



ACNE, ROSACEA, AND RELATED DISORDERS (INCLUDING HIDRADENITIS SUPPURATIVA)

DIETARY EXPOSOME AND NUTRIGENOMICS IN ACNE VULGARIS

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Introduction: Accumulating evidence underlines the role of hyperglycemic carbohydrates and milk consumption in the pathogenesis of epidemic acne vulgaris.

Objective: Epidemiological evidence for the pathogenetic impact of the dietary exposome in acne vulgaris needs to be related to recent advances in deviated endocrine signaling in acne metabolomics.

Methods: By means of translational research, epidemiological data on diet-induced acne are related to nutrigenomic effects of hyperglycemic carbohydrates and milk signaling.

Results: Epidemiological studies identified two major factors of Western diet promoting acne vulgaris: high intake of hyperglycemic carbohydrates and milk consumption. Hyperglycemic carbohydrates and milk intake increase insulin as well as insulin-like growth factor 1 (IGF-1) signaling. Both, insulin and IGF-1 activate the kinase AKT, the key stimulator of the nutrient-sensitive kinase mechanistic target of rapamycin complex 1 (mTORC1). Increased expression of IGF-1 and mTORC1 has been confirmed in sebaceous glands of acne patients compared to acne-free controls. AKT enhances the nuclear export of the transcription factor FoxO1, which is a negative regulator of androgen receptor and sterol regulatory element-binding factor 1 (SREBF1), the key transcription factor of sebaceous lipogenesis. In addition, AKT activates mouse double minute 2 (MDM2), the key suppressor of the transcription factor p53, which is a negative regulator of androgen receptor, IGF-1 receptor, and mTORC1 signaling but enhances the expression of FoxO1 and FoxO3.

Conclusion: In accordance with reported epidemiological evidence, enhanced insulin/IGF-1/AKT/mTORC1 signaling and reduced p53/FoxO1/FoxO3 signaling mediated by key components of Western diet, sugar and milk, promote acne pathogenesis. Epidemic acne vulgaris is a disease of Western civilization promoted by nutrigenomic effects of Western diet.

