



ALCOHOL AND THE SKIN: A COMPREHENSIVE REVIEW OF CUTANEOUS MANIFESTATIONS AND UNDERLYING MECHANISMS

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ABSTRACT

Alcohol consumption, a longstanding component of human social and cultural practices, is associated with a wide range of systemic health effects. While its deleterious consequences on hepatic, neurological, and oncological outcomes are well established, its impact on dermatological health remains comparatively underexplored. Increasing clinical and epidemiological evidence indicates that both acute and chronic alcohol intake may influence the onset, exacerbation, and clinical course of various dermatological conditions. This review critically examines the relationship between alcohol consumption and several cutaneous disorders, including acne vulgaris, rosacea, psoriasis, atopic dermatitis, and both melanoma and non-melanoma skin cancers. Special attention is given to the pathophysiological mechanisms by which alcohol contributes to skin disease, including

immunedysregulation, increased oxidative stress, impaired skin barrierfunction, and carcinogenic pathways. By synthesizing current research, this article aims to elucidate the dermatological implications of alcohol use and underscore the necessity for further targeted investigations in this under recognized area of clinical relevance.

INTRODUCTION

Ethanol is a small, amphiphilic molecule soluble in both lipids and water, allowing it to permeate and affect nearly all tissues in the body. The extent and duration of tissue recovery following alcohol-related injury are influenced by the amount and chronicity of alcohol consumption.^[1] Although the cutaneous effects of ethanol are generally less pronounced than its impact on the liver, gastrointestinal tract, and central nervous system,^[2] they warrant

attention given the skin's status as the largest organ of the human body. Orally ingested ethanol is primarily metabolized in the liver, where it is oxidized to acetaldehyde by alcohol dehydrogenase and subsequently converted to acetic acid. Ethanol and its metabolites have been implicated in the initiation and exacerbation of various dermatologic conditions.^[3]

Alcohol consumption may contribute to the development or exacerbation of skin diseases through several interrelated mechanisms:

Skin–Gut Axis Dysregulation: Alcohol alters gut microbiota composition, which plays a critical role in systemic and skin inflammation. Many patients with dermatologic conditions, such as rosacea, often present with concurrent gastrointestinal disorders, including small intestinal bacterial overgrowth (SIBO) and *Helicobacter pylori* infection.^[4]

Induction of Cutaneous Inflammation: Ethanol enhances the production of pro-inflammatory cytokines in keratinocytes and promotes lymphocyte proliferation, thereby aggravating chronic inflammatory dermatoses such as psoriasis.^[5]

Increased Vascular Permeability: Alcohol induces microvascular dilation and increases capillary permeability,^[6] which may account for facial flushing commonly observed after alcohol intake. This vascular response can further contribute to cutaneous inflammation and edema; alcohol produces acetaldehyde and generates reactive oxygen species, which affect the normal biological function of DNA through oxidative stress and epigenetic effects.

How Alcohol Affects Certain Skin Conditions

Alcohol consumption is associated with an increased risk of several chronic skin disorders and may exacerbate existing conditions through various systemic effects.

a) Increased Risk of Skin Infections

Excessive alcohol intake impairs immune function and nutrient absorption, both of which are essential for maintaining skin integrity and defense. Additionally, alcohol-related injuries, combined with compromised immunity, increase susceptibility to bacterial and fungal skin infections.^[7]

Rosacea

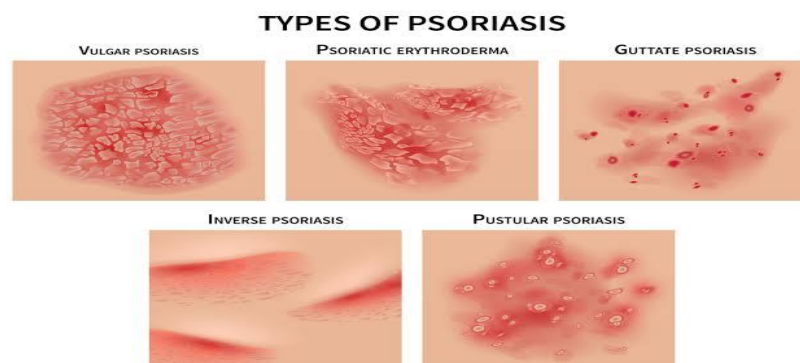
Rosacea is a chronic inflammatory skin disorder characterized by facial erythema, telangiectasia, and inflammatory lesions. Although alcohol does not cause rosacea, it can

trigger and worsen symptoms such as flushing and redness. Rhinophyma, a phymatous subtype marked by nasal tissue hypertrophy, is often mistakenly linked exclusively to chronic alcohol use. This misconception contributes to stigma, as rhinophyma can occur independently of alcohol consumption and should be recognized as a late-stage manifestation of rosacea.^[8]



Psoriasis

Excessive alcohol consumption is associated with an increased risk and severity of psoriasis, a chronic autoimmune inflammatory skin disease. Heavy drinking in patients with psoriasis may contribute to treatment resistance. Furthermore, the presence of alcohol-related liver disease often limits therapeutic options, as several systemic psoriasis treatments are contraindicated in liver impairment.^[9]



Seborrheic Dermatitis

Seborrheic dermatitis is a chronic, relapsing inflammatory skin condition characterized by erythematous, greasy, and scaly patches, typically affecting sebaceous gland-rich areas. Excessive alcohol consumption has been associated with increased incidence and severity of seborrheic dermatitis, potentially due to associated nutritional deficiencies. Chronic alcohol use can lead to reduced intake and absorption of essential nutrients such as zinc and vitamin B6, both of which are implicated in the pathogenesis of the condition.^[10,11]

Porphyria Cutanea Tarda Porphyria cutanea tarda (PCT) is the most common subtype of porphyria and results from decreased activity of the hepatic enzyme uroporphyrinogen decarboxylase. It presents with photosensitive blistering, skin fragility, and hyperpigmentation. Alcohol consumption is a major precipitating factor, primarily due to its role in inducing hepatic injury and iron overload, which contribute to PCT pathogenesis.^[12]



Nummular Dermatitis

Nummular dermatitis, also known as discoid eczema, is characterized by coin-shaped, pruritic, eczematous plaques. It has been reported more frequently in individuals with chronic alcohol use, particularly those with underlying liver dysfunction, suggesting a potential association with alcohol-related hepatic impairment.^[13]



b) Increased Risk of Skin Cancer

Chronic alcohol consumption is associated with an elevated risk of skin cancer, including both melanoma and non-melanoma types. Alcohol impairs immune surveillance and enhances susceptibility to ultraviolet (UV) radiation-induced DNA damage. Additionally, ethanol metabolism produces acetaldehyde, a recognized carcinogen, which may contribute to photocarcinogenesis.

Skin cancer

Along with increasing the risk of liver, pancreatic, and breast cancer, alcohol increases the risk of skin cancer including squamous cell carcinoma, basal cell carcinoma, and melanoma.

Vascular Effects of Alcohol

Chronic alcohol consumption can cause persistent facial erythema due to cutaneous telangiectasia, resulting from dysregulation of vascular tone. Acute alcohol intake commonly induces transient facial flushing, primarily due to accumulation of acetaldehyde, a metabolic byproduct of ethanol. Acetaldehyde promotes vasodilation, partly via histamine release. Up to 40% of individuals of East Asian descent exhibit alcohol-induced flushing due to a genetic variant in ALDH2, the enzyme responsible for acetaldehyde metabolism, leading to its accumulation.^[14]

DISCUSSION

The relationship between chronic alcohol consumption and skin health is complex, involving direct physiological effects, nutritional deficiencies, systemic organ dysfunction, and behavioral factors. This review synthesizes current evidence on how alcohol contributes to diverse dermatologic conditions. Chronic alcohol use is associated with various cutaneous manifestations, including facial flushing, rosacea, psoriasis exacerbation, pruritus, impaired wound healing, and increased susceptibility to infections. These outcomes stem from interconnected mechanisms such as immune suppression, hepatic detoxification impairment, dehydration, and oxidative stress, all of which undermine skin integrity. Notably, alcohol-related liver disease produces characteristic dermatologic signs—jaundice, spider angiomas, and palmar erythema—that reflect underlying systemic pathology.

Nutritional deficiencies common in alcohol use disorder, particularly of zinc and vitamins A, C, D, and B-complex, further impair epidermal function and immune defense, contributing to xerosis, skin atrophy, ecchymosis, and delayed healing. The psychosocial burden of alcohol-related skin disorders is significant; visible skin changes often lead to stigma, social isolation, and psychological distress, which can exacerbate alcohol dependence and hinder medical care, creating a vicious cycle. This highlights the need for integrated treatment addressing both dermatologic and mental health aspects.

Future research should emphasize longitudinal and mechanistic studies to elucidate causal pathways and therapeutic targets. Evaluating dermatologic interventions during alcohol

rehabilitation may improve outcomes. A multidisciplinary approach combining dermatology, addiction medicine, nutrition, and mental health care is essential for optimizing patient management.

CONCLUSION

Alcohol negatively impacts skin health via metabolic, immune, vascular, and nutritional mechanisms. Chronic use worsens inflammatory skin diseases, infections, porphyria cutanea tarda, and skin cancers, often signaling systemic dysfunction such as liver disease and malnutrition. Recognizing these signs enables early intervention. Future studies should explore ethanol's direct effects, skin recovery after abstinence, and targeted treatments. Multidisciplinary care is essential to improve patient outcomes.

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