Psoriasis: Targets and Therapy

Integrating lifestyle-focused approaches into psoriasis care: improving patient outcomes?

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Abstract: Psoriasis is one of the most well described cutaneous disorders, with a large body of literature devoted to describing its pathogenesis and treatment. In recent years, attention has turned toward the mechanisms by which lifestyle can impact psoriatic disease, and how lifestyle interventions may help to alleviate cutaneous, rheumatological, and comorbid disease in the setting of psoriasis. The following review explores our current understanding of the interaction between lifestyle factors and psoriasis and describes outcomes of interventions meant to target these factors.

Keywords: psoriasis, lifestyle, tobacco, alcohol, obesity, depression

Introduction
Psoriasis is perhaps the most studied of dermatologic disorders, with an expansive body of literature devoted to describing its pathogenesis and treatment. It is largely understood to be an inflammatory disorder with both genetic and environmental factors contributing to its development. As such, treatment strategies for psoriasis tend to focus on anti-inflammatory and immunomodulating therapies ranging from topical corticosteroids to systemic immunosuppressive agents including biologics. Over the last decade, interest has grown in the way that lifestyle impacts disease progression in patients with psoriasis, and how lifestyle modification-based strategies can help to create a holistic approach to therapy for optimal outcomes in the face of cutaneous, rheumatologic, and comorbid manifestations. The following review outlines our current understanding of how lifestyle factors impact psoriasis and its many manifestations, and describes outcomes of interventions targeted at optimizing these factors.

Lifestyle factors and interventions
Smoking and smoking cessation
Tobacco smoking represents one of the greatest public health risks, due to its widespread use (estimated at 22% of the population) and role as a risk factor for the development of cerebrovascular disease, coronary artery disease, myocardial infarction, COPD, and a host of malignancies. Evidence has also shown that tobacco smoking is associated not only with the development of psoriasis, with a larger effect seen in women, but also worsening severity of cutaneous disease in a dose-dependent fashion and altered response to treatment. These effects are well described in a systematic review and meta-analysis by Armstrong et al, which found an increased risk of developing psoriasis with both greater duration of smoking and quantity of cigarettes.
smoked per day in both current and former smokers, as well as a higher likelihood of current or former tobacco use in psoriasis patients versus controls.\textsuperscript{7}

It is thought that tobacco use impacts psoriatic disease much in the same way that it exerts its toxic effects on the pulmonary and cardiovascular systems. First, cigarette smoking is a source of oxidative stress, introducing free radicals into the body, and decreasing antioxidant resources/systems.\textsuperscript{9} Given lesional psoriatic skin was found to be overwhelmed with an overabundance of reactive oxygen species and therefore depletion of antioxidants, smoking would only increase this imbalance.\textsuperscript{10} Substances found in tobacco have also been found to alter the expression of a host of inflammatory signals, including many known to be dysregulated in psoriatic disease.\textsuperscript{11–16}

It has also been known for some time that patients with psoriasis often exhibit a greater risk for medical comorbidities including hypertension, diabetes, obesity, dyslipidemia, and the metabolic syndrome.\textsuperscript{17–21} These together with the known effects of smoking may create a perfect storm of risk factors for a cardiovascular or cerebrovascular event. Taking all of the above information into account, one can posit that smoking cessation would be a positive step toward alleviating cutaneous disease and diminishing comorbid risk factors for psoriasis patients. However, there is a glaring gap in the literature when it comes to smoking cessation initiatives and outcomes in patients with psoriasis, leaving many questions unanswered. While we know that smoking can affect psoriatic disease in a dose-dependent fashion, we have yet to discover whether or not smoking cessation improves cutaneous disease, or response to treatment. While smoking cessation has been shown to alter the course of cardiovascular and cerebrovascular disease, it has yet to be demonstrated whether or not the effect size of this intervention is altered in patients with psoriasis. Lastly, patients with psoriasis undergo additional physical and psychological stress, which may explain the higher rate of tobacco use in psoriasis patients over the general population. It is not yet known whether specific smoking cessation interventions are less effective for patients with psoriasis. In the absence of this data, providers should screen for tobacco use as part of a comprehensive approach to care, and offer smoking cessation interventions to motivated patients with psoriatic disease.

Obesity and weight loss interventions
Obesity has reached epidemic proportions in the global population, with obesity rates doubling since 1980.\textsuperscript{22} While many factors contribute to obesity, certain disease states, psoriasis among them, have been found to be independent risk factors for the development of obesity.\textsuperscript{23} Several meta-analyses have found that patients with psoriasis have >50% increased odds of obesity, and that this association is greater with increased severity of cutaneous disease.\textsuperscript{18,24} Furthermore, more profound cutaneous disease is associated with a greater degree of obesity.\textsuperscript{25} These associations are reflected in epidemiologic data demonstrating that psoriasis patients have a higher prevalence of obesity and a greater average body mass index when compared to the general population.\textsuperscript{25}

Obesity is similar to psoriasis in that it is largely understood as a pro-inflammatory state. Adipose tissue is immunologically active, releasing soluble signals in the form of adipokines in response to pro-inflammatory signals from macrophages.\textsuperscript{18} The result of this process is augmentation of circulating pro-inflammatory cytokines, including IL-6 and TNF-\alpha, which are dysregulated and used as treatment targets in psoriasis and psoriatic arthritis.\textsuperscript{26} While these correlations provide a basis for understanding the association between obesity and psoriatic disease, a mechanism of progression from one condition to the other has not been identified. Given the known links that both psoriasis and obesity have to cardiovascular and cerebrovascular disease, one pro-inflammatory etiology may link these conditions, their comorbidities, and their ultimate outcomes.

While some studies have found that obesity does not impact the course of treatment in patients with psoriasis, several prospective studies have shown decreased severity of disease and comorbid conditions when patients are randomized to low calorie diets with or without prescribed exercise programs.\textsuperscript{27–29} There have also been several reported cases documenting remission of cutaneous disease following bariatric surgery.\textsuperscript{30,31} These reports considered, there is still a dearth of information regarding targeted weight loss interventions for patients with psoriasis. Larger, more detailed trials documenting progression of disease, response to treatment, and overall mortality in psoriasis patients who are prescribed weight loss regimens will be key to fully elucidating the link between obesity and psoriatic disease. Nevertheless, providers should screen psoriasis patients for obesity, and recommend appropriate interventions to control weight including diet, exercise, and consultation with a dietician.

Alcohol use and alcohol abstinence measures
The association between alcohol abuse and psoriatic disease has been known for decades, though the mechanism of this association is still unknown.\textsuperscript{32} While low level alcohol
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