Pawlicki Mateusz, Pawlicki Bartłomiej, Łopuszyńska Anna, Misztal Zofia, Lorenc Karol, Kozioł Magdalena. The role of diet in development of acne vulgaris. Journal of Education, Health and Sport. 2019;9(8):857-862. eISSN 2391-8306. DOI http://dx.doi.org/10.5281/zenodo.3370436
http://ojs.ukw.edu.pl/index.php/johs/article/view/7256

The journal has had 7 points in Ministry of Science and Higher Education parametric evaluation. Part B item 1223 (26/01/2017).
1223 Journal of Education, Health and Sport eISSN 2391-8306 7

The authors declare that there is no conflict of interests regarding the publication of this paper.

Received: 05.07.2019. Revised: 25.07.2019. Accepted: 18.08.2019.

The role of diet in development of acne vulgaris

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Abstract:

Acne is one of the most widely spread dermatoses in the world mainly among adolescents and young adults. Because of its problematic symptoms it is very willingly treated with diverse available drugs. Despite its popularity, there is still lack of precise information about this disease. Even though risk factors like infections or hormonal changes are said to be well known, medicine is still not certain about some of them. Diet is one of the

examples. It is supposed to play a huge role in acne genesis. Considering the ease of diet modifications it is crucial to understand its participation.

Key words: acne; acne vulgaris; diet

Introduction:

Acne, also known as acne vulgaris, is a common chronic skin disease affecting mainly teenagers during their puberty. It manifests itself with comedones, papules, pustules and even nodules and cysts in severe cases. Although the disease is not very dangerous, changes in patients' appearance may lead to decrease in their acceptance and thus higher risk of anxiety or depression. Worldwide, acne affects about 85% of teenagers and young adults (age 12-25) mainly in well developed regions of the world like Western Europe, high-income North America or Asia. This makes it the most prevalent dermatosis in this age group in the world [1]. Typically acne tends to disappear at the end of adolescence but it may persist in 3% of men and 12% of women [2].

Pathophysiology of the disease is strictly connected with sebaceous glands and processes taking place around them. Elevated level of sebum production and its changed composition, hyperkeratinization together with colonization by *Cutibacterium acnes* are the main causes of inflammation leading to typical skin problems [3]. All of these components are linked influencing each other. At the very beginning, pores of hair follicles plug as a result of excessive amount of dead skin cells. Simultaneous fluctuations of androgens levels induce the glands to produce more sebum which help gathering skin cells together. This is how the microcomedones are created. Further transformations require the presence of bacteria. Since *Cutibacterium acnes* are anaerobic, environment established by previous actions is suitable for its growth.

Risk factors:

Several risk factors of acne are still being identified and studied. One of the best known are hormones. Increased level of androgens contribute with higher prevalence of the disease. This correlation has been observed in clinical practice in patients with polycystic ovarian syndrome, congenital adrenal hyperplasia or hormonal tumors. This group has been characterized by higher rates of acne comparing to healthy individuals [4]. This partially explains why mainly teenagers are affected and the difference of frequency between both sexes [1,2].

Another common cause believed to worsen the symptoms is stress that has been confirmed in a few studies. One of them showed an improvement of skin condition in patients using relaxation techniques. Abandonment resulted in recurrence of comedones [5]. Another research focused on a group of 22 students struggling with exams at their university. Stress associated with such event increased severity of acne. The limitations of this particular analysis were small research sample and lack of adjustment for other factors [6]. However data gathered in Iran by Ghodsi et al. on a sample of over 1000 students from Tehran let us agree with previous discoveries [7].

Also many medicaments list acne vulgaris as one of their side effects. Despite obvious examples such as hormone derivatives like dehydroepiandrosterone which is similar to testosterone already mentioned as one of the factors [8], there are lots of other drugs causing acneiform drug eruptions. Some of them, like glucocorticoids, phenytoin, isoniazid or bromides are widely used to treat severe diseases or as an antibiotics. It is often impossible to replace them with other medicines [9].

There is proven evidence for correlation between acne and genetics as well. Chinese research showed 78% heritability in first-degree relatives. What is more, the symptoms tend

to be worse and appear earlier in people who are genetically loaded [6]. Zaenglein state that the influence of genetics is even higher with 81% of population variance dependent on genetic factors [10].

The last and still uncertain risk factor of acne vulgaris is diet. The impact of dietary habits on its development has been discussed for years excluding more and more products, especially these containing huge dose of carbohydrates. The aim of this study is an evaluation of available knowledge of correlation between diet and acne prevalence. The disease is treated by various techniques but, because of their poor effectiveness, pharmacological therapy is most commonly used. It is often done by the use of antibiotics or other aggravating medicines such as retinoids. Therefore it is important to better understand the genesis instead of managing with developed disorder.

Description of knowledge:

Diet is always mentioned as one of the most contributing factors of acne pathogenesis. Studies mostly focus on carbohydrates and thus on glycaemic index (GI) of the food present in patients' diet. High sugar content stimulates pancreas to release insulin which directly or indirectly through insulin-like growth factor 1 (IGF-1) activates a cascade of following reactions resulting in improper work of androgen receptor. That leads to hyperkeratisation and then processes described above. For the reason that products with high GI are mostly spread in developed areas where people are generally wealthier, acne is less typical for rural regions of the world. Cordain et al. carried out an observation of two populations of Papua New Guinea and Paraguay. Both of these countries are marked as developing ones. Despite enlisting around 1300 individuals, none of them had acne diagnosed during over 840 days of the study. Analysis of their diet has shown significant differences compared to European and North American diet. Kitavan people (Papua New Guinea) ate mostly tubers, fish, coconuts and fish, while group from Paraguay preferred mainly sweet manioc, peanuts, rice and meat of wild animals. Western food's share was about 8% reducing pasta, flour or bread intake to minimum [11]. What is interesting, these findings may change after moving from rural to urban regions. A 30-year observation of Inuit population in Canada led by Schaefer had not reported any case of acne until western habits such as soda drinking or beef eating were presented to those people. The same conclusions were created by Thiboutot and Strauss after examination of young women from Ireland. They claimed not to report any problems with acne until migration to United States [12]. Another study consisted of short, 12-week single-blind randomized controlled trial of 43 men. Divided into two seperate groups, half of them had a diet with low GI, while the others ate huge amounts of carbohydrates. At the end, the first group had greater drop of total lesions count (-23.5 vs -12,0) and reduction of weight (-2,9 vs 0,5) than the control group [13]. Similar findings were reported by Kwon et al. in his research on 32 people (24 males and 8 females). The group with low glycaemic load diet had their non-inflammatory lesion counts decreased by a bigger score than the control group. Also overall size of sebaceous glands dropped from 0,32mm^2 at baseline to 24mm^2 after 10 weeks of the study [14]. Fabbrocini et al. came up with an idea of uniting acne and diabetes treatment by adding metformin intake to diet therapy. 10 of 20 young males with altered metabolic profile took metformin twice a day daily alongside with low hypocaloric diet (1500-2000 calories). The second group only continued its symptomatic acne therapy as well as the first one. After 6 months of treatment Global Acne Grading System scores were obtained. It turned out that group with metformin scored significantly better than the other group (25,1 to 14,1 vs 24,9 to 19,4 respectively). This indicates possible connection in pathophysiology between these two disorders and casts light at role of insulin resistance in acne genesis [15].

Another frequently appearing topic are dairy products. Based on milk, they are said to induce acne mechanism through either amino acids activating cascade of reactions leading to altered lipogenesis and protein synthesis in sebaceous glands or hormones left in milk even after its processing [1]. Several studies checked if there is connection between milk consumption of different fat content and occurrence as well as severity of the disease. One of the biggest studies performed on this particular field were three studies led by Adebamowo et al. In the first one 47 355 women diagnosed with severe acne were asked to fill in a questionnaire on their high-school diet. The multivariate prevalence ratios (PR) were 1,22 for whole milk, 1,16 for low-fat milk and 1,44 for skim milk. Food like cottage cheese, cream cheese, sherbets or breakfast drinks were also associated with the disorder [16]. Next, scientists focused on girls aged 9-15 and their habits of drinking milk. Primary analysis was carried out on a cohort of 3841 girls. Multivariate PRs for total milk were 1,20, 1,19 for whole and skim milk and 1,17 for low fat milk. This result again showed positive correlation between acne prevalence and milk consumption [17]. The last study included 4273 boys. Scientists were looking for the same findings as in the previous researches. Having the highest (more than 2 servings a day) and the lowest (less than 1 serving a week) numbers of servings compared, multivariate PRs were counted. The results were 1,16 for total milk, 1,10 for whole/2% milk, 1,17 for low-fat milk and finally 1,19 for skim milk [18]. Analysis of these statistics clearly shows that milk consumption has influence on acne vulgaris occurrence. What is more, they pointed skim milk as the strongest risk factor for people loaded with predispositions for acne. LaRosa et al. seem to agree with these conclusions. Case-control study of 225 participants aged 15-19 with moderate or without any acne was carried out. Attendants were examined using Global Acne Assessment Scale. Those with acne consumed much more low-fat or skim milk than healthy ones. Full-fat milk was not positively correlated with the disease [19]. However other scientists tend to agree with Adebamowo's conclusions only partially. Ulvestad et al. on a cohort of 2489 adolescents (1112 boys and 1377 girls) have certainly shown dependence of drinking milk and appearance of acne. Only 11,7% of teenagers who did not consume any dairy presented the symptoms, while those with moderate and high daily intake were 13,4% and 15,4% respectively. Norwegian researchers have come to the different conclusion. They found no significant association between acne prevalence and skim milk consumption. Their work suggests full-fat dairy products as more harmful in this case [20]. Even with these contrasts, all of the papers state that drinking milk can induce acne outbreaks.

Many people believe that high intake of chocolate can lead to presence of acne as well. Despite the fact that there are several studies on this topic, most of them are carried out on a small groups and thus they are often insufficient to be used in a debate. For example Delost et al. carried out a single-blind randomized crossover study on 54 college students (average 21,4 years old). They were randomly given 43g of milk chocolate or 15 jelly beans both providing the same glycemic load. Changes were evaluated after 48 hours by a dermatologist using photographs. Crossover was performed after 4 weeks. Results showed visible increase in acne lesions in the group of chocolate consumers (+4,8 lesions) compared to the group with jelly beans (-0,7 lesions) [21]. Chocolate was investigated also by Suppiah et al. in their research in Malaysia. A total of 114 participants (57 healthy and 57 with acne) were recruited to examine the impact of various products on acne development. Calculated odds risk was as high as 2,40 firmly confirming the correlation [22]. Teams led by Caperton et al. and Vongraviopap et al. both worked on possible exacerbations of acne caused by chocolate. First of them designed double-blind, placebo-controlled, randomized, controlled trial with 14 men (18-35 years old) as its members. They were told to swallow capsules with unsweetened 100% cocoa, hydrolyzed gelatin powder or both of them. Results were visible after just 4 days. Mean numbers of lesions and comedones increased from 3,9 at baseline to

10,5 at day 4 and from 3,3 to 7,9 respectively in 13 of participants who took cocoa pill and completed the research [23]. In the second study 25 male subjects aged 18-30 were enrolled. Before the beginning, they were asked to avoid any cocoa products for 4 weeks. After that, they had to consume 25g of dark 99% chocolate every 20 hours. They weren't allowed to start new sunscreens, moisturizers or to use any other cleanser other than non-soap one provided by the scientists for 8 weeks. Weekly evaluations were performed using Leeds revised acne score. After 4 weeks scores jumped from 2,04 at baseline to 2,48. The biggest changes involved comedones (11,84 vs 20,28) and inflammatory papules (6,60 vs 13,70). Pustules and nodules were not altered that much (0,32 vs 0,40 and 0,84 to 0,28) [24]. Despite some limitations, these researches provide some data useful to consider chocolate's influence. There is definitely much space for further research on this field.

Conclusions:

Diet is confirmed to be an important risk factor of many different diseases including acne vulgaris. Researches point its undoubted correlation with consumption of milk, chocolate and products rich in carbohydrates. In company with these conclusions, more and more information about acne pathophysiology is discovered. Possible common pathomechanisms and their better understanding can open a whole new palette of treating schemes. Since prevention is always better than curing, knowledge gathered in described studies may help to reduce very high acne occurrence around the world. Nonetheless, many of them have significant limitations and so further research should be conducted to provide stronger evidence.

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