2018

Associations Among Acne Vulgaris and Western Diet

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Recommended Citation
Suda, Kate Meredith, "Associations Among Acne Vulgaris and Western Diet" (2018). Physician Assistant Scholarly Project Posters. 25.
https://commons.und.edu/pas-grad-posters/25

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Acne vulgaris is one of the most common dermatologic conditions, especially among the adolescent population.

The pathogenesis of acne is largely multifactorial, with heredity and hormones strongly contributing to one's risk of developing the chronic inflammatory skin condition.

High prevalence rates of acne in the adolescent population cannot be attributed solely to the influence of Western diet (WD) that stimulates insulin-like growth factors (IGFs).

The purpose of this scholarly project is to determine if there is an association between the presence of acne vulgaris and the consumption of WD, as a physiological link between the high glycemic food and dairy products that compose the typical WD exists.

There are numerous studies that have suggested a correlation between the presence of acne vulgaris and the consumption of WD. Associations have been found between acne vulgaris and dairy intake, as well as a high glycemic load and dairy-rich WD.

While not fully understood, it is hypothesized that with the increase in consumption of high glycemic-load and dairy-rich foods composing the WD, concentrations of insulin, IGFs, as well as free-circulating androgens has increased, disrupting the endocrine cascade and promoting acne formation.

The purpose of this scholarly project is to explore the pathophysiology of acne vulgaris, and determine if there is an association between the presence of acne and the consumption of WD and to establish a link connecting high glycemic foods and dairy products and their propensity to cause inflammatory acne.

Acne vulgaris, the most common skin ailment in the United States, has become a growing concern in today’s population; more than 14 million office visits a year are attributed to the diagnosis and treatment of acne (Mancini, 2008).

This inflammatory disease of the sebaceous follicle now affects around 85% of adolescents in Westernized nations (Malvich, 2018). Acne is thought to be a disease of wealthy nations, where an abundance of food, particularly processed food, is readily available. Researchers seem to believe two major cellular processes, regulation of the steroid receptor co-activator (SRC-1) in the function of epidermal keratinocytes, are disrupted in patients with acne (Szabo & Khan, 2011).

There have been numerous studies that have suggested a relationship between acne vulgaris and consumption of the high glycemic and dairy-rich WD. While not fully understood, it is hypothesized that with the increase in consumption of high glycemic-load and dairy rich foods comprising the WD, concentrations of insulin, IGFs, as well as free-circulating androgens has increased, disrupting the endocrine cascade and promoting acne formation. Many studies have found that not a single case of acne vulgaris was observed in the high glycemic-load group, during the observation periods and acne presentation is likely a result of the environmental factor, diet.

High prevalence rates of acne in the adolescent population cannot be attributed solely to the influence of Western diet (WD) that stimulates insulin-like growth factors (IGFs). Associations between Hills Glycemic Load Diet and Acne Vulgaris

A randomized controlled trial performed by Cerman, et al. (2016) found that diets with high-glycemic load were positively associated with acne vulgaris for participants with present acne. Glycemic index and glycemic load were found to be higher (p = .022) when compared to the control group (p = .001). There was a positive correlation between acne severity and glycemic index values (r = .14, p = .014). A European, longitudinal, questionnaire-based population study (Adebamowo et al., 2011) showed improvement in insulin sensitivity and the control group, during the observation periods and acne presentation is likely a result of the environmental factor, diet.

How consuming high-glycemic-load diet meals actively increases hepatic secretion of very low density lipoproteins (VLDLs), in turn increasing endocrine and homeostatic changes leading to painful papule or pustule (DynamPlus, 2018).

Associations between Hyperinsulinemia and Acne Vulgaris

How hyperinsulinemia can cause a shift in the endocrine pathways related to growth, and therefore upregulation of insulin growth factors, more specifically, IGF-1, which reduces insulin-like growth factor-binding protein 3 (IGFBP-3).

How hyperinsulinemia can cause overexpression of the epidermal growth factor receptor (EGF-R) by elevating fatty acids in the plasma, which induces production of transforming growth factor beta (TGF-beta 1), and in turn, increased concentrations of both EGF and TGF-beta1 can depress localized keratinocyte synthesis of IGFBP-3, which blocks IGF-1 to keratinocyte receptor and therefore promotes proliferation of keratinocytes.

Another biochemical pathway published by Melnik (2016), found that intake of hyperglycemic carbohydrates and milk products both have the propensity to induce postprandial rises of insulin and glucose, which signal the activation of mTORC1. Increased expression of mTORC1 kinases in the skin promotes cell growth and proliferation of keratinocytes.

Recent studies have suggested that as diets begin to Westernize, prevalence rates of acne vulgaris have increased.

High-glycemic-load carbohydrates and dairy products now comprise nearly 40% of the daily energy in the typical Western diet in the United States, which is a significant increase in the glycemic load that has been occurring in the last few decades.

With the increase in consumption of high-glycemic-load and dairy rich diets, the concentrations of insulin and IGF-1, as well as free-circulating androgens, has increased, leading to acne vulgaris.

Taken together, these data suggest that the endocrine cascade induced by hyperinsulinemia enhances sebum synthesis with androgenic hormones, contributing to the development of acne vulgaris, this increase is paramount.

All in all, more current studies are warranted to thoroughly investigate the associations between the high glycemic load and dairy-rich foods that make up Western diet.