of niacinamide**

ompounds and drugs [3], this allows skin absorption. Besides, Feldmann et al. [4] proved percutaneous penetration of a topical preparation of niacinamide in human skin. On the contrary, nicotinic acid is able to overcome the epidermal barrier function only with dificulty. For this reason, both systemic and topical use of niacinamide are popular in dermatology.

2. Nicotinamide for photoprotection and skin cancer prevention

Photoprotection and prophylaxis of skin cancer are probably the best documented indications of nicotinamide in dermatology.

2.1. Nicotinamide and photoprotection

As early as 1997 and 1999, Gensler et al revealed that topical as well as systemic nicotinamide prevented UV-induced immune suppression and tumour formation in mice [5, 6]. Later, investigators demonstrated that both topical and oral forms of nicotinamide had similar effects in reducing UV immune suppression [7—9] and the development of non- melanoma skin cancers [10] in humans. Exposure to UV radiation causes the development of skin cancers via a range of mechanisms including direct cell damage (via substitutions in DNA of pyrimidines, gene mutations and oxidative stress), the activation of local inflammatory responses and suppression of cutaneous anti-tumour immunity [9]. Nicotinamide increases ATP production which increases DNA repair, protecting the skin from photodamage [9].

2.2. Nicotinamide and DNA damage

Direct damage to DNA in human keratinocytes by UV results in the formation of cyclobutane pyrimidine dimers (CPDs) and pyrimidine (6—4) pyrimidine photoproducts [11]. These UV-induced CPDs give rise to C → T and CC → TT transitional mutations within DNA, increased levels of which are found in animal and human models of basal cell carcinoma and squamous cell carcinoma, and these UV-induced mutations can alter the function of the *p53* gene, an important tumour suppressor gene in skin carcinogenesis [9]. Such mutations of *p53* are an important step in the dysregulation of epidermal cell growth in the pathway leading to malignancy.

In solar-simulated, UV-irradiated, niacinamide-treated human HaCaT keratinocytes, an increased amount and rate of DNA excision repair was demonstrated in cells treated with nicotinamide *vs.* control [12]. It therefore appears that by augmenting access to ATP in UV-irradiated keratinocytes, DNA repair is increased, which would reduce both UV-induced immune suppression and the formation of mutations in oncogenes and tumour suppressor genes.

2.3. Nicotinamide and UV-induced inflammation

Ultraviolet (UV) radiation has profound effects on human skin, causing sunburn, inflammation, cellular-tissue injury, cell death, and skin cancer. Most of these effects are mediated by a number of cytokines produced by keratinocytes, in particular interleukin (IL)-1β, IL-6, IL-8, IL-10, monocyte chemoattractant protein (MCP)-1 and tumour necrosis factor (TNF)-α. In a study [13], it was investigated whether nicotinamide (NCT) might have a protective function in reducing the expression of these cytokines in UV-irradiated keratinocytes. Nicotinamide significantly downregulated IL-6, IL-10, MCP-1 and TNF-α mRNA expression, whereas it did not exert any significant effect on IL-1β or IL-8 expression. Based on this result, nicotinamide could be a possible therapy to improve or prevent conditions induced or aggravated by UV light. In vivo in humans, however, nicotinamide did not reduce the erythema caused by UV radiation, with two studies showing no significant change in minimal erythema dose in humans receiving nicotinamide orally at 500 mg or 1500 mg daily [9], nor when NAM was applied topically either before or immediately after low-dose UV exposure [7].

2.4. Nicotinamide and UV-induced immune suppression

UV irradiation diminishes local cutaneous immune responses even at very low, sub-erythemal doses [14] by a number of mechanisms, including reducing tumour antigen-presenting cell function [15] and increasing the production of immunomodulatory cytokines [16] which results in diminished UV-induced contact and delayed- type hypersensitivity responses [15]. Unrepaired DNA photolesions in the skin are a key trigger for UV-induced immune suppression [17]. Using the Mantoux model of skin immunity in healthy volunteers, oral nicotinamide was compared to placebo (both administered for 1 week) in a randomized, double-blinded, crossover design against the effects of solar-simulated UV radiation on delayed-type hypersensitivity to tuberculin purified protein derivative. Immunosuppression, calculated as the difference in Mantoux induced erythema of irradiated sites compared with unirradiated control sites, was determined in volunteers taking oral nicotinamide and placebo. Significant immunosuppression occurred in an UV dose-dependent manner in the presence of placebo. Oral nicotinamide, at doses of either 1500 or 500 mg daily, was well tolerated and significantly reduced UV immunosuppression with no

immune effects in unirradiated skin [9]. Importantly, nicotinamide did not alter immune responses at unirradiated sites [9] indicating that it acts to normalise cutaneous immune responses rather than boosting baseline immunity.

2.5. Nicotinamide and skin cancer prophylaxis

To determine whether oral nicotinamide, at different doses, reduced AKs in sun-damaged individuals, healthy, immune-competent volunteers with > 4 palpable actinic keratosis (AKs) (face, scalp and upper limbs) were recruited and assigned to take nicotinamide 500 mg or matched placebo twice daily (Study 1 — 35 volunteers) or once daily (Study 2 — 41 volunteers) for 4 months [12]. At baseline, 2 and 4 months, palpable AKs were identified visually and by touch by a blinded observer. A 35 % relative reduction in AK count at 4 months ($p = 0.0006$) was estimated from Study 1 (with similar results at 2 months). A 29 % relative reduction in AK count at 4 months ($p = 0.005$) was estimated from Study 2 (with smaller but significant differences observed at 2 months). For Studies 1 and 2 combined, 37 patients were randomized to placebo and 37 to nicotinamide. 81 and 79 % respectively of placebo and nicotinamide patients, respectively, had previous, histologically confirmed skin cancers. During the 4-month trials, 11 placebo patients developed 20 new skin cancers (12 basal cell carcinoma (BCC) and 8 SCC) and 2 nicotinamide patients developed 4 cancers (2 BCC and 2 SCC). The results of these phase II studies suggest nicotinamide is effective in reducing AKs and shows promise for skin cancer chemoprevention.

In a phase 3, double-blind, randomized, controlled trial [10], 386 participants who had had at least two nonmelanoma skin cancers in the previous 5 years were randomly assigned, in a 1 : 1 ratio, to receive 500 mg of nicotinamide twice daily or placebo for 12 months. Participants were evaluated at 3-month intervals for 18 months. The primary end point was the number of new nonmelanoma skin cancers (i.e., basal-cell carcinomas plus squamous-cell carcinomas) during the 12-month intervention period. Secondary end points included the number of new squamous-cell carcinomas and basal-cell carcinomas and the number of actinic keratoses during the 12-month intervention period, the number of nonmelanoma skin cancers in the 6-month postintervention period, and the safety of nicotinamide. At 12 months, the rate of new nonmelanoma skin cancers was lower by 23 % in the nicotinamide group than in the placebo group ($p = 0.02$). Similar differences were found between the nicotinamide group and the placebo group with respect to new basal-cell carcinomas (20 % lower rate with nicotinamide, $p = 0.12$) and new squamous-cell carcinomas (30 % lower rate, $p = 0.05$). The number of actinic keratoses was 11 % lower in the nicotinamide group than in the placebo group at 3 months ($p = 0.01$), 14 % lower at 6 months ($p < 0.001$), 20 % lower at 9 months ($p < 0.001$), and 13 % lower at 12 months ($p = 0.001$). the number or types of adverse events during the 12-month intervention period were similar in both groups. These results show that oral nicotinamide was safe and effective in reducing the rates of new nonmelanoma skin cancers and actinic keratoses in high-risk patients [10]. It is hypothesised that nicotinamide's ability to reduce the incidence of both BCC and SCC is thereby a result of its ability to enhance DNA repair, and boost skin immunity.

3. Nicotinamide and skin barrier function

In disorders such as in atopic dermatitis [18], ageing skin [19] and weather-induced xerosis of the skin in winter [20], there is a deficiency and related restriction of the epidermal barrier function, corresponding to an increase in trans-epidermal water loss (TEWL) and horny layer moisture deficiency. Using cell cultures of human keratinocytes, Tanno et al. [21] demonstrated that niacinamide led to improved differentiation of keratinocytes and increased synthesis of ceramides, free fatty acids and cholesterol [22]. *In vivo*, the same authors demonstrated, by the topical application of niacinamide in the context of a vehicle-controlled study in the case of winter xerosis, an improvement in epidermal barrier function, corresponding to a reduction of trans-epidermal water loss and an improvement in horny layer moisture [22]. Similar results were observed in another study [23] who also showed improvement in horny layer moisture in combination with a reduction in trans-epidermal water loss after the topical application of 2 % niacinamide. In another trial [24], 28 patients with atopic dermatitis, with symmetrical lesions of dry skin on both forearms, were enrolled, and were instructed to apply nicotinamide cream containing 2 % nicotinamide on the left forearm and white petrolatum on the right forearm, twice daily over a 4- or 8-week treatment period. Trans-epidermal water loss and stratum corneum hydration were measured by instrumental devices. At the end of this period, it was observed that nicotinamide significantly decreased TEWL, whilst white petrolatum did not show any significant effect. Both nicotinamide and white petrolatum increased stratum corneum hydration, but nicotinamide was significantly more effective than white petrolatum, suggesting that nicotinamide was a more effective moisturizer than white petrolatum on atopic dry skin.

4. Niacinamide and wound healing

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 [25]. In the case of monotherapy with saline solution, the skin flaps were 45.67% (± 31.14) vital. Niacinamide increased the vitality rate to 85.30% (± 9.24). These results are statistically highly significant. Surprisingly, following this promising study, no other investigation was performed on niacinamide in this important indication, and further trials would be welcomed.

5. Niacinamide and blistering disorders

Based on its ability to inhibit proinflammatory cytokine pathways, trials were conducted with nicotinamide on blistering disorders. There have been multiple case studies reporting the use of nicotinamide as an adjunct (to tetracycline antibiotics) in a host of bullous dermatoses, most notably bullous pemphigoid and linear IgA bullous dermatosis [26, 27] with therapeutic benefit reported with doses up to 2 g/day. The largest trial to date reported that oral nicotinamide (500 mg three times daily) in combination with tetracycline antibiotics was comparable to prednisolone (40—80 mg daily), but with fewer adverse effects [26]. Here also, more investigations are needed in order to support use of nicotinamide in these indications.

6. Niacinamide and pruritic disorders

To determine the efficacy of oral nicotinamide compared with placebo to ameliorate uremic pruritus (UP), a prospective, randomized, double-blind, 4-week study was conducted in 50 chronic kidney disease patients with refractory UP [28]. The patients were randomly allocated to nicotinamide tablet 500 mg twice/day or placebo. Severity of pruritus was evaluated before the start of the study and at the end of each week for four weeks by using a traditional Visual Analogue Scale and a modified questionnaire method (pruritus score). At the end of study, no significant benefit was found.

7. Niacinamide and pigmentation disorders

In a study [29] 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 evaluation. In both 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. In a second study by the same author [29] conducted in 120 Japanese women, using the same evaluation parameters as in the previous study, comparisons were made among an SPF15 sun protection cream with and without 2% niacinamide and the relevant vehicle. As a result, with 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. Later, a double blind, randomized clinical trial of niacinamide 4% *versus* hydroquinone 4% in the treatment of melasma was published [30]. 27 women with melasma were included and randomized in a double-blind manner to receive one treatment on the left and the other on the right side of the face, namely a 4% niacinamide cream or 4% HQ cream. Treatment was administered for the period of 8 weeks, with basal evaluation and follow-up at 4 and 8 weeks. Assessments included a skin pigment evaluation by a chromameter, melasma area and severity index (MASI), physician global assessment (PGA), conventional photography, and infrared thermography. The onset average MASI score for the HQ side was 4 (5% CI, 90.9—1.8) and 1.2 (95% IC, 0.8—1.6) after eight weeks ($p < 0.001$). The initial MASI score for the niacinamide side was 3.7 (95% CI, 2.9—4.4) and 1.4 (95% CI, 3.3—4.7) at the end of the study ($p < 0.001$). The average decrease for HQ was 70 and 62% for niacinamide. This improvement was registered using conventional photography with no perceptible differences between both sides. The PGA rated the niacinamide side improvement as excellent in three patients, good in nine, moderate in seven, and mild in eight. The HQ-treated side was rated excellent in seven, good in eight, moderate in six, and mild in six patients. As per colorimetric assessment, the lightening efect of HQ and niacinamide was apparent at 4 weeks of treatment, whereas it was more evident at 8 weeks. Colorimetric measures showed no statistical diferences between both treatments. As a conclusion, niacinamide was proposed as an efective, integral, and safe therapeutic alternative in the melasma treatment, with minimal side effects. The aim of another trial was to compare the efficacy of niacinamide 4% and desonide 0.05% emulsions vs. placebo in the treatment of axillary hyperpigmentation [31]. Twenty-four women aged 19—27 years with hyperpigmented axillae (phototype III—V) were randomly assigned to receive the study treatments in the axillary region. Improvement was assessed at baseline, then clinically and by colorimetry 9 weeks later. Both niacinamide and desonide induced significant colorimetric improvement compared with placebo; however, desonide showed a better depigmenting effect than niacinamide. A good

to excellent response was achieved in 24% of cases for niacinamide, 30% for desonide, and 6% for placebo.

8. Niacinamide and inflammation

In our chapter 2.3 it was reported that nicotinamide was capable of decreasing the expression of a number of proinflammatory cytokines. Therefore, it is not surprising that nicotinamide can display anti-inflammatory properties. First, studies were initiated regarding the hypothesis that nicotinamide could inhibit the production of tumour necrosis factor alpha (TNF-α) and the inflammatory response as well as induce apoptosis via inhibition of NF-κB [32]. The investigator's data have shown that nicotinamide gave dose dependent inhibition of lipopolysaccharide-induced TNF-α in the mouse within the dose range of 10—500 mg/kg. These data strongly support the notion that nicotinamide has potent anti-inflammatory and antitumor properties, because its primary mechanism of action is regulated by inhibition at the gene transcription level of NF-kappaB, which in turn inhibits TNF-α and induces apoptosis. Later, in an in vitro model of endotoxemia, human whole blood was stimulated for two hours with endotoxin at 1 ng/ml, achieving high levels of the proinflammatory cytokines IL-1β, IL-6, IL-8 and TNF-α [33]. When co-incubating whole blood, endotoxin and nicotinamide, all four cytokines measured were inhibited in a dose dependent manner. Inhibition was observed already at a nicotinamide concentration of 2 mmol/l and at a concentration of 40 mmol/l, the IL-1β, IL-6 and TNF a responses were reduced by more than 95% and the IL-8 levels reduced by 85%. These data demonstrate that nicotinamide is a potent modulator of several proinflammatory cytokines, and has a potent immunomodulatory effect *in vitro*, and may have great potential for treatment of human inflammatory disease.

9. Antibacterial properties of niacinamide

At least one recent trial considered the antibacterial properties of nicotinamide [34]. To determine whether niacinamide 3% formulation exerts an antimicrobial benefit on skin, *E. coli* & *S. aureus* survival on the volar forearm of human volunteers was evaluated after formulation application. Bacterial survival was measured 6 hours after the application of the niacinamide formulation *in vivo*. The decrease in account of *E. coli* was 55.1% whilst in *S. aureus* it was 33.1%. To rule out the possibility of direct antimicrobial action of niacinamide, *E. coli* kill was measured using two different *in vitro* protocols. Pure niacinamide was added to bacterial growth medium, and *E. coli* survival was assessed. Additionally, niacinamide formulations were applied on artificial skin which again contains no biological materials. Six hours after application, *E. coli* survival was measured: neither niacinamide formulations nor pure niacinamide exhibited direct antimicrobial effect against *E. coli*. Similar to *E. coli*, niacinamide formulations did not impact *S. aureus* survival *in vitro*. Taken together, data clearly indicated that niacinamide formulations tested in the human volunteer study had no direct effect on bacterial survival. Using primary skin keratinocytes, antimicrobial peptide (AMP) psoriasin levels were investigated after niacinamide treatment in vitro. According to the results, niacinamide treatment enhanced psoriasin secretion from primary skin keratinocytes, but also gene expression of AMPs like RNase 7 and calprotectin. This study shows that a niacinamide containing formulation can boost the skin's antimicrobial properties and provide protection from bacteria 6 hours after formulation application *in vivo*.

10. Use of nicotinamide in acne

At that time, to the best of our knowledge, four studies used nicotinamide as a single-agent topical product, either at 4% [35—37] or at 5% [38]. In three studies, nicotinamide was compared to 1% clindamycin gel [36, 37] or 2% clindamycin gel [38]. In all four studies, a significant improvement was noted in acne vulgaris from baseline, whilst in all three studies vs. clindamycin, it was stated that nicotinamide and clindamycin resulted in similar reductions in acne lesions.

11. Cosmetic applications of niacinamide

In a study on cell cultures [39], it was found that in ageing 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 effect on collagen synthesis, epidermal biopolymers (proteins) keratin, flaggrin 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.

12. Safety profile of nicotinamide

12.1. Side effects of nicotinamide

The side effects of oral nicotinamide develop three major axes: Parkinson's disease (PD), diabetes and liver damage. Niacin and nicotinamide are neuroprotective at low doses but may be toxic at high doses [40]. Excess nicotinamide has been hypothesized to be involved in the development of PD, but this was not definitely demonstrated [41]. Overloading rats with cumulative doses of nicotinamide or

N-methyl-nicotinamide induced acute insulin resistance [42]. This was a relatively small study, however, and effects may not be the same in humans. In one of the largest double-blind, placebo controlled human trials of nondiabetics with relatives with islet cell antibodies, there was no difference in incidence of diabetes in the treatment and placebo group when 1.2 g/m^2 of nicotinamide was given daily for 5 years [43]. However, a study of children given 1.2 g/m^2 nicotinamide per day over an average follow up period of 7.1 years found there was a statistically very significant reduction in the incidence of diabetes. Development of diabetes in those treated with nicotinamide was only 41 % that of the group not treated with nicotinamide. This shows that nicotinamide may in fact be protective against the development of diabetes [44]. Concerning liver toxicity, a study using high doses of nicotinamide in rats caused liver cell enlargement, increased glycogen deposition, and increased total hepatic lipids [45]. Doses of 10 g/day did however cause acute heptocellular changes in humans but these disappeared when treatment was ceased [46]. Conversely, nicotinamide used in conjunction with methotrexate in arthritis studies in animals protected the liver from the usual damage caused by methotrexate [47]. This finding could be of interest in dermatological treatments using methotrexate.

Side effects from the topical application of nicotinamide are minor and rare and include: mild burning, pruritis, and erythema. These side effects improve with continued use [48].

12.2. Niacinamide in paediatric populations

Elliott et al's [44] large study of slow-release nicotinamide in 20 195 children (aged 5—8 years) reported no liver enzyme changes with doses of 1.2 g/m^2/d. The duration of administration was varied between participants with maximum length of treatment 3 years and maximum follow-up of 4.7 years at time of publication. In this report, there was no other mention of adverse effects, and their overall conclusion was that nicotinamide is safe in humans at the dose delivered.

12.3. Niacinamide in pregnancy and breastfeeding

Nicotinamide crosses the placenta, and there have been no teratogenic effects observed in murine studies [49]. It has, however, not been formally assessed in pregnancy in humans.

Conclusions

Nicotinamide is commonly prescribed in dermatology, as well in oral as in topical form.

Its use is widely documented in some important dermatological indications such as photoprotection and skin cancer prophylaxis, skin barrier disorders, inflammation or pigmentary disorders, but much less in bullous diseases, pruritus or wound healing for instance. Based on the promising results found in these latter, it would be interesting developing these axes of researches, all the more because oral as well as topical administration of nicotinamide proved to be safe and without side effects.

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К. Діл

Університет Гульєльмо Марконі, Рим, Італія

Використання нікотинаміду в дерматології

Нікотинамід (синонім — ніацинамід) є водорозчинним амідним ізотипом вітаміну B_3, а ніацин (синонім — нікотинова кислота) — відповідним ізотипом кислоти. Нікотинамід використовують у дерматології вже протягом тривалого часу за різними показаннями, тому доцільно зробити оновлений огляд численних сфер його застосування.

Нікотинамід зазвичай надходить до організму з їжею; м'ясо і риба дуже багаті на ніацинамід, менше його міститься в овочах. Дефіцит цього вітаміну може спричинити пелагру, яка виявляється тріадою: деменцією, дерматитом

і діареєю. Нікотинамід виступає каталізатором численних молекулярних реакцій в усьому організмі і перетворюється на кілька коферментів, включаючи нікотинамідаденіндинуклеотид (NAD) і нікотинамідаденіндинуклеотид фосфат (NADP), які відіграють ключову роль в обміні речовин. NAD і NADP є коферментами безлічі дегідрогеназ, що беруть участь у перенесенні водню.

Їхня основна функція полягає в подачі водню до дихального ланцюга мітохондрій для окиснення і вироблення енергії. Нікотинамід збільшує вироблення АТФ, що сприяє відновленню ДНК і захисту шкіри від фотоуражень. У НаСаТ кератиноцитах людини, стимульованих сонячною енергією, опромінених ультрафіолетом і оброблених ніацинамідом, виявлено збільшену кількість і швидкість відновлення ексцизій ДНК у клітинах, оброблених нікотинамідом, порівняно з контролем.

Цей огляд має на меті описати основні дерматологічні розлади, для усунення яких використовували нікотинамід, головним чином фотозахист та профілактику раку шкіри, недоліки бар'єрної функції шкіри — особливо атопічний дерматит, порушення пігментації, запальні захворювання або вугрі. Застосування перорально або місцево у цієї категорії хворих нікотинаміду ретельно задокументовано і має терапевтичні перспективи.

Також перспективним, але менш документованим є використання нікотинаміду в деяких інших випадках – для загоєння ран, у разі бульозних порушень, що супроводжуються свербежем, призначення за косметичними показаннями. Крім того, доцільно вивчити антибактеріальні властивості цього вітаміну. Розглянуто побічні ефекти нікотинаміду, виразність яких є незначною, що дає змогу зробити висновок про те, що цей вітамін варто активніше використовувати в дерматології.

Ключові слова: нікотинамід, ніацинамід, побічні ефекти, фотозахист, рак шкіри, шкірний бар'єр, вугрі, пігментація.

К. Дил

Университет Гульельмо Маркони, Рим, Италия

Использование никотинамида в дерматологии

Никотинамид (синоним — ниацинамид) является водорастворимым амидным изотипом витамина B_3, а ниацин (синоним — никотиновая кислота) — соответствующим изотипом кислоты. Никотинамид используют в дерматологии уже в течение длительного времени по разным показаниям, поэтому целесообразно сделать обновленный обзор многочисленных сфер его использования.

Никотинамид обычно поступает в организм с едой; мясо и рыба очень богаты ниацинамидом, меньше его содержится в овощах. Дефицит этого витамина может вызвать пеллагру, проявляющуюся триадой: деменцией, дерматитом и диареей. Никотинамид выступает катализатором многочисленных молекулярных реакций во всем организме и превращается в несколько коферментов, включая никотинамидадениндинуклеотид (NAD) и никотинамидадениндинуклеотид фосфат (NADP), которые играют ключевую роль в обмене веществ. NAD и NADP являются коферментами множества дегидрогеназ, участвующих в переносе водорода.

Их основная функция заключается в подаче водорода в дыхательную цепь митохондрий для окисления и производства энергии. Никотинамид увеличивает выработку АТФ, что способствует восстановлению ДНК и защите кожи от фотоповреждений. В НаСаТ кератиноцитах человека, стимулированных солнечной энергией, облученных ультрафиолетом и обработанных ниацинамидом, обнаружено увеличенное количество и скорость восстановления эксцизий ДНК в клетках, обработанных никотинамидом, по сравнению с контролем.

Этот обзор имеет целью описать основные дерматологические расстройства, для устранения которых использовали никотинамид, главным образом фотозащиту и профилактику рака кожи, недостатки барьерной функции кожи – особенно атопический дерматит, нарушения пигментации, воспалительные заболевания или угри. Применение перорально или местно у этой категории больных никотинамида тщательно документировано и имеет терапевтические перспективы.

Также перспективным, но менее документированным является использование никотинамида в некоторых других случаях – для заживления ран, в случае буллезных расстройств, сопровождающихся зудом, назначение по косметическим показаниям. Кроме того, нуждаются в изучении антибактериальные свойства данного витамина. Рассмотрены побочные эффекты никотинамида, выраженность которых достаточно незначительна, что позволяет сделать вывод о том, что данный витамин следует активнее использовать в дерматологии.

Ключевые слова: никотинамид, ниацинамид, побочные эффекты, фотозащита, рак кожи, кожный барьер, угри, пигментация.

Дані про автора:

Dr. Christian Diehl, Department of Dermatology, Universitá Degli Studi Guglielmo Marconi
Via Plinio, 44, 00193, Rome, Italy. E-mail: chdiehl@hotmail.com