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## Prospects for inclusion of natural herbal remedies in the diet of patients with acne to improve skin condition

The article discusses the etiological factors of acne occurrence in different age groups and provides the general principles of treatment depending on the stage of the disease. The article reveals experience of «Finer» as a dietary supplement in the diet of patients of childbearing age with a diagnosis of acne. Positive effect of the herbal preparation «Finer» on the skin of patients is proved in current report. «Finer» inhibits the activity of *Propionibacterium acnes*, showing a pronounced anti-inflammatory effect — that is harmoniously integrated approach to the management of acne by dermatologist.

### Key words:

Acne, etiology, treatment, *Propionibacterium acnes*, anti-inflammatory effect.

Acne, or acne conglobata (AC), is one of the most pressing problems in dermatology. This is due to the high prevalence of human skin diseases, which is explained by the rapid pace of life in society, an increase in emotional stress, and the development of a discrepancy between the capabilities of human biological nature and living conditions. Despite numerous clinical and laboratory studies, the treatment and prevention of acne exacerbations are challenging and often a prolonged process [1], [3], [21], [40], [41]. Pathological changes in appearance, frequent relapses, and a persistent chronic course reduce patients' functionality, negatively impact their emotional state, and contribute to the development of depression, low self-esteem, social maladjustment, and even suicidal thoughts [4], [19], [41]. These factors indicate the importance of developing a pathogenetically justified therapy for acne, especially focusing on external treatment of the disease.

In patients aged 12 to 24 years, acne is referred to as acne vulgaris. Its occurrence in older age groups is classified as acne tarda. In recent years, there has been an increase in the number of women with late-onset acne. According to a study by Ch. Colleir and colleagues, the frequency of acne in adolescence is practically the same among individuals of different genders, whereas late-onset acne is most commonly observed in women. For instance, the proportion of female patients aged 25 to 40 years with acne breakouts ranges from 40 to 54%.

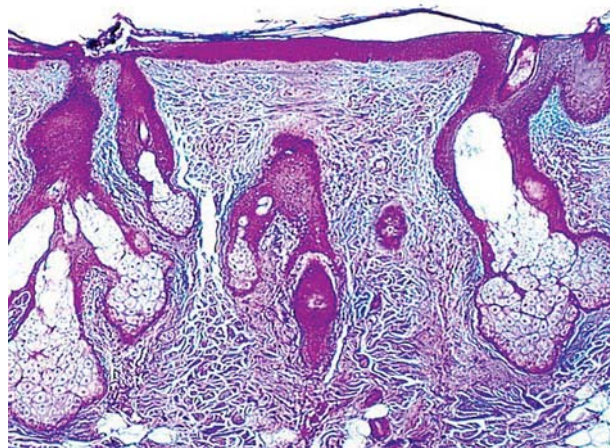
In the studies by G. Dummont-Wallon et al., and J. Rosso et al., the average age of women with acne was 31.8, 32.4, and 26.5 years, respectively. In a multicenter study conducted in the USA between 1990 and 1999, it was found that the average age of acne patients increased from 26.5 to 40.5 years [7, 13, 17, 20]. One of the main questions discussed in the literature regarding acne tarda is the age of the patient at which we can diagnose late-onset acne.

The majority of domestic and foreign experts consider the age limit for acne to be 25 years, regardless of whether it started during adolescence or as persistent recurrences. In most women with late-onset acne (up to 80% according to various data), the disease follows a continuous (persistent) course from the pubertal period. Less commonly (20-40%), late-onset acne occurs in individuals aged 25 and above. The rarest form of late-onset acne is recurrent acne, or acne with "bright intervals," which first appears during adolescence and is characterized by prolonged periods of remission and reoccurs in mature age [7, 17, 42]. Among the clinical manifestations of late-onset acne in women, the most common form is papulopustular acne (70-80%), while comedonal acne and the most severe form of acne - acne conglobata - occur in 15-20% of patients. Unlike adolescent acne, which is usually characterized by a prevalence of pathological processes and a predominance of moderate to severe forms, in the case of late-onset acne, comedonal and papulopustular forms of the

condition are diagnosed in the majority of patients. The process is mostly localized on the face, less commonly on the upper third of the trunk. Severe nodulocystic acne is rare. In patients with late-onset acne, in addition to acne eruptions, signs of skin dehydration due to previous medicamentous treatment, irrational basic care, and age-related skin changes are observed [30-32, 40].

Development and the course of dermatosis largely depend on familial (genetic) predisposition, as well as on skin type, color, and ethnic characteristics. A general pattern is observed: the more frequently and severely acne is observed in close relatives, the more severe and prolonged the course of the disease in offspring [1, 8, 12, 31, 43]. The human skin and especially its appendages - hair follicles, sebaceous and sweat glands - have steroid-sensitive receptors that perceive the hormonal influence on the development and secretory activity of these structures. During puberty, these interactions begin to manifest actively for the first time. The main skin targets for sex steroids include the epidermis, hair follicles, sebaceous glands, melanocytes, and fibroblasts. German scientist S. Schmitt referred to the skin as the largest endocrine gland in the human body. The skin actively participates in the metabolism of steroid sex hormones, particularly in the extraglandular formation of androgens from precursor steroids while also serving as the main target tissue for androgens. It plays a crucial role in the peripheral metabolism of male sex hormones, and their effects are mediated through specific androgen receptors present in various androgen-dependent skin structures. Stimulating androgen receptors increases the mitotic activity and differentiation of epidermal cells, enhances the synthesis of intercellular lipids, accelerates hair growth, and increases sebum secretion [5, 29, 44, 45].

According to modern concepts, there are four factors that play an important role in the pathogenesis of acne. One of these factors is hyperandrogenism, which is a condition characterized by an excessive amount of androgens in the blood and urine. Hyperandrogenism can be genetically determined and classified into glandular, ovarian, and mixed types. It may result from tumorous or functional changes in the ovaries and adrenal glands, such as polycystic ovary syndrome or non-classical congenital adrenal cortex dysfunction. Several studies have emphasized the significant role of steroid hormones in women with late-onset acne [2, 6, 9, 33], providing valuable insights into the relationship between hormones and acne development.



**Fig. 1. Acne on a micro level**

Prevalence of dyskeratosis and proliferation over desquamation of the sebaceous-hair follicle epithelium (SHFE). Follicular dyskeratosis and closure of the SHFE duct. Open and closed comedones depending on the level of SHFE blockage (E. Makrantonaki et al., 2011).

In the absence of an increase in the levels of main androgens in the blood plasma and with normal sex hormone-binding globulin binding capacity (testosterone bound to it is least active), a receptor form of hyperandrogenism is assumed. This form is characterized by increased sensitivity of receptors to normal or reduced amounts of androgens. The main cause of receptor-relative hyperandrogenism is the increased activity of the enzyme 5-alpha-reductase I, which converts testosterone into a more potent metabolite - dihydrotestosterone. This metabolite directly stimulates the proliferation and maturation of sebocytes [2, 33, 50, 51]. Under the influence of hormonal factors, the volume of sebum (skin oil) increases, with production rising by 1.3 times in mild acne cases, 1.7 times in moderate cases, and 1.9 times in severe cases. In the enlarged volume of sebum, the concentration of essential  $\alpha$ -linolenic acid, the main regulator of keratinocyte differentiation in the sebaceous-hair follicle duct (SHFD), decreases, and the expression of transglutaminase enzyme increases. This leads to a predominance of dyskeratosis and proliferation processes over epithelial squamation in the SHFD, resulting in follicular hyperkeratosis and closure of the SHFD, clinically manifested as open and closed comedones depending on the level of SHFD obstruction (Fig. 1). The occlusion of the SHFD and lipid-rich sebaceous glands create favorable conditions for the proliferation of facultative anaerobes - *Propionibacterium acnes* and *P. granulosum* (Fig. 2), as well as other representatives of saprophytic and opportunistic microflora, including *Staphylococcus epidermidis*, *aureus*, and *Pityrosporum ovale* [3, 38].



Fig 2. Microphotography of *Propionibacterium acnes* (D. Saper та співавт., 2015)

These microorganisms produce the enzyme lipase, which hydrolyzes diacyl- and triacylglycerides into glycerol and free fatty acids.

These substances, along with the antigens of microorganisms, attract neutrophils and phagocytes from the peripheral blood, which produce IL-1 $\alpha$ , IL-1 $\beta$ , IL-8, and TNF- $\alpha$ . These pro-inflammatory cytokines activate the enzyme cyclooxygenase, which promotes the formation of the main inflammatory mediator, leukotriene B<sub>4</sub>, from arachidonic acid. In turn, it stimulates the release of hydrolytic enzymes and nitric oxide from neutrophils, T-lymphocytes, monocytes, and eosinophils, leading to the breakdown of the sebaceous gland wall, the release of its contents into the dermis, and the development of an inflammatory reaction in the form of papulopustular and nodulocystic elements [1, 3, 8, 17, 18]. Furthermore, *P. acnes* can induce the expression of B-defensins - cationic peptides of the immune system that are active against bacteria, fungi, and many viruses. Apart from their direct anti-infective action, they perform many other important functions: acting as mediators of inflammation, influencing chemotaxis, and possessing immunomodulatory and cytotoxic activities. Recent studies have shown that *P. acnes* can activate the insulin-like growth factor-1 (IGF-1) and insulin-like growth factor receptor type I (IGF-R 1) system in the epidermis, similar to insulin, and promote keratinocyte proliferation. *P. acnes* exhibits high pro-inflammatory activity, significantly higher than *Staphylococcus aureus* and *Streptococcus* [22, 23, 46].

The most common adolescent acne vulgaris affects about 80% of individuals aged 15 to 24 years. The condition usually begins during puberty and is characterized by the appearance of comedones on the face (less commonly, on the chest and back) - small (up to 5 mm in diameter) bright red papules, sometimes with pustules on the surface.

After the completion of eruptions, pigmentation remains, and less commonly, superficial scars remain. Sebum secretion is increased, and the skin exhibits a characteristic oily sheen. The hair also becomes oily, and there may be slight seborrheic thinning of the skin. In the early stage, comedonal acne is non-inflammatory. However, later, a small number of inflammatory elements appear on the face, and the condition progresses to more severe forms. The number of comedones increases, and hair follicle openings enlarge and become more prominent. Comedones appear not only on the face, back, and chest but also in the ear areas and on the scalp. The inflammatory reaction around the comedones becomes more pronounced, leading to the formation of large inflammatory nodules deep within the skin. This condition is known as nodular acne. After several weeks, the infiltrate softens, and the nodules open, forming cavities from which viscid purulent exudate is discharged (monoseptic acne). After healing, deep scars remain, distorting the skin. The most common form with a pronounced inflammatory component is conglobate acne. The clinical presentation is characterized by the appearance of large nodules on the back, chest, and face, located deep in the dermis, sometimes involving the upper layers of the subcutaneous tissue. The nodules can reach a diameter of 1.5 to 2.0 cm. They are extremely painful, with acute perifocal inflammation. When they merge, they form conglomerates and may lead to abscesses. After these abscesses rupture, they leave long-lasting ulcers, which eventually turn into thick scars with partitions and sinus tracts. Occasionally, deeply located inflamed nodules merge to form epithelial sinus tracts that "undermine" the skin of the face and torso, resulting in sinus acne. One of the rare variants of inflammatory acne, in which there is an acute transformation of typical inflammatory acne into an extremely pronounced destructive inflammation, is acne fulminans. It mainly occurs in young men. Numerous painful inflammatory nodules covered with ulcers and areas of necrosis, along with scattered pustules, appear on an erythematous background. The process is accompanied by fever, leukocytosis, joint pain, and transient glomerulonephritis. After healing, thick scars remain. Sometimes, there is facial pyoderma - a severe form of acne with a difficult course, which most often affects young women who have experienced emotional stress or previously received androgens for endocrine disorders. Dermatitis has a sudden onset and progresses rapidly. It is localized in the central part of the face, in the areas of the forehead, temples, and chin.

An evident facial swelling with a cyanotic skin tint occurs, and after 1-2 days, painful furuncle-like nodular eruptions appear, resembling conglomerate acne, large (over 5 mm) pustules. There are no comedones, and the inflammation has sharply defined borders. The most convenient and widespread classification of acne in dermatological practice is proposed by the American Academy of Dermatology. According to this classification, the severity of acne is distinguished as follows: 1st degree - presence of comedones (closed and open) and up to 10 papules; 2nd degree - comedones, papules, and up to 10 pustules; 3rd degree - comedones, papulopustular eruptions, and up to 3 nodules; 4th degree - pronounced inflammatory reaction in the deep layers of the dermis with the formation of numerous painful nodules and cysts. However, this classification does not take into account the prevalence of the process. Therefore, in addition to the classic division based on the severity of acne, it is advisable to classify patients into subgroups depending on the localization and extent of the process (A, B, C): A - for localized eruptions in one anatomical area; B - for eruptions localized in two anatomical areas (e.g., face and back); C - in case of involvement of three or more anatomical areas. This classification most comprehensively reflects the extent of skin involvement in the pathological process.

It should be noted that acne belongs to a particular group of skin disorders called psychosomatic dermatoses, where psychological and emotional disturbances play a significant role, particularly in girls and women, related to the issue of "appearance defect." The localization of the skin condition in exposed areas causes deep psychological suffering for patients, reducing self-esteem, negatively affecting their quality of life, social status, professional activities, and personal life. In the overall prevalence of anxiety and depressive disorders, acne patients, according to some data, rank second, surpassing patients with other somatic and skin diseases, including oncological patients. Patients with acne demonstrate a higher level of anxiety and depression compared to other dermatological patients. Around 30% of adolescents and 5% of adults require active psycho-psychiatric assistance [32]. The British Association of Dermatologists has established that a specific type of dermatosis increases the risk of suicidal attempts. Elements of self-harming behavior, which in some cases have led to completed suicide, were most frequently observed in acne, psoriasis, eczema, and urticaria.

Patients with acne have a high risk of suicide attempts and may also pose a threat to their doctors [18, 27]. According to J.K. Tan et al., factors such as female gender, mature age, and a disease duration of over 5 years negatively impact the quality of life, significantly reducing it. Female acne patients are the most psychologically vulnerable. 64% of female acne patients are unemployed and have an unsatisfactory personal life. Additionally, it has been found that in 40% of patients, the skin condition associated with cosmetic defects leads to a decrease in their social status and negatively affects their professional activity [6, 8, 15, 35]. Research by Russian dermatologists indicates that various degrees of psycho-emotional disorders are observed in 41.3% of patients with acne vulgaris, predominantly in females. It has been revealed that depressive symptoms caused by acne are more frequent and pronounced in women compared to men [4, 14].

Literature data regarding the relationship between stress factors and the occurrence of acne are conflicting. For instance, V. Goulden et al. found that only 12 out of 71% of women considered stress to be a triggering factor for their acne. However, F. Poli et al., G. Dumont-Wallon et al. noted that stress is one of the significant factors provoking acne breakouts in women, as a triggering factor in 34-50% of observations. The connection between stress and acne exacerbation is now explained by the production of neurotransmitter (substance P), which influences the differentiation and proliferation of sebocytes and stimulates the production of sebum. The skin of individuals prone to acne is characterized by a large number of nerve endings, nerve fibers capable of secreting substance P, as well as basophils [7, 13, 17, 32]. Acne exacerbations are most frequently observed in winter and spring. The aggravation of the condition is caused by constant mechanical trauma to the skin (picking at pimples, frequent washing with soap), the negative effects of the external environment (skin contamination with oils, gasoline, industrial dust), the use of certain medications (steroid hormones, halogen-containing and anti-tuberculosis agents, antidepressants, antiepileptic drugs), and the inappropriate choice of cosmetic products for skin care. Sun exposure has a positive effect on the course of acne in most patients and contributes to the partial resolution of acne lesions, but in 8-10% of patients, intense sun exposure worsens the condition [13, 31, 32, 47]. Recently, there have been publications on the influence of dietary products on the appearance of acne lesions.

Such a hypothesis was already put forward about half a century ago, but it was later rejected. Currently, research is underway abroad on the impact of the diet on the course of acne. It is believed that products with a significant amount of sugar and other carbohydrates, by increasing the level of glycemic load, affect the concentration of insulin and IGF-1, which in turn increases the levels of main androgens in the blood plasma. This leads to hypersecretion of sebum and the appearance of acne. Intensive consumption of iodine- or bromine-containing food products, including large amounts of iodized salt and seafood, can lead to an increase in inflammatory eruptions in acne patients. Additionally, incorporating products containing antioxidants, omega-3 fatty acids, dietary fiber, and vitamins A, E, C into the diet positively affects the course of acne, contributing to a reduction in the number of eruptions [19, 25, 26, 37].

The medical tactics for acne in women depend on the stage, extent of the process, presence of pathological changes in the hormonal-endocrine status, as well as the psychological and emotional state of the woman. When choosing the treatment strategy, the patient's age, accompanying pathology, the possibility of adhering to the treatment regimen, and previous therapies should also be taken into account.

General principles of acne treatment:

- limiting the consumption of carbohydrate-rich products, coffee, alcoholic beverages, spicy condiments, and spices if they increase skin oiliness and contribute to disease exacerbation;
- treatment of concomitant diseases, normalization of the endocrine system function, and improvement of the psychoemotional state;
- rehabilitation of sources of chronic infection;
- usage of facial cleansers regardless of the severity of the condition; although the appearance of acne breakouts is not related to poor facial skincare, an acidic or neutral environment helps prevent inflammatory and purulent complications;
- the general and local effects of using medicinal and cosmetic products, particularly in the context of acne treatment with medical devices.

Preparations and techniques in dermatology and cosmetology are selected depending on the prevalence and severity of the condition. Approaches to treating the disease involve the prescription of various systemic and topical medications that target different links in the pathogenesis of acne [16, 30, 48, 49]. Systemic therapy is prescribed for 3rd and 4th-degree acne, for persistent cases of 2nd-degree acne, as well as for extensive breakouts and complicated hormonal-endocrine conditions for any degree of acne (hormonal therapy) [34, 36, 39].

At the 1st-degree acne, only local therapy is used, with topical retinoids (table), being the first-choice drugs. At the 2nd-degree acne, systemic therapy is added to the topical treatment, usually involving antibiotics. The use of high-intensity blue light and zinc preparations is possible, but the level of evidence for these types of therapy is not high [40, 42]. When prescribing topical antibiotics, it should be taken into account that, according to recent data, clindamycin is the most effective drug for acne eruptions.

Women with severe forms of acne are recommended to be prescribed oral contraceptives (OC) with antiandrogenic effects in combination with topical anti-acne agents. In severe cases, systemic antibiotics may be used. To achieve a significant therapeutic effect, combined OCs should be prescribed for a prolonged period of 6 months to 1 year. If they are contraindicated, systemic retinoids are used. The frequency of positive results with systemic retinoid therapy for severe forms of acne is dose-dependent. Prolonged use of the medication leads to a significant suppression of sebaceous gland activity, histologically confirmed by a reduction in their size. To achieve the highest treatment effectiveness, prolong remission, and reduce the frequency of recurrences, it is advisable to use a course dose of 120-150 mg/kg. Systemic retinoids should be applied for 3-6 months. In case of contraindications to both OCs and systemic retinoids, it is most appropriate to use systemic antibiotics in combination with azelaic acid preparations (gel, cream), and other topical agents for at least 1 month.

In recent years, various natural plant-based preparations, including dietary supplements with active ingredients affecting not only the pathogenesis but also the etiological factors of acne, have been widely used to improve the condition of the skin in acne patients [12, 16, 20, 21, 37, 45]. One of such remedies is "Finer," a comprehensive plant-based preparation containing azadirachtin, a substance with antiparasitic, antibacterial, and anti-inflammatory properties. Specifically, "Finer" targets Demodex mites since azadirachtin inhibits their maturation and reproduction [9-11]. Additionally, "Finer" exhibits antibacterial effects as azadirachtin inhibits the activity of *Propionibacterium acnes* [22, 27]. Furthermore, "Finer" exerts a pronounced anti-inflammatory effect by suppressing inflammatory reactions, thereby preventing the formation of infiltrates, ulcers, and scars [25].

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Table

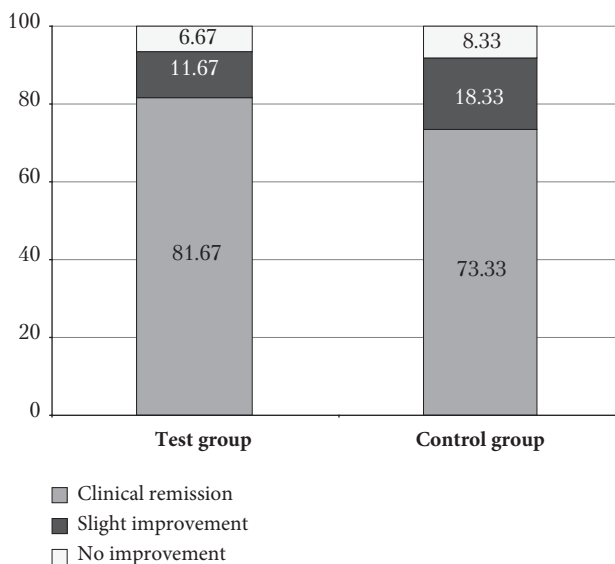


Fig. 3. Effectiveness of treatment for moderate acne among patients in the test and control groups, %.

On the basis of the Department of Dermatology and Venereology at the O.O. Bogomolets National Medical University, experience has been accumulated regarding the inclusion of the product "Finer" in the diet of female patients undergoing comprehensive treatment for moderate acne. Two groups of women aged 18 to 32 were under observation. The patients in the research group (n=60) received the following treatment: azelaic acid twice a day, benzoyl peroxide with clindamycin once a day for 6 weeks (topically), and oral azithromycin 250 mg once a day, starting from the 5th week (course - 14 days). The "Finer" supplement, a herbal remedy to improve the skin condition, was introduced into the diet of these patients. The control group (n=60) received azelaic acid and benzoyl peroxide with clindamycin once a day for 6 weeks (topically) and oral azithromycin 250 mg once a day, starting from the 5th week (course - 14 days).

Analysis of the treatment results of female patients in the study group revealed clinical remission in 81.67% of women (Fig. 3), which is 8.34% higher than in the control group ( $p \leq 0.05$ ). The mean values of the Dermatological Acne Index (DIA) at 4 and 8 weeks were 1.9 points (Fig. 4), which is 1.4 points lower than in the control group ( $p \leq 0.05$ ). In both groups, the Dermatological Index of Quality of Life (DIQL) scores indicated a stable trend towards improvement (Fig. 5), but in the study group, there was a better positive change dynamics after 8 weeks.

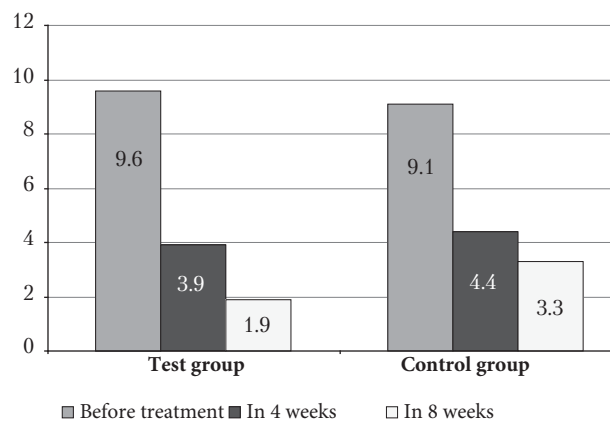


Fig. 4. Dynamics of decrease in dermatological acne index (DIA) in patients of the test group, compared to the control group data ( $p \leq 0.05$ ).

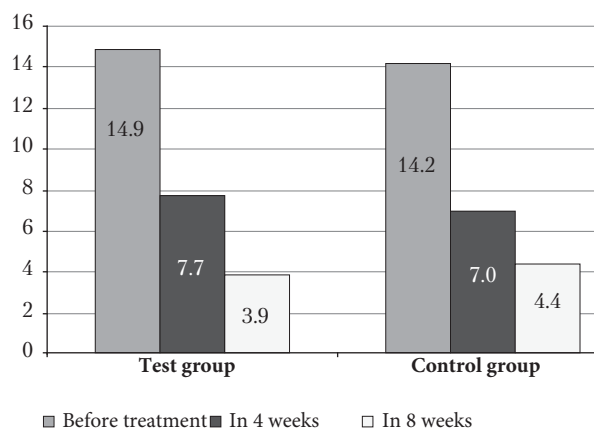


Fig. 5. Dynamics of changes in the dermatological index of quality of life (DIQL) in patients of the test group compared to the data of the control group ( $p \leq 0.05$ ).

In this way, adding the food supplement "Finer" to the daily diet of patients undergoing complex acne treatment provides conditions for the regression of inflammatory elements and normalization of the skin's functional state. It has been confirmed that "Finer" is a safe natural remedy for skin cleansing, promotes acne involution, and improves overall body condition and quality of life.

## References

1. Адаскевич В.Л. Акне вульгарные и розовые. — М.: Медицинская книга, Н. Новгород: Изд-во НГМА, 2003. — 160 с.
2. Гусаков Н.И. Акне. — Акне, 2003. — С. 76.
3. Доброхотова Ю.Э., Дзобова Э.М., Рагимова З.Ю. и др. Синдром гиперандрогении в практике акушера-гинеколога, дерматолога, эндокринолога: руководство для врачей. — М.: ГЭОТАР-Медиа, 2009. — 112 с.
4. Кутасевич Я.Ф., Маштакова И.А., Багмет А.Н., Шаповалова О.В. Микробиоценоз кожи у больных угревой болезнью и пути его коррекции // Укр. журн. дерматол., венерол., косметол. — 2003. — № 1. — С. 43–47.
5. Майорова А.В., Шаповалов В.С., Ахтямов С.Н. Угревая болезнь в практике врача-дерматокосметолога. — М.: Фирма Клавель, 2005. — 192 с.
6. Монахов С.А., Иванов О.Л. Акне: методическое пособие для врачей. — М., 2010.
7. Самцов А.В. Акне и акнеформные дерматозы. — М.: ООО «ЮТКОМ», 2009. — 288 с.
8. Суворова К.Н., Котова Н.В. Тяжелые формы акне // Междунар. мед. журн. — 2000. — С. 732–726.
9. Abdel G., Quraishy S., Rasheid K.A., Mehlhorn H. Efficacy of a single treatment of head lice with a neem seed extract: an in vivo and in vitro study on nits and motile stages // Parasitol. Res. — 2012. — Vol. 110 (1). — P. 277–280.
10. Abdel-Shafy S., Zayed A.A. In vitro acaricidal effect of plant extract of neem seed oil (*Azadirachta indica*) on egg, immature, and adult stages of *Hyalomma anaticum* excavatum (Ixodoidea: Ixodidae) // Vet. Parasitol. — 2002. — Vol. 106 (1). — P. 89–96.
11. Al-Rajhy D.H., Alahmed A.M., Hussein H.I., Kheir S.M. Acaricidal effects of cardiac glycosides, azadirachtin and neem oil against the camel tick, *Hyalomma dromedarii* (Acari: Ixodidae) // Pest. Manag. Sci. — 2003. — Vol. 59 (11). — P. 1250–1254.
12. Carson C.F., Hammer K.A., Riley T.V. Melaleuca alternifolia (Tea Tree) oil: a review of antimicrobial other medicinal properties // Clin. Micro Rev. — 2006. — Vol. 19 (1). — P. 50–62.
13. Choi J.M., Lew V.K., Kimball A.B. A single-blinded, randomized, controlled clinical trial evaluating the effect of face washing on acne vulgaris // Pediatr. Dermatol. — 2006. — Vol. 23 (5). — P. 421–427.
14. Cotterill J.A., Cunliffe W. Suicide in dermatological patients // British Association of Dermatologists. — 1997. — Vol. 137. — P. 246–250.
15. Danby F.W. Nutrition and acne // Clin. Dermatol. — 2010. — Vol. 28 (6). — P. 598–604.
16. Darne S., Hiscutt E.L., Seukeran D.C. Evaluation of the clinical efficacy of the 1,450 nm laser in acne vulgaris: A randomized split-face, investigator-blinded clinical trial // Br. J. Dermatol. — 2011. — Vol. 165. — P. 1256–1262.
17. Del Rosso J.Q., Bikowski J., Baum E. Prevalence of truncal acne vulgaris: a population study based on private practice experience // J. Am. Acad. Dermatol. — 2007. — Vol. 56. — AB 3.
18. Del Rosso J.Q. 6 % benzoyl peroxide foaming cloth cleanser used in the treatment of acne vulgaris // J. Clin. Aesthet. Dermatol. — 2009. — Vol. 2 (7). — P. 26–29.
19. Dumont-Wallon G., Dreno B. Specificity of acne in women older than 25 years // Presse Med. — 2008. — Vol. 37. — P. 585–591.
20. Enshaleh S., Jooya A., Siadat A.M., Irajy F. The efficacy of 5 % topical tea tree oil gel to moderate acne vulgaris: a randomized, double-blind placebo-controlled study // Ind. J. Derm. Venereol. Leprol. — 2007. — Vol. 73 (1). — P. 22–25.
21. Ghosh V.K., Nagore D.H., Kadbhane K.P., Patil M.J. Different approaches of alternative medicines in acne vulgaris treatment // Oriental Pharmacy and Experimental Medicine. — 2011. — Vol. 11 (1). — P. 1–9.
22. Hamid Nasri, Mahmoud Bahmani, Najmeh Shahinfard et al. Medicinal Plants for the Treatment of Acne Vulgaris: A Review of Recent Evidences // Jundishapur. J. Microbiol. — 2015. — Vol. 8 (11). — P. e25580.
23. Hamilton F.L., Car J., Lyons C. et al. Laser and other light therapies for the treatment of acne vulgaris: Systematic review // Br. J. Dermatol. — 2009. — Vol. 160. — P. 1273–1285.
24. Isard O., Knol A.C., Aries M.F. et al. Propionibacterium acnes activates the IGF-1/IGF-1R system in the epidermis and induces keratinocytes proliferation // J. Invest. Dermatol. — 2011. — Vol. 131. — P. 59–66.
25. Jain A., Basal E. Inhibition of Propionibacterium acnes-induced mediators of inflammation by Indian herbs // Phytomedicine. — 2003. — Vol. 10 (1). — P. 34–38.
26. Jappe U., Igham E., Henwood J., Holland K.T. Propionibacterium acnes and inflammation in acne: P acnes has T-cell mitogenic activity // Br. J. Dermatol. — 2002. — Vol. 146. — P. 202–209.
27. Khaled M.M. Koriem. Review on pharmacological and toxicological effects of oleum azadirachti oil // Asian Pac. J. Trop. Biomed. — 2013. — Vol. 3 (10). — P. 834–840.
28. Korting H.C., Lehmann P. Acne vulgaris // Hautarzt. — 2010. — Bd. 61 (2). — S. 97–98.
29. Logan A. Omega-3 fatty acids and acne // Arch. Dermatol. — 2003. — Vol. 139. — P. 941–942.
30. Melnik B.C., Schmitz G. Role of insulin, insulin-like growth factor-1, hyperglycaemic food and milk consumption in the pathogenesis of acne vulgaris // Exp. Dermatol. — 2009. — Vol. 18 (10). — P. 833–841.
31. Nagy I., Pivarsci A., Koreck A. et al. Distinct strains of Propionibacterium acnes induce selective human beta-defensin-2 and interleukin-8 expression in human keratinocytes through toll-like receptors // J. Invest. Dermatol. — 2005. — Vol. 124. — P. 931–938.
32. Nast A., Dréno B., Bettoli V. et al. European Evidence-based (S3) Guidelines for the Treatment of Acne // JEADV. — 2012. — Vol. 26 (suppl. 1). — P. 1–29.
33. Pang Y. et al. Combination of short CAGE and GGN repeats in the androgen receptor gene is associated with acne risk in North East China // J. Eur. Acad. Dermatol. Venereol. — 2008. — Vol. 22 (12). — P. 1445–1451.
34. Poli F., Dreno B., Verschoore M. An epidemiological study of acne in female adults: results of a survey conducted in France // J. Eur. Acad. Dermatol. Venereol. — 2001. — Vol. 15. — P. 541–545.
35. Preneau S., Dreno B. Female acne — a different subtype of teenager acne? // J. Eur. Acad. Dermatol. Venereol. — 2012. — Vol. 26 (3). — P. 277–282.
36. Rivera R., Guerra A. Management of acne in women over 25 years of age // Actas. Dermosifiliogr. — 2009. — Vol. 100. — P. 33–37.
37. Sakamoto F.H., Torezan L., Anderson R.R. Photodynamic therapy for acne vulgaris: a critical review from basics to clinical practice. Part II. Understanding parameters for acne treatment with photodynamic therapy // J. Am. Acad. Dermatol. — 2010. — Vol. 63 (2). — P. 195–211.
38. Savage L.J., Layton A.M. Treating acne vulgaris: systemic, local and combination therapy // Expert Rev. Clin. Pharmacol. — 2010. — Vol. 18. — P. 563.
39. Seirafi H., Farnaghi F., Vashghani-Farahani A. et al. Assessment of androgens in women with adult-onset acne // Int. J. Dermatol. — 2007. — Vol. 46. — P. 1188–1191.
40. Strauss J.S., Krowchuk D.P., Leyden J.J. et al. Guidelines of care for acne vulgaris management // J. Am. Acad. Dermatol. — 2007. — Vol. 56 (4). — P. 651–663.
41. Tan J.K. et al. Divergence of demographic factors associated with clinical severity compared with quality of life impact in acne // J. Cutan. Med. Surg. — 2008. — Vol. 12 (5). — P. 235–242.
42. Taylor M., Porter R., Gonzalez M. Intense pulsed light may improve inflammatory acne through TNF- $\alpha$  down-regulation // J. Cosmet. Laser Ther. — 2014. — Vol. 16. — P. 96–103.
43. Thiboutot D.M., Weiss J., Bucko A. et al. Adapalene-benzoyl peroxide, a fixed-dose combination for the treatment of acne vulgaris: results of a multicenter, randomized, double-blind controlled study // J. Am. Acad. Dermatol. — 2007. — Vol. 57 (5). — P. 791–799.

44. Thiboutot D. Versatility of azelaic acid 15 % gel in treatment of inflammatory acne vulgaris // J. Drugs Dermatol. — 2008. — N 7 (1). — P. 13–16.
45. Thring T.S., Hili P., Naughton D.P. Antioxidant and potential anti-inflammatory activity of extracts and formulations of white tea, rose, and witch hazel on primary human dermal fibroblast cells // J. Inflammation. — 2011. — Vol. 8 (1). — P. 27.
46. Truter I. Evidence-based pharmacy practice: acne vulgaris // SA Pharma J. — 2009. — Vol. 76. — P. 12–19.
47. Veith W.B., Silverberg N.B. The association of acne vulgaris with diet // Cutis. — 2011. — Vol. 88 (2). — P. 84–91.
48. Vexiau P. et al. Acne in adult women: data from a national study on the relationship between type of acne and markers of clinical hyperandrogenism // Ann. Dermatol. Venerol. — 2002. — Vol. 129 (2). — P. 174–178.
49. Webster G. Combination azelaic acid therapy for acne vulgaris // J. Am. Acad. Dermatol. — 2000. — Vol. 43 (2 Pt 3). — P. 47–50.
50. Webster G.F. Acne vulgaris. Clinical review // B.M.J. — 2002. — Vol. 325. — P. 475–479.
51. Williams C., Layton A.M. Persistent acne in women: implications for the patient and for therapy // Am. J. Clin. Dermatol. — 2006. — Vol. 7. — P. 281–290.

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## Перспективы введения в рацион пациенток с акне натуральных растительных средств для улучшения состояния кожи

В статье рассмотрены этиологические факторы возникновения акне у больных разных возрастных групп, а также предоставлено общие принципы лечения в зависимости от стадии заболевания. Приведены опыт применения препарата «Файнер» в качестве диетической добавки в рацион женщин фертильного возраста с установленным диагнозом акне. Доказано положительное влияние растительного средства «Файнер» на состояние кожи. «Файнер» ингибирует активность *Propionibacterium acnes*, оказывает отчетливый противовоспалительный эффект, то есть гармонично дополняет комплексный подход дерматолога к менеджменту акне.

**Ключевые слова:** акне, этиология, лечение, *Propionibacterium acnes*, противовоспалительный эффект.

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## Prospects for inclusion of natural herbal remedies in the diet of patients with acne to improve skin condition

The article discusses the etiological factors of acne occurrence in different age groups and provides the general principles of treatment depending on the stage of the disease. The article reveals experience of «Finer» as a dietary supplement in the diet of patients of childbearing age with a diagnosis of acne. Positive effect of the herbal preparation «Finer» on the skin of patients is proved in current report. «Finer» inhibits the activity of *Propionibacterium acnes*, showing a pronounced anti-inflammatory effect — that is harmoniously integrated approach to the management of acne by dermatologist.

**Key words:** acne, etiology, treatment, *Propionibacterium acnes*, anti-inflammatory effect.

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